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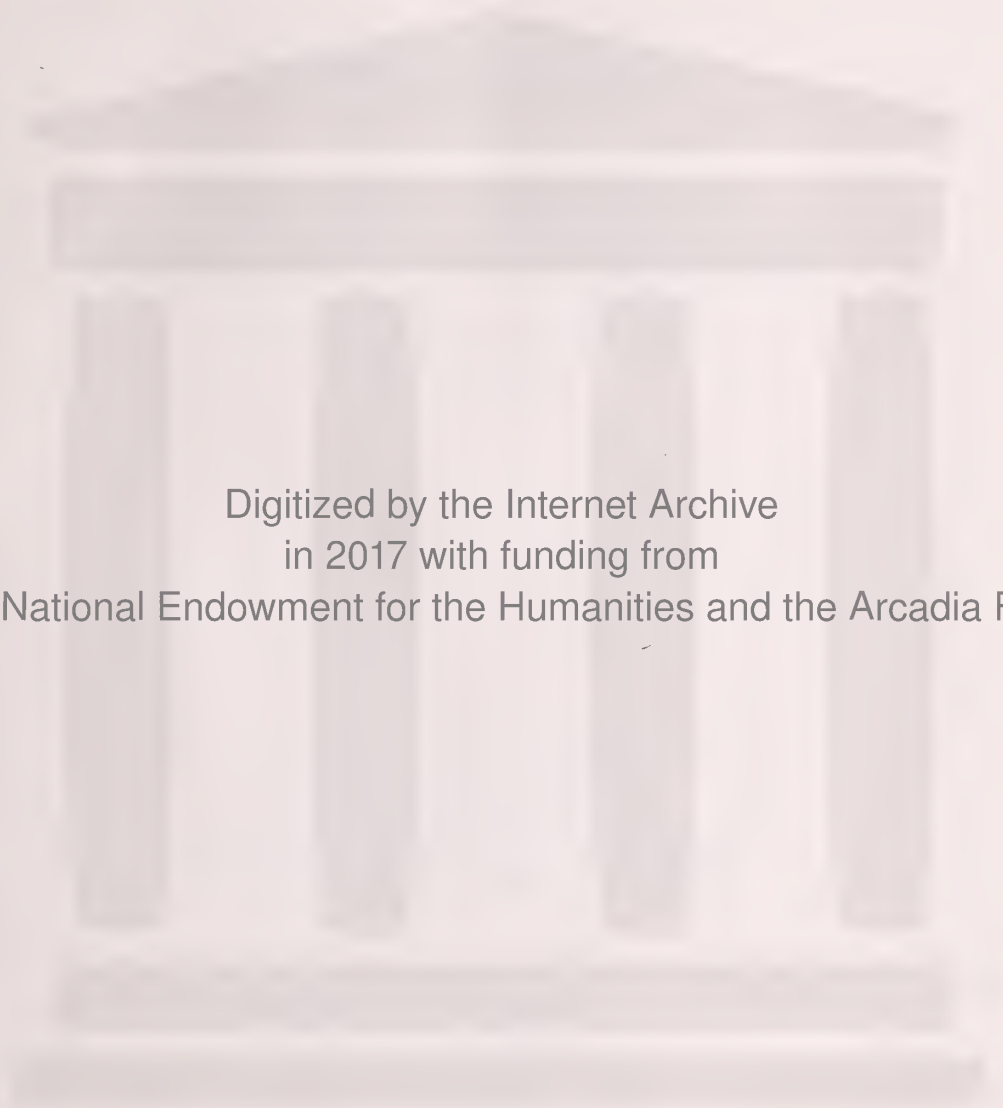

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Devoted to the interests of the medical profession and public health in Texas

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Deluge from Grass Roots Needed in Washington

"This is a call to arms! Now is the time to begin an all-out effort to defeat the King-Anderson bill," declared Dr. Edward R. Annis, AMA President, in a recent letter to all state and county medical societies.

When hearings on the bill resume, testimony will include a major presentation by the AFL-CIO in favor of the measure. "We need a deluge of expression from the grass roots of this nation reaching Washington in such volume that it cannot be ignored," stressed Dr. Annis. Emphasizing the value of letters from persons in all walks of life, Annis said that every word of opposition from a non-doctor has tremendous impact on Congressmen, especially if these persons helped to elect him.

In Texas, "Operation Hometown" chairmen are waging a relentless battle against the King-Anderson bill, as are the officers of the Texas Medical Association. In a statement filed in November with the House Ways and Means Committee, TMA President Robert Mayo Tenery strongly illustrated the value of the already-existing Kerr-Mills program in Texas, as well as the development of private insurance policies available to persons over 65. This testimony is to be printed in pamphlet form and mailed to all members of TMA, the Woman's Auxiliary, and others who request it.

In addition, four newspaper advertisements will be made available to county medical societies, through TMA, for use in furthering the campaign against King-Anderson legislation. The state Association also will continue to cooperate with Blue Cross-Blue Shield of Texas in providing special

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debate kits for high school students for interscholastic debate on the issue of compulsory medical care for all citizens.

TMA will sponsor a legislative mission to Washington early in 1964, sending a physician to represent each Congressional District, as well as the General Counsel and the Executive Secretary, to confer with Texas Senators and Congressmen and to present a current progress report on the Texas program for the care of the needy aged.

As the showdown on medicare approaches, TMA will continue to fill requests for speakers through the Speakers Bureau.

Figures continue to register growing opposition; in its Dec. 28 issue, *JAMA* reported that latest results of polls conducted by members of Congress showed that 67.6 per cent of the respondents opposed the use of social security financing, 26.1 per cent favored it, and 6.3 per cent had no opinion.

In other testimony, a Catholic priest struck at the very basis of the King-Anderson proposal: ". . . let the government continue to help the aged who have a proven need for medical assistance, but do not let it set up a system whereby it infringes upon man's right and duty to provide for himself. To do so is to belittle and debase the citizens of our land."

These are ideas less remote to the layman than is the threat of socialized medicine. There are many lay organizations opposing the King-Anderson bill; they may oppose it on slightly different grounds. Whether they call it a step toward socialized medicine, a strike at the basic American philosophy of personal freedom and responsibility, or an unnecessarily expensive solution to a problem which is relatively minor makes no difference. The important point is, as Dr. Annis expressed it, ". . . if you want their help—and you do—you have to ask for it, and urge them to demand rejection of the legislation by the Committee . . . . American medicine and its many allies can win this battle. We can win because our stand is right and in the best interests of the people—our patients."

New Cover for a New Era

WE HAVE A new look this month, the most drastic new cover fashion adopted since 1949 when the *Journal* began to make use of color on its exterior. With the fresh clean design, four colors will be used alternately throughout the year. A deep gold is used this month to symbolize the change; February's cover will be in a muted aqua, March's in brown, and April's in olive green. The cycle will then repeat itself. The change in color each month is designed to help the reader to distinguish each new issue from the preceding one.

It is the hope of the Board of Publications, the Journal Advisory Committee, and the staff that this bright bold new appearance amply illustrate the goals of progress which have been outlined for "Texas Medicine" as depicted in our state's medical journal.

The interior appearance and makeup of the *Journal* has been revised over the past four years to allow the reader to find articles more easily and to make reading kinder on his eyes. This month, several new revisions are instigated to further aid the reader. Compartmental symbols are placed conveniently at the beginning of an article to give the reader some advance idea about the type of the article before him, whether it be an original research, clinical, review, or historical paper, a clinicopathologic conference, a case report, or part of a symposium.

Advisors for the *Journal* do not feel that it should lag behind in telling the story of Texas medicine or in obtaining good quality scientific papers for publication. An increasing supply of papers allows the Journal Advisory Committee to choose better papers and it is hoped that physician-writers in the Lone Star State will continue to put their best thoughts and research forward for evaluation and possible publication.

In all humility, the Journal advisors feel that "Texas Medicine" need not be relegated to the disparaging realm of "only a state journal," as medical students and practicing physicians have been overheard to say. It is the lofty goal of the six physician advisors that the *Texas State Journal of Medicine* be ranked among the very top journals and that it truly reflect the outstanding type of medicine and research being practiced in the state in this era of increasing technical complexities. Just recently, the *Journal* received its

tenth award since 1957. The latest one was a first place award in the association or membership category in competition sponsored by the Mid-Continent Conference of Industrial Editors.

The Journal Advisory Committee has established broader and higher goals for the *Journal* in the belief that general practitioners want and need more information on a wider variety of subjects than ever before and that specialists also need information on areas of the human body and soul outside their own immediate interest.

For these reasons, the Committee would enlist the support of all of Texas' physicians in making our publication better than ever. It is the individual Texas physician and his good quality of work that can make the *Journal* rank among the great by his contributions to it.

Doctors Can Expand Civic Service Through Work with Chamber of Commerce

THE PHYSICIAN'S MISSION is a life of service. Under the Hippocratic oath, he has been committed from ancient times to a high and unbending code of ethics in treating the sick. The influence of this discipline is seen in the work of physicians in every city, town, and hamlet in the land.

But we are living in a time when service is more demanding than in any previous time in our history. Man-made problems, multiplying on every hand, seem as formidable as unsolved health problems. Individual bafflement at man-made problems often has surrendered to government attempts at solution.

The physician now has a broader concept of service, namely, the ancient one defined by Hippocrates plus the modern necessity of civic responsibility. What greater service can be performed than to work to preserve the traditional integrity and independence of the American community?

The community needs the competent, intelligent, and better-educated citizen both for his professional skill and for his contribution to public discussion and action. In the same degree, all citizens need the common welfare efforts of an informed and enterprising community. The physician has a special contribution to make to community action; he also stands to benefit from the concerted action of intelligent citizens.

The local chamber of commerce offers the physician a means for taking part in community affairs. It brings together the talents and resources of business and professional people for community improvement, in the traditional American spirit of free enterprise, local initiative, and voluntary action. It also offers opportunities for participation in decision-making on matters of statewide and national interest.

In a majority of cases, the local chamber is a member of the Chamber of Commerce of the United States, a federation that brings together a national consensus of the American business community and others who believe in free enterprise principles. In states that have state chambers of commerce, the local chamber usually is a member of the state body. In Texas, there is no state organization. Four regional chambers of commerce (East Texas, South Texas, West Texas, and Lower Rio Grande Valley) are well organized, highly efficient, and will welcome applications from each reader.

While the local chamber is entirely independent and autonomous, it finds in the state chamber a statewide voice, and in the national chamber a voice for the support of policies to preserve traditional American principles for the entire nation. The national chamber, for example, has supported plans for health care for elderly persons unable to pay their medical bills. However, it has insisted that these programs should be carried out primarily through state, local, and private action. It has opposed a compulsory health care program under the Social Security System as leading inevitably to a broader plan of federally-directed health care. The range of cooperative action between local, state, and national chambers is extensive and covers many fields.

Some physicians object to joining their local chambers. Here are some of the main objections, along with answers given by physicians who are already members:

Too Busy.—Nearly all useful men are

busy; if you want something done effectively, ask a busy man. But a physician should not be too busy to attend to civic responsibilities that affect himself and everybody else. He at least can give the local chamber his support and let it work for him while he is otherwise occupied.

Costs Too Much.—Dues in the local chamber usually range between $20 and $50 a year for the individual, and they are tax deductible. By comparison, dues in the United Automobile Workers' Union are $90 a year.

The Local Chamber Accepts Some Not-Too-Desirable Members.—The chamber accepts persons who indicate an interest in the community. But wouldn't it be a shame to let people with whom you don't agree run the show, when by your participation you could improve the tone and outlook of the organization?

Physicians Contribute Through Clinics and Charity Services.—In these services, they help a needy few, but in embracing civic responsibility they help the entire community and perhaps even the state and nation.

The Chamber Is Not Democratic.—Local chambers are run in a truly democratic manner. The way to find this out is to join and become an *active* participant. The rule is that anyone really interested is sincerely welcomed.

Members Are Impractical Do-Gooders.—Again, too bad that they lack your influence. The truth is that there probably is not a community in the country that has not been bettered to some extent by its local chamber.

Physicians Should Not Be Identified Publicly with Any Social or Ideological Group.—Any organization as active in promoting the general welfare as the local chamber hardly fits this concept. Although it sometimes is accused of being anti-this or -that, actually it is not anti-anything except apathy, indifference, and an attempted negation of the American way of life.

Membership can bring personal satisfaction to the doctor, as well as a richer life for his family and community, and offers a powerful link between the grass roots and the Capitol of our nation.

—J. C. TERRELL, M.D., Stephenville.

Cautious Congress Should Be Thanked

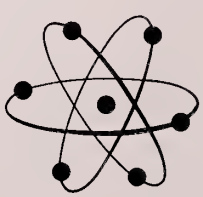
THE NEWS MEDIA are frequently reporting that our current Congress has "done nothing." I believe that our individual senators and representatives should be thanked for *refusing* certain requests.

A "do-nothing" Congress studies issues carefully. If fewer bills are passed this year, fewer amendments and repeals will be necessary next year.

Our present Congress was criticized for failing to pass appropriation bills for established departments of the government. When this happens, the department operates under a "continuing resolution" and CANNOT spend more than was spent during the past fiscal year. It may be quite difficult for a member of Congress to say "no" to a request; but inaction can be just as effective in saving tax money.

Please write your senators and representatives (including your representative-at-large) to thank them for their restraint.

—DICK CASON, M.D., Hillsboro.



personal

Opinions and Views

Journal Article Challenged

Doctor Questions Actual Value Of Experiments with Animals' Pain

AN ARTICLE entitled "Doctors Must Defend Use of Animals in Experiments" appeared on page 824 of the August, 1963, issue. I wish to take exception to the statement in that article that ". . . it would also deny human patients the benefits of pain-relieving drugs which can be tested only by creating painful situations and then demonstrating the drug's powers of relief." This was one of the arguments put forth against proposed legislation which would prohibit experiments likely to cause pain to an animal.

While the whole subject is not suitable for complete discussion in the confines of a short letter, I should like to remind you that some physicians, who are neither out of their minds nor hold such extreme views as the proverbial old ladies' society for the care of sick cats, are of different views on this subject than those which apparently reflect the official thinking of the American Medical Association.*

First, some of the most important analgesic and anesthetic drugs were discovered without animal experimentation, among these being opium, cocaine, and ether. Then most interestingly, just how are you going to ask an animal whether something does not hurt any more or whether the drug induces side reactions such as nausea or disorientation? The statement that human patients would be denied pain relieving drugs is worthy of a television drama, or a scheme cooked up by an advertising agency to cadge funds for a hospital building program or polio drive.

Of course as the article shows, that is just the weakness of the animal experimenter's position. He first states that his standards are high, and that usually the animals suffer no pain, but every once in a while someone slips and admits that experiments are done which are painful, and makes some spurious plea that human suffering is going to be avoided, just as if there were no drugs available to counteract pain today, which is not true at all.

In another sense the article has, I believe, inadvertently shown the truth of part of the situation when it speaks of the necessity for more money. That is just as many things are today including businesses, churches, education, and medicine. To some extent medicine has become something which might be vaguely analogous to abstract art, wherein only the initiated can understand some of its esoteric mysteries.

The worst thing is that animal experimentation is on the way to becoming a sort of big business also, just as is supplying band uniforms and instruments and building stadiums in the field of education, and as vari-

*The *Journal* article was based on a story in the *JAMA*.

Readers of the *Texas State Journal of Medicine* are invited to submit their "Personal Opinions and Views" on subjects they believe might be of interest to readers of the Journal. Submissions will be reviewed by Journal advisors for their publication merit. Items may be directed to the Managing Editor of the Journal at 1801 North Lamar Blvd., Austin.

ous other lines of enterprise are in the field of religion.

To discuss the exact function of research in medical education is beyond the scope of a letter. The most casual observer can see that medical education is engaged in many activities which are not directly related to teaching the students how to care for patients. In space medicine, for instance, many activities do not appear to be conducted by experienced physicians who are patient-oriented as far as the ordinary patient is concerned, exclusive of aviators and astronauts.

While the development of the human race may still, in a sense, be in part of the Middle Ages, I am not convinced that those of us who received a medical education 30 years ago are comparatively ignorant, or that there has been such tremendous progress since that time, all because of animal experimentation.

I notice great concern on the part of the profession as to what the laity thinks of the medical profession generally, and the members individually. I earnestly commend you to look at this subject realistically to determine just what is actually to the point in animal experimentation. The arguments put forth in the article are ridiculous. Consider that in the earlier Middle Ages the tortures of the Inquisition were thought to be quite all right for humans, as was also the excision of hearts on the altars of certain gods, but that just as times have changed in this respect, some of us hope that they will change for animals also.

Just because man has the power to do these things to animals certainly does not justify it morally, no matter what presumed benefits might result. Furthermore, I believe that those presumed benefits are to a considerable extent exaggerated. In this connection I should like to mention that churches seem not to regard this point very much when they have medical schools.

I should like to point out a quotation from page 39 of the Aug. 17, 1963, issue of the *Journal of the American Medical Association,* which certainly should be authoritative. In the article by Dr. F. Clarke Fraser, Montreal, appears the following statement:

> "Testing in animals may provide clues as to the possible action of a drug, but the results of such testing cannot be extrapolated to man."

Well! And just when *JAMA* has been plugging for animal experimentation. It is almost like those situations in which the fountain sources of political thinking suddenly change their party line, much to the dismay and chagrin of official publications which already have printed something else.

—E. G. REUTER, M.D., San Antonio.

New Outlook for Aged May Prevent Senility

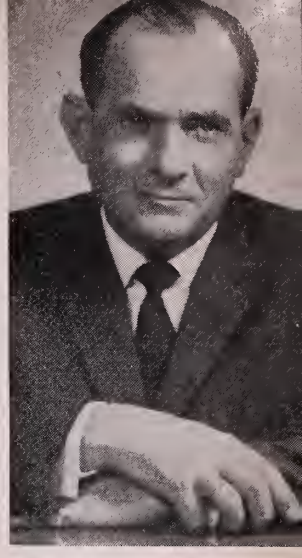
Although the aging process begins at birth, the dreaded state of old age is largely preventable, and it certainly is not inevitable. In a few years, physicians anticipate a century of living for the average person; this, however, is distinguished from a century of merely existing.

Today, medical progress has raised the life expectancy of a child to 80 or 90, but medicine is more interested in adding meaningful time to the life span than it is in just adding more years.

The American Medical Association Committee on Aging, after studying aging for seven years, feels that adding this meaningful time will require adjustment in the thinking patterns of America today. Instead of excluding the elderly person from family life and barring him from his essential function of work, society must allow him to maintain his sense of purpose and contribution to life.

After all, if the spirit is willing to continue with a rich life into old age, the flesh, fortified by modern medical understanding, is certainly strong enough to carry on.

People do not die of old age, according to the committee. No one has ever been found whose body just quit functioning because it was too old. Disease and accidents, not old age, are the culprits.



Physicians And Malpractice Suits

Physicians are becoming better informed as to the precautions that should be taken to protect themselves against malpractice suits; however, the profession as a whole must consider the causative agents that have increased the incidence of such claims. Then the profession may find the means to prevent some of the unjustified claims.

Today people are better educated in medical knowledge, and with the advances of medical science they expect more of their physicians. Articles frequently appear in lay publications that tell of gross mistakes by doctors and hospitals; this is undermining the public confidence in our profession. Many of these articles are greatly exaggerated, are isolated cases, and reveal a lack of information. Some of these isolated cases refer to a bad result in a case even though the physician may have used a high degree of care and skill in treating his patient.

A patient is less apt to bring suit against a physician when there is complete understanding, cooperation, and courtesy. But, many factors are damaging the close personal relationship between the doctor and his patient. Some people are critical of all physicians because of an unpleasant experience, such as being unable to obtain a doctor at night or on a weekend, waiting for a long period in a reception room, being rushed through an office visit, or feeling the frustration of the unknown because the doctor will not take time to explain. When a patient feels that he has been overcharged, he may be more critical of the care that he has received. Overly zealous collection techniques may also aggravate a dissatisfied patient.

County medical societies should take the initiative in disciplining those physicians who are guilty of acts which cast a poor light on the entire profession. Grievance committees and boards of censors should be fully utilized to investigate all complaints. On the state and national level medical societies can deal with the overall problem by improving the public image of the doctor and by recommending corrective measures so that good medicine is practiced in all communities. Where good medicine is practiced, there are few meritorious claims.

We should continue our efforts toward a better understanding between the medical and legal professions on a state and local level so the problems of each profession can be better understood. We should make available qualified impartial expert testimony when needed. Individual cases must be met by the involved physician, his attorney, and his insurance carrier.

Robert Mayo Tenny

President
Texas Medical Association

Biological Warfare

Nature and Consequences

SYMPOSIUM

BIOLOGICAL WARFARE (BW) is the use of living microorganisms to reduce the military capability of an enemy. Its most important effect would be in disrupting the economy of a nation through incapacitation of its working force and reduction of its agricultural crops and domestic animals. Man's physical assets—his buildings, his churches, his museums, would be uninjured. There would not be the total destruction such as would result from the use of nuclear or high-explosive weapons. Agricultural land would not be rendered infertile for the following season.

This form of warfare has never been employed on a planned, overt, military basis. Its potential threat has been considered sufficiently great, however, that this country has supported a research and development program since early in World War II. Most of this research is conducted in well-equipped laboratories at Fort Detrick in Frederick, Md.

Biological warfare is primarily a strategic weapon. It has no quick-kill effect and is, therefore, unsuitable for hand-to-hand encounter. Close-contact tactical use is precluded by the incubation period of infectious diseases. More importantly, the greatest effectiveness of BW would result from the possibility of covering extensive target areas. This demands our most thoughtful attention in planning our defense.

Basic Requirements

Biological agents must possess certain basic properties and characteristics in order to be useful for military purposes. Some of these characteristics are:

1. The agent must be highly infectious. There is much medical and epidemiological information to suggest which microorganisms are the most infectious. Those organisms which frequently cause infections among laboratory workers belong in this category. The literature[1-12] is replete, moreover, with instances in which human volunteers have been inoculated by various routes. The aerosol dose of two organsims has been determined experimentally in volunteers during the course of studies of the effectiveness of vaccines. The dose of *Coxiella burnetii* was found by Tigertt and Benenson[13] to be 10^{-9} gm. of homogenized, infected chick embryo tissue. Saslaw and associates[14] found the aerosol dose of *Pasteurella tularensis* for man to be 15 to 50 organisms. It was demonstrated in these latter experiments that vaccination with a live, attenuated organism provided excellent protection against subsequent challenge with a highly virulent strain.

2. The organism should have sufficient viability and virulence stability to meet minimal logistic requirements. There is a good probability of improving these properties through research.

3. The microorganism should be capable of being produced on a militarily significant scale.

4. The organism should not be unduly injured in the field and it should have a minimum decay rate in the aerosol state.

5. There should be minimal immunity in the target population to the particular organism. Immunity is relative, however, against most microorganisms. It is possible to break through it, in many cases, by an enlarged dose of the organism.

6. There is always a possibility that an enemy may develop an agent with unusual properties through appropriate genetic research. We are all aware of the capacity of

Humans, animals, birds, insects, and even crops are subject to the effects of biological warfare. Possible agents are mentioned; methods of delivery and mechanisms of transmitting them are reported. Immunization, trained personnel, laboratory facilities, and more research are vital to defense.

LEROY D. FOTHERGILL, M.D.

some organisms to develop resistance to specific drugs and/or antibiotics.

Bearing in mind these several characteristics, it becomes obvious that only a limited number of microbiological agents meet them. Conversely, many do not.

Methods of Delivery

There are several methods by which agents might be applied to a target. The most important of these would be through the overt military delivery of weapons systems designed to create an aerosol or cloud of the agent. Another would be through some covert method.

The concept of creating a cloud or aerosol of biological agent stimulated an intensive research program concerning the behavior and properties of small particles containing viable microorganisms. It has required the design and development of a variety of specialized equipment, including chambers, exposure equipment, aerosol generators, and sampling and assessment devices. The associated technology has been developed to the point of great quantitative precision.

This research has yielded, among other things, much information concerning the pathogenesis of respiratory infections. The exposure of animals to such particles through natural breathing is, of course, far different from the older technique of respiratory inoculation by the intranasal instillation of a fluid suspension in an anesthetized animal.

A major contribution of this research has been the demonstration of the importance of particle size in the initiation of infection. The anatomic and physiologic features of the upper respiratory tract, such as the turbinates of the nose and the cilia of the trachea and larger bronchi, are ideally suited for impinging out the larger particles to which we are ordinarily exposed in our daily existence. Very small particles, in a size range of 1 to 5 μ in diameter, are capable of passing these barriers and entering the alveolar bed of the lungs—an area that is highly susceptible to infection. There is considerable literature describing experimental investigations with a bearing on this phenomenon.[15-27]

Table 1, reporting the data of Day and Persichetti,[28] summarizes the relationship between particle size and infecting dose of four different pathogens for the guinea pig. This ratio varies among different animals, depending no doubt on the size of the air channels and the complexity of the anatomic structures of the upper respiratory system.

TABLE 1.—*Influence of Particle Size on Respiratory Virulence of Four Microorganisms for Guinea Pigs.*

Particle Median Diameter Range (μ)	Agent Respiratory Lethal Dose$_{50}$ Values			
	Bacillus anthracis (Spores)	*Pasteurella tularensis* (Bacilli)	*Coxiella burnetii** (Rickettsii)	Venezuelan Equine Encephalomyelitis† (Virus)
0.3-1.5	23,000	2.48	1,000,000	20
4.0-6.5	221,000	6,500	52,700,000	19,000
8.5-13	700,000	19,500	$> 2.4 \times 10^9$	280,000

**Dose in guinea pig, intraperitoneal median infective doses.

†Dose in mouse, intracerebral median lethal doses.

The turbinates of the guinea pig are more complex than those of the rhesus monkey, for example. The ratio between particle size and infecting dose for these two animals is illustrated in Table 2, Goodlow and Leonard.[29]

TABLE 2.—*Cells of Bacterium Tularense Required as Lethal Dose for 50% of Hosts Exposed to an Aerosol.*

Particle Median Diameter (μ)	Guinea Pig (No. of cells)	Monkey (No. of cells)
1	2.5	17.3
6.5-6.9	6,460	240
11.5-12.0	19,600	550
19-22	170,000	3,750

Some additional properties of small particles need emphasis. They can remain suspended in air for a long period of time, particularly if there is some atmospheric turbulence. Thus, the smaller the particle, the farther it will travel downwind before settling out. An aerosol of such small particles will diffuse through structures in much the same manner as a gas, thus giving it a remarkable property for target searching.

There are several critical meteorologic conditions that must be met for a biologic aerosol to exhibit optimum effect. For example, bright sunlight is rapidly destructive for living microorganisms suspended in air. There is an optimum humidity for longest survival of most organisms when airborne. Neutral or inversion meteorologic conditions are necessary for a cloud to travel along the surface. It will rise during lapse conditions. There are, of course, certain times during the daily cycle (during the late night and early morning hours) when most of these conditions will be favorable.

The behavior of a cloud of small particles containing a nonpathogenic biological agent can be illustrated by the following field trial. An aqueous suspension of the spores of *Bacillus subtilis, var. niger,* generally known as *B. globigii,* was aerosolized using commercially available nozzles. A satisfactory cloud was produced even though these nozzles were only about 5 per cent efficient in producing an initial cloud in the size range of 1 to 5 μ. In this test, 130 gal. of a suspension of spores was aerosolized. The spraying operation was conducted along a two-mile course from the rear deck of a small naval vessel, cruising two miles offshore, and vertical to an onshore breeze. There was a slight lapse condition, a moderate fog, and 100 per cent relative humidity. A network of sampling stations had been set up on shore.

The results of this trial are illustrated in Fig. 1. The cloud was sampled for a distance of 23 miles downwind from the source. Approximately 100 square miles were covered within the area sampled.

It would be appropriate to reiterate that this trial was carried out with a line source only two miles in length and that the aerosol was generated by improvised equipment. In view of this, it requires little imagination to conceive of:

1. The design of efficient military equipment to accomplish this.
2. The possibility of increasing the source strength to any desired degree.
3. Extending such a line for 10 miles, 100 miles, or any distance for which equipment might be designed, thus covering a very extensive target area.

It should be clear from these considerations that the possibility of the strategic coverage at long range of vast target areas is the major threat of BW to our nation. It is not unlikely that mixtures of agents might be used. Some antianimal agent might be included with an antipersonnel charging. Agents with a high zoonotic potential are a particular threat.

A variety of biological agents are suitable for delivery through enemy sabotage. One's imagination can run wild. There are some crucial targets, however, of great importance. The air-conditioning and ventilating systems of large buildings are obvious points for attack. Our country possesses enormous food processing industries, including the preparation of soft drinks, and the processing of milk and milk products, that are subject to sabotage. Huge industries are involved in the production of biological products, drugs, and cosmetics which are liable to this type of attack.

There are certain effects, other than the immediate infection of the target host that might result from covering a large area with a biological aerosol. Such an extensive coverage might create certain ecologic and environmental problems of great importance. For one thing, bearing in mind the capability of aerosols to penetrate structures, it is obvious that there would be widespread contamination in hospitals, food supplies, public and domestic kitchens, restaurants, warehouses, and other vital points.

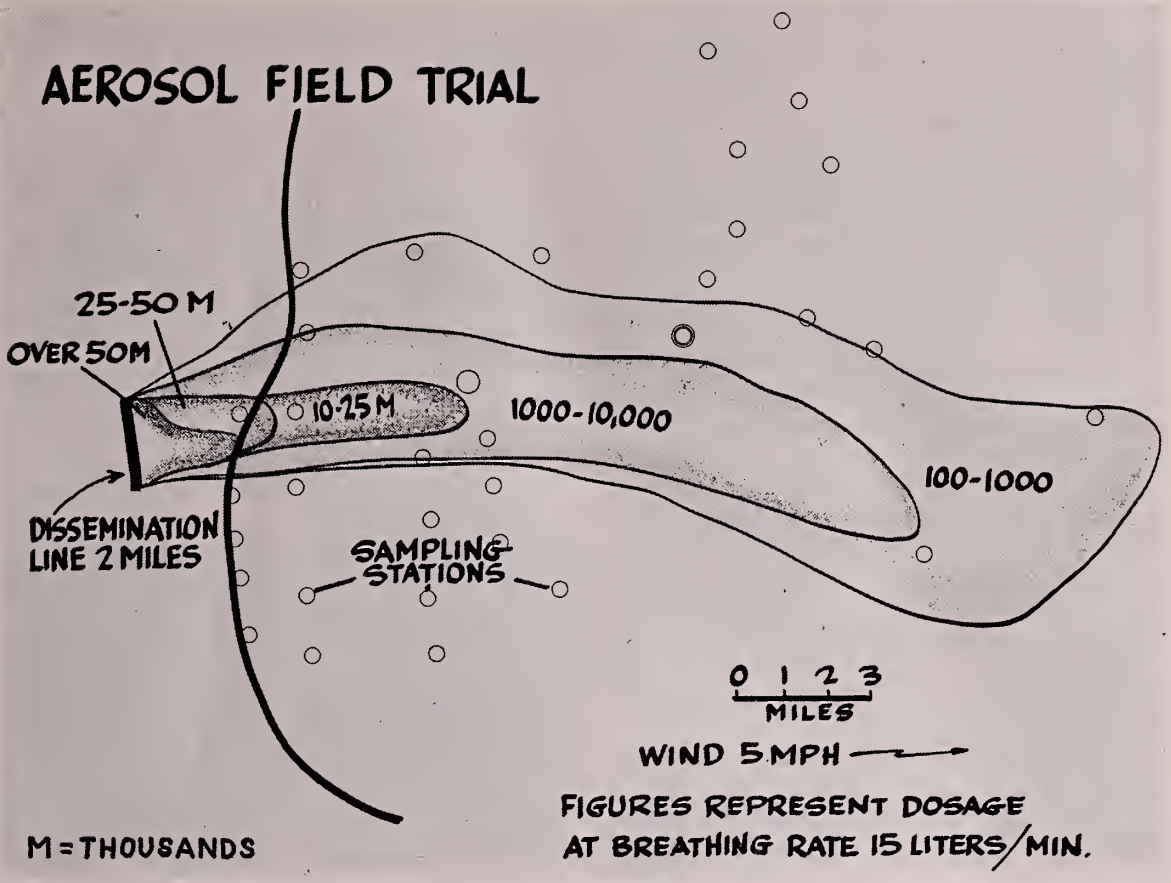


Fig. 1.

There is another and even more serious problem to be considered.[30-32] All living things in the target area would have an opportunity to inhale the aerosol. Before considering this circumstance in detail, it would be well to review briefly the major mechanisms involved in the transmission in nature of the agents of the more important epidemic diseases. Certain generalizations can be drawn from this, realizing of course that in general biology there are always exceptions.

Mechanisms of Transmission

Four major mechanisms may be considered:

1. Many of the great plagues of man are vector-borne. Among these are bubonic plague, malaria, epidemic or classical typhus fever, yellow fever, dengue, the acute encephalitides, trypanosomiasis, filariasis, and other less dramatic diseases. Many types of disease-producing organisms are involved in this category—viruses, rickettsiae, bacteria, protozoa, and nematodes.

In these diseases, infection is by penetration of the skin; an actual inoculation through the bite of the vector. The infecting microorganisms may be rubbed into the skin from the feces of the vector in some instances. The infecting dose for man is probably small in most cases, that is, the amount of agent presented during the course of the vector's bite.

Probably the most important generalization in considering this mechanism is that *various zoologically unrelated host species are involved.* In every case, both man and the vector are implicated. Man, mosquitoes, equines, birds, amphibians, and reptiles may be involved in some of the acute encephalitides. This may be a natural evolutionary result of frequent exposure to and transmission among a multiplicity of different hosts. This is a vital point in considering large area coverage with an aerosol of certain microorganisms.

2. Water-borne agents are associated with certain major epidemic diseases. Asiatic cholera is the most spectacular of these. Others are typhoid fever and dysentery—both bacillary and amebic. In this case, the

organism enters the body by mouth. (One exception would be the skin penetration by the cercariae of schistosomiasis.)

In all cases where the organism is obtained by mouth, the infecting dose is probably extremely variable—in many instances it may be very large. The dose may depend, in part, on the physiologic state of the individual at the moment of ingestion; for example, the level of gastric acidity. Most of these infections are highly host-specific. Transmission is man-to-man with water serving as the transporting medium.

3. In the third major mechanism, respiratory or inhalation, some level in the respiratory tract is involved, at least initially. The disease may become generalized at later stages in the infection in some cases. Among the many epidemic respiratory diseases are influenza, smallpox, diphtheria, whooping cough, measles, scarlet fever, chickenpox, the common cold, and pneumonias of various etiologies.

The organism generally becomes air-borne in droplet nuclei in the spread of these maladies. The *range of dissemination is relatively short,* usually a matter of a few feet, as one individual to another in close proximity.

Interestingly, the agents of most of these diseases are highly host-specific; they are largely diseases of man and, in some cases, it has been difficult or impossible to reproduce them in experimental animals. Again, this may be because they have evolved in association only with man. Because of the short range of dissemination of these agents, *there is little reason for animal and bird life to become exposed under natural conditions, particularly the wild species.*

4. The fourth mechanism represents a large and varied group of infections in which the agent is generally spread by some mechanical process—the classical tetrad of fingers, flies, food, and fomites. The mechanisms of spread may be exceedingly varied in this group.

Effects of Mass Contamination

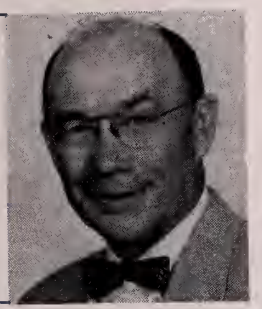
The situation that might occur when large air masses are contaminated can now be examined in the context of these considerations. Everything that breathes in the exposed area has an opportunity to be exposed to the agent. This will involve vast numbers of mammals, birds, reptiles, amphibians, and insects. Various natural history surveys have indicated surprising numbers of wild life inhabiting each square mile of countryside. It is possible that many species would be exposed to an agent for the first time in their evolutionary history. We have no knowledge of the range of susceptibilities of these many species of wildlife to specific microorganisms, particularly through the respiratory route of administration of infectious aerosols. What would be the consequences? Would new and unusual zoonotic foci of endemic disease be established? Would it create the basis for possible genetic evolution of microorganisms in new directions with changes in virulence for some species? Would it establish public health and environmental problems that are unique and beyond our present experience? This could be an especially serious problem with microorganisms having a natural zoonotic behavior, particularly those involving a considerable spectrum of host species, such as the causative viruses of the acute infectious encephalitides.

Covering a large area with an infectious aerosol may create a unique epidemiological situation. The sudden exposure of all of the multitudinous fauna of a large area to an infectious aerosol is unprecedented in epidemic history. The temporal and spatial expansion of the disease would be entirely different from natural occurrences because of the sudden seeding of an extensive area with the agent. The natural, gradual build-up of agent resulting from host-to-host transfer would be by-passed. Instead of point foci, there would be a large area focus.

We must face the threat of the possible delivery by an enemy of truly exotic agents against our human and domestic animal populations and against our agricultural crops. Indeed, many of these possible agents, such as the viruses of Rift Valley fever and louping ill, are known to be infectious for both men and domestic animals. Some of these agents, such as the viruses of Japanese or type B encephalitis, Russian spring-summer encephalitis, and Venezuelan equine encephalomyelitis, may conceivably become established in some of the bird, animal, or insect populations in the target area and thus create serious new enzootic foci. We may be faced, too, by a reintroduction of diseases that heretofore had been eliminated from our country at the cost of a great deal of

money and effort. Such diseases as Asiatic cholera, yellow fever, smallpox, and malaria of man; and pleuropneumonia, glanders, and foot-and-mouth disease of animals are examples. If such diseases were reestablished, their control again might be exceedingly costly. Finally, agents that are currently endemic or periodically epidemic might be used, and some might be disseminated over areas where they are not now prominent. Among these may be mentioned the organisms of plague, tularemia, tuberculosis, brucellosis, and coccidioidomycosis. Our country has spent millions of dollars, for example, for the eradication and control of brucellosis in agricultural animals. Cattle, swine, sheep, and goats, as well as wild animals, would breathe an aerosol of this agent. This might, then, infect animals in large areas that are now relatively free of such a disease.

Dr. Fothergill is scientific advisor to the U. S. Army Biological Laboratories, Fort Detrick, Frederick, Md. He presented this paper in Austin in February, 1963, at the Stateline Course on Medical Aspects of Civil Defense.



A specially designed program of research could provide much useful information concerning this total ecological threat. There are several experimental approaches to this problem. It would be useful to determine the susceptibility of a variety of wild faunal species, obtained by live trapping, to a variety of microorganisms through aerosol exposure. The behavior of the agent in such hosts should be studied in detail if infections become established. This would be especially important with those organisms that are normally associated with vector transmission.

Antifood Biological Warfare

So far, the broad principles of biological warfare, particularly antipersonnel, have been considered. Some of the concomitant problems in relation to domestic animals have been mentioned. It should be pointed out and, indeed, emphasized that specific agents may be used for the reduction of agricultural crops and domestic animals; in other words, antifood biological warfare. The importance of food, especially during war, needs no emphasis. In addition to providing food, some crops are of critical importance in other aspects of the economy. The fiber crops, cotton and hemp, are examples. Some industrial chemicals, such as alcohol, rubber, and certain oils, are produced from agricultural crops. Certain pleasure-producing crops, such as tobacco, tea, coffee, and various herbs and spices, should be mentioned. A number of important drugs, such as digitalis, opium, and quinine, are obtained from specific crops. Domestic animals contribute a fair share to the overall economy. In addition to meat, dairy, and poultry products, the animal industry provides draft power, transport in undeveloped areas, fertilizer, leather, wool, glue, gelatin, and necessary pharmaceutical products. A critical item in considering defense is the embryonated egg and its use in the preparation of various viral and rickettsial vaccines.

Biological anticrop agents would be extremely damaging if used in a large area. Agents would be selected for their capacity to propagate. Experience with natural epiphytotics indicates that immense crop areas may be affected. The spread of stem rust of wheat up the Mississippi Valley and the Great Plains area and on into Canada is a typical example. The famine in Ireland in 1846 and 1847, caused by a widespread epiphytotic of late blight of potatoes, is illustrative also. Blast disease has caused repeated damage to the rice crop in the Orient.

The United States is in a relatively favorable defensive position in anticrop warfare. Our unusual agricultural position is that overproduction is a major problem. As a result, we have several crop years of most major products in storage. This may be a valuable national asset. Our agriculture, moreover, is diversified and biological agents are highly specific. Actually, countries that depend for climatic, agronomic, or traditional reasons, on a single crop are the most vulnerable.

Our position with respect to antianimal agents is more critical. Our animal populations are highly susceptible to the major plagues of livestock. Introduction of these agents could be very serious.

This, then, is a general picture of the threat of biological warfare. It is hoped that it will serve as a framework upon which defensive thinking and planning can be built.

Early Warning

One of the most difficult problems in defense is detection or early warning. Biological clouds are invisible, odorless, and tasteless. The importance of immediate warning is that it may permit certain physical defensive actions. The respirator, for example, affords excellent protection to the respiratory tract if it is available and can be put on in time. For civilians, a respirator has been designed which can be produced at relatively low cost. Early warning may permit timely entrance into collective shelters, should they exist. It is possible to design quite efficient structures for this purpose. Progress is being made on methods and techniques for rapidly detecting unusual concentrations of particulate matter in the air.

A related problem is rapid specific identification of the particular agent. This is important as a guide to immediate medical care and treatment. The ordinary methods used in the diagnostic laboratories are far too slow. Identification of viruses and rickettsiae is especially tedious. Progress is being made in this field.

An adequate epidemiologic intelligence service and network is important. An unusual occurrence of disease in a particular location may be the first warning of a BW attack, particularly if it is of sabotage origin. The prompt recognition and reporting of such episodes is essential in order that all necessary actions may be taken to contain or limit the spread of the outbreak.

Prompt recognition and reporting is important in anticrop and antianimal BW also. It is especially important in the latter case. The rapidity of spread of an animal disease is dramatically illustrated by the recent epizootic of vesicular exanthema. This viral disease of swine, long confined to California, appeared in Grand Island, Neb., in June, 1952, among hogs that had been shipped from Cheyenne, Wyo., where they had been fed garbage from a transcontinental train. The malady had spread to 40 states within a year.

There must be available an organized network of laboratories having the qualifications and equipment necessary to recognize and identify unusual or exotic agents. Such services are urgently needed in the viral and rickettsial fields. The personnel in such laboratories should be trained and indoctrinated in the unique features of BW, including the use of new detection devices and procedures for rapid identification of agents, especially in specimens obtained from the environment where contamination is extensive.

Defense

One of the most important of all defenses is immunization. A number of effective immunizing materials are already available for some infectious diseases. On the other hand, there are a number of potential BW agents against which there is no method of effective immunization. There are many instances, also, where the value of the immunizing material is questionable, and improvement must be sought. It is imperative to encourage all research in the development of new, or improving existing, methods of active immunization.

The administrative problems of immunizing large populations against a number of agents are enormous. This, too, is an area where research should be fruitful because simplified methods for rapid, mass immunization are essential. Considerable effort is being devoted to the development of combined or multiple vaccines with some success. The Russians[33] have reported another approach to this problem. They have exposed human volunteers by the aerosol route to live, attenuated agents of anthrax, plague, brucellosis, and tularemia. Groups of people were exposed simultaneously in a small room. The efficacy of the procedure was determined by the subsequent demonstration of various positive immunologic reactions. There are some limitations to this technique of mass immunization. It may not allow for the medical screening of persons who might pose a special risk. Individuals with certain types of chronic pulmonary conditions, for example, might react unfavorably.

Finally, one must emphasize the need for detailed planning for the care and treatment of mass casualties. Such planning must be done on an individual community basis since it will depend on the availability of facilities, resources, and trained or professional personnel.

A complete reference list is available from the author.

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Medical Effects Of Nuclear Weapons

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SYMPOSIUM

"NUCLEAR WEAPONS and the possibility of nuclear war are facts of life we cannot ignore today," the late President Kennedy said more than two years ago. Since physicians are involved, a general knowledge of the types of injuries produced by nuclear weapons, and the mechanism of production, is necessary to appreciate that injuries can be minimized, and that survival in the nuclear age is possible.

In nuclear warfare large numbers of casualties will be produced within a very short time. During the early post-impact period, there will be a wide disparity between the number of patients requiring treatment and available medical resources. Early treatment will be unlikely, evacuation will be slow or nonexistent, and the remaining medical installations will be overwhelmed. What can be done to meet this challenge? There are three possible approaches to the solution.

1. Increased emphasis must be placed on training nonmedical personnel in first aid so that they can have maximum self-aid and can give first aid to others. A good illustration of the need for this type of training is the Worcester, Mass., tornado, which injured about 1,500 people. Less than 10 per cent reached the hospitals with any evidence of first aid having been administered. If this is what happens in a disaster of this magnitude, what can be anticipated in handling a disaster such as experienced in Hiroshima—70,000 people injured almost instantaneously?

2. Another approach is to increase instruction of all medical personnel in the principles of the management of mass casualties. They must know what they are up against and why certain compromises must be made in the care and treatment of the sick and injured.

3. The most logical and sensible approach to this problem is to increase emphasis on prevention. This is the only way to keep casualties to a level at which they might possibly be manageable. The medical profession would make a far greater contribution to solving the medical problem from nuclear warfare if more of our efforts were devoted to prevention than to treatment.

If casualties are to be minimized, it is essential to understand the effects of these weapons.

When a nuclear weapon is detonated tremendous energy is released in a short time in a limited space. This energy is distributed in the form of blast, heat, and nuclear radiation.

About half of the energy is in the form of blast, 35 per cent in the form of heat and light, and 15 per cent in the form of radiation (Fig. 1).

Associated with each of these physical effects of a nuclear detonation, some type of injury can be expected. From blast, mechani-

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Nuclear weapons, like conventional bombs, cause the familiar injuries associated with blast and heat, but nuclear bombs produce overwhelming numbers of these casualties. Nuclear radiation is the unique feature involved; physicians must understand it clearly. Casualties can be greatly reduced through proper preventive measures.

Review of this paper does not imply Department of Defense indorsement of factual accuracy or opinions stated therein.

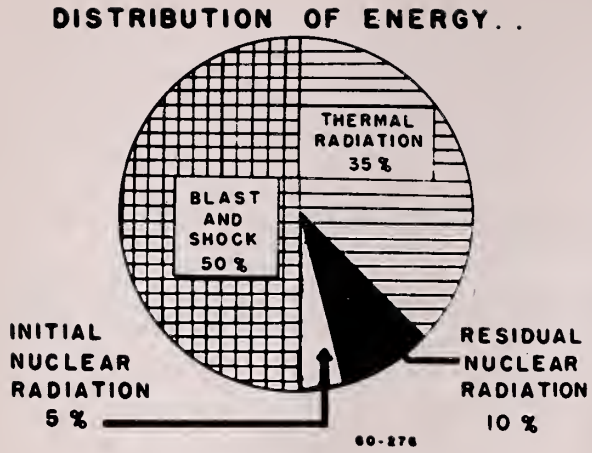

Fig. 1.

cal injuries such as fractures and lacerations are, on the whole, similar to those caused by conventional bombs. The important difference is the larger number and variety of injuries suffered in a short interval of time in the case of a nuclear explosion. From thermal radiation, flash and flame burns, retinal burns, and flash blindness or dazzle are produced. Burns may be the most serious problem from the standpoint of demands placed on the medical resources. The requirements are staggering. As an example, the Cocoanut Grove fire in Boston, Mass., in 1942 resulted in about 500 severely burned patients. Despite the excellent quality and the quantity of Boston's medical facilities, these 500 patients taxed severely not only the facilities of this major city but those of the surrounding communities. A few passages at random from "Hiroshima Diary," the journal of a Japanese physician, will convince anyone of the seriousness of the burn problem after a nuclear attack. Those who have treated seriously burned patients report that ten, admitted simultaneously to any large hospital, will almost completely disrupt its function. Thus the problem of handling 35,000, as experienced at Hiroshima, appears to be almost hopeless. Furthermore, when one considers that the weapon detonated over Hiroshima was roughly 20 kilotons, and today one talks glibly about weapons in the megaton range, the catastrophic nature of the problem becomes obvious.

From ionizing radiation, the only effect which is completely absent from conventional explosions, deep cellular and tissue injury and beta burns are produced.

One of the difficulties in assessing the importance of injuries of various types lies in the fact that from a nuclear detonation there is a compounding effect, or the superimposing of one injury on another. Many people at Hiroshima and Nagasaki were not only injured by blast but were also burned and irradiated.

Blast Injuries

What is blast? Blast is a wave of compressed air that travels at approximately the speed of sound. At the instant of detonation, a fireball is formed containing gases at extremely high pressure and at temperatures approaching those in the center of the sun. The fireball expands so rapidly that a layer of highly compressed air is formed on its surface. As the fireball begins to cool and its rate of expansion slows up, the blast wave breaks away from the fireball and moves outward in all directions. The strength of this blast wave is measured in pounds per square inch of overpressure (psi); that is, pressure above normal atmospheric pressure. This blast wave can injure people in two ways—directly or indirectly. Direct or primary blast injuries result from the blast wave literally wrapping itself around the individual and squeezing him so as to cause damage to the lungs, stomach, intestines, and eardrums. Indirect or secondary blast injuries result from buildings collapsing and from the great quantities of debris thrown about by the winds that accompany the blast wave. In addition, the winds can pick up an individual and hurl him against solid objects. This is often called the translational or tertiary effect of blast.

Extremely high overpressures are required to cause severe injury or death. As a frame of reference and a "feel" for blast effects, 5 psi will completely destroy the average American home.

With this in mind, it would appear that much less overpressure would be required to severely injure or kill a man. This is not the case however. Tests indicate that 5 psi might rupture a man's eardrums. Fifteen psi is probably the threshold for lung injury, and 30 to 80 psi the range in which severe injury or death might occur, although people have been known to survive short duration overpressures of about 200 psi. Generally, if one is in the zone where overpressures are sufficient to produce severe direct blast injury, he is simultaneously lethally burned and leth-

ally irradiated. For a 20 KT airburst, for example, 30 psi are experienced about .38 miles from ground zero. Here, the thermal energy would be measured in the hundreds of calories and the nuclear radiation in the thousands of roentgens or rad.

A possible exception could occur in an inadequately designed shelter which provided shielding from radiation and thermal injury but which permitted entry of blast waves or actually caused reinforcement or amplification of blast that entered. The important point is that man is relatively resistant to blast, and it is doubtful that we will see many living persons with direct blast injuries from a nuclear disaster.

More important than direct blast injuries are the indirect or secondary effects of collapsing buildings, missiles flying through the air, and the possible physical displacement of man by the blast winds. In Hiroshima and Nagasaki, most people were hurt by flying objects, or were crushed or buried under buildings. Very few survivors were injured by being hurled forcibly against solid objects.

Complete protection from blast is virtually impossible; however, much can be done to reduce the number of casualties. The most effective single measure is blast-resistant underground shelters. If unavailable, protection should be sought in the strongest building available. Individuals should stay away from windows and easily breakable materials, such as plaster walls or ceilings. In the collapse of buildings, heavy materials will fall or be hurled about. There is a dual hazard of being hit and trapped; therefore, positions next to walls in basements offer the best protection. Above ground the safest locations are generally near, but not against, walls and away from doors and windows. In the open, some protection may be obtained by falling prone, with the head directly toward or away from the explosion, and remaining in that position until the blast wave has passed.

Burns and Thermal Injuries to Eyes

Fire has been a weapon since earliest historical times. Everyone is familiar with burn injury, but the development of nuclear weapons has introduced thermal injury on a scale far exceeding anything known to man.

To reduce the number of burn injuries, we must first understand the characteristics of thermal radiation in order that effective passive defense measures can be taken. If we know what we are up against, we are in a better position to act intelligently and effectively.

The principal characteristics of thermal radiation are: (1) travels at the speed of light, (2) travels in a straight line, (3) has very little penetrating power, (4) can easily be absorbed or attenuated, (5) can be scattered and reflected, and (6) emission time increases with yield.

Since thermal radiation is transmitted at the speed of light, it begins to strike the individual instantaneously with the detonation. For tactical yield weapons in the kiloton range, there is virtually no time for an individual to take protective measures or evasive action. For weapons in the megaton range, it takes appreciably longer for the thermal energy to be emitted, so that an individual taking evasive action within a second or two of the appearance of the fireball might be able to reduce the severity of thermal radiation injury.

Unless scattered, thermal radiation from a nuclear explosion, like ordinary light in general, travels in straight lines. Any solid, opaque material such as a wall, a hill, a tree, a fold in the ground, or a vehicle between the individuals and the fireball will act as a shield and provide considerable protection from thermal radiation. Even one layer of a light colored cloth between the skin and the fireball may spell the difference between no burn and a serious second or third degree burn. Fog, rain, or smoke will reduce or stop thermal radiation. Some radiation can be absorbed or scattered by dust, or by other solid impurities suspended in the air. Conversely, it can be reflected by clouds or the ground, thus increasing the amount received by an individual. These characteristics of thermal radiation should be kept in mind when considering shielding. A shield which merely intervenes between a given target and the ball of fire but does not surround the target may not be entirely effective under hazy atmospheric conditions. A large portion of the thermal radiation received, especially at considerable distances from the explosion, will be scattered and will arrive from many directions.

Personnel are extremely vulnerable to the effects of thermal radiation. They can be burned by the heat and dazzled by the light. The more important of these effects is the heat. The quantity of heat or thermal en-

ergy delivered is expressed in calories per square cm. Heat from a nuclear explosion is delivered extremely fast, and causes rapid temperature rise in the skin (or anything else) it strikes, causing burns or charring or ignition of some materials.

Burns, irrespective of their cause, fall in the familiar first, second, and third degree classifications. The amounts of heat required to produce burns from three ranges of weapons are shown in Table 1.

TABLE 1.—*Calories Per Square Cm. Required to Produce Burns from Three Weapon Yields.*

Yield	Burns on Bare Skin— 1st Degree	2nd Degree
1 KT	2	4
100 KT	2.5	5
10 MT	3.5	9.2

(No differentiation is made between second and third degree, since an extensive second degree burn is just as incapacitating as is a third degree burn.)

Notice that as the yield is increased, the total calories which are required to produce a specific burn are increased. In a 1 KT weapon the maximum radiant or burning power is reached in approximately .03 second, while in a 10 MT weapon it is reached in about 3 seconds. During this longer period of time for delivery of the thermal energy, the skin can dissipate some of the energy by reflection, convection, and other mechanisms. Further, there is time to take evasive action. To illustrate the importance of rate of delivery of thermal energy, consider the effect of the sun on an individual. The heat from the sun on a clear day measures about .032 calorie per square cm. per second. If an individual were to remain outside for 1,000 seconds, or about 17 minutes, he would receive 32 calories per square cm. Theoretically, this amount should burn one to a crisp; yet delivered in 17 minutes it does not. If received in one second, the results would be quite different.

The vulnerability of personnel to thermal radiation is indicated in Table 2, which shows the approximate ranges from ground zero on a clear day over which second degree burns may occur to exposed personnel from three weapon yields.

It is of interest that the diameter of the fireball for the 10 MT weapon is about 3.5 miles with complete destruction within this range. Persons unable to take evasive action

TABLE 2.—*Radii of Thermal Damage—Second Degree Burns to Exposed Personnel (Low Air Burst).*

1 KT	100 KT	10 MT
.5 mi. (4 Cal./cm.²)	4 mi. (5 Cal./cm.²)	24 mi. (9.2 Cal./cm.²)

would sustain second degree burns on unprotected skin (generally the face, hands, and legs) at a distance of approximately 24 miles from ground zero (9.2 Cal./cm.²). At about 21 miles, clothing might ignite and cause severe flame burns (13 Cal./cm.²). With light colored, loosely fitted, multiple layers of clothing, the amount of thermal energy required to cause flame burns would be much higher.

Two general types of burns result from a nuclear detonation—flash burns caused by the absorption of radiant energy by the skin, and flame burns just like those caused by any fire. The distinguishing feature of a flash burn is the sharp limitation to exposed areas of the skin which were in direct line of sight with the fireball (Fig. 2). Although flash burns are confined largely to exposed parts of the body, such burns can occur through clothing, especially in areas where clothes are tightly drawn over the skin, at the elbows and shoulders, for example. (Such burns may result from contact with the hot fabric rather than from the direct effect of thermal radiation; this is especially true with black clothing which absorbs the radiation, becomes hot, and thus causes contact burns.)

Typical flame burns occur when the clothing is ignited. Flame burns are in general more severe than flash burns.

The treatment of severe thermal injury on areas in excess of 20 per cent of the body surface represents a grave medical problem, even in a modern hospital under the best of circumstances. Consider the materials and personnel required in the actual treatment of one patient with a 40 per cent, second degree, moderate temperature burn: 42 tanks of oxygen, 36 pints of plasma, 40 pints of blood, 2 physicians, 104 pints of fluids, 2.7 miles of gauze, 3 nurses, and mountains of drugs. This does not include the requirements for subsequent hospitalization for corrective measures. Furthermore, many patients do not survive despite such treatment.

The requirements to treat the 35,000 se-

Fig. 2. Flash burns occur on unprotected parts of the body. Note the sharp demarcation between trunk and upper arms, protected by clothing, and burned thighs and forearms.

verely burned casualties of Hiroshima would be staggering. Somewhere in the neighborhood of 8,000 tons of medical supplies and 175,000 doctors and nurses would be needed. Just to move the supplies would require approximately 160 freight cars, or 2,000 two-and-a-half ton trucks. It would tie up approximately 30 per cent of all the doctors and 20 per cent of all nurses in the United States. Obviously, the logistical requirements for handling mass burn casualties cannot be met with our presently available resources. Limited treatment facilities, inadequate supplies, and a shortage of medical personnel will prevail and necessitate a great deal of sorting in order to provide no disservice to the many in favor of a few. Many burns may be inadequately treated during the early post-impact period, with infection as the inevitable consequence. In Japan the formation of keloids after the healing of the more severe burns was seen (Fig. 3). The degree of keloid formation undoubtedly was influenced by infection, malnutrition, and the known predisposition for keloid formation among many individuals.

Thermal injury to the eye warrants consideration in view of the extremely long distances over which damage can be sustained. It may be divided into flash blindness (dazzle) and retinal burns. Flash blindness is a temporary condition caused by the brilliant flash of nuclear detonation, which is many times brighter than the sun. It is not necessary for one to look at the fireball to develop this condition. The eye is flooded with brilliant light scattered from the sky, ground, and other surroundings. Dazzle from a burst during daylight hours does not persist for

Fig. 3.

more than about two minutes; however, at night when the pupil is dilated, it may last from a few minutes to many hours and can seriously reduce effectiveness.

Retinal burns are produced by looking at the fireball. Macular burns can be produced by looking directly at the fireball; burns elsewhere on the retina may occur if the fireball is within the field of vision. The lens can focus enough energy to produce a burn on the retina at such a distance from the explosion that the thermal radiation intensity is too small to produce a skin burn. Permanent scarring and interference with vision may occur, and the fireball image may be permanently etched on the retina. Again, this is more likely to occur at night and is greatly intensified by the use of telescopes or binoculars. A few retinal burns have been experienced at a distance of 10 miles from a 20 KT explosion. On an exceptionally clear day, retinal burns can occur up to 36 miles away in daytime and 40 miles away at night from a 20 KT weapon. With megaton weapons detonated at high altitudes (over 100,000 feet), minimal retinal burns can be produced on the ground at distances over 200 miles. Lack of atmospheric attenuation and the rapid rate at which the thermal energy is delivered ac-

counts for this. Reflex actions, such as blinking (100-150 milliseconds) and contraction of the eye pupil, give only limited protection. Although retinal burns are a distinct possibility, the chances that an individual will be looking directly at the ball of fire are small, particularly for low-yield nuclear weapons which have a short period in which the rate of thermal radiation emission is high.

Among the survivors in Hiroshima and Nagasaki, eye injuries directly attributable to thermal radiation were relatively rare. There were many cases of temporary flash blindness, but more severe eye injuries were not common.

The overwhelming magnitude of the problem of burns makes it imperative that a major portion of our efforts be directed to prevention. With advance warning, proper clothing discipline, adequate shelters, and knowledge of the characteristics of thermal radiation, casualties can be reduced to a manageable level.

Ionizing Radiation

Since we must live with ionizing radiation, it is essential that we learn as much about it as possible. Man has always lived with radiation. We literally live in a sea of it—in the air, in the earth, in our bodies. Actually there is nothing new about it. From natural background sources, we receive approximately .15 rad per year. No one questions that radiation is harmful to man, but so are many other things we live with. Persons who show extreme fear of radiation should be reminded of this. Gasoline is a good example. It cannot be consumed by a human being without dire results. It is an extreme fire and explosion hazard. High octane aviation gasoline will severely burn the skin on contact. And fumes from a gasoline engine are lethal. Man has learned not only to live with gasoline, but also to use it to great advantage. The same general principles apply to radiation.

Each of us soon will be directly or indirectly concerned with nuclear radiation. It is being used more often for diagnostic and therapeutic purposes. Fallout from nuclear tests has contaminated a major portion of the earth's surface—the level of contamination, of course, is very low. Nuclear reactors are being built in many countries for power, and some contaminated wastes are discharged into the soil, water, and atmosphere. Industry is using more and more radioisotopes. Whether we like it or not, it is here to stay.

The radiation liberated by nuclear detonation can be divided into two categories—prompt and residual. Prompt radiation is generally defined as that emitted from both the fireball and nuclear cloud within the first 60 seconds after the explosion. Residual radiation is that which is emitted later than one minute after the burst, and associated with fallout and neutron-induced activity.

There are four types of nuclear radiations with which we are most concerned. They are alpha particles, beta particles, gamma rays, and neutrons.

The effects of radiation harmful to man appear to be caused by the ionization produced in the living tissue cells. As a result of ionization, some of the constituents, which are essential to their normal functioning, are damaged or destroyed. If enough cells are thus affected in too short a time, the individual becomes sick and the clinical picture that develops is described as the acute radiation syndrome.

The response of an individual to nuclear radiation depends upon several factors, some of which are dose, time, individual biological susceptibility, extent and part of body exposed, and type of radiation.

Dose.—Following are approximate radiation dosages that may be used as a rough guide in predicting the effects of acute, whole-body irradiation:

0-100 rad—No clinical evidence of disease.
100-200 rad—Minor complaints of nausea, usually not incapacitating.
200-1,000 rad—Incapacitating. Hospitalization for vast majority.
1,000-5,000 rad—Fatal within weeks.
over 5,000 rad—Fatal within hours.

Time.—The effects of nuclear radiations on people depend not only on the total dose, but also on the rate at which it is received. As an extreme example, 1,000 rad received within 24 hours would be fatal if the whole body were exposed, but it would not cause death or have any noticeable external effects if received more or less evenly over a period of 30 years.

Individual Biological Susceptibility.—It is impossible to predict exactly how a given in-

dividual will respond to a specified dose of radiation, although the expected average effect on a large group may be known if the conditions are precisely defined.

Extent of Bodily Exposure.—The injury caused by a certain dose of radiation depends upon the extent and part of the body that is exposed. For example, 700 rad applied to a small region of the body may result in considerable biological damage to the irradiated area, but the overall health of the individual may be apparently unaffected. When the exposure is restricted, the unexposed region can contribute to the recovery of the injured area. If the whole body is exposed, many organs are affected and recovery is much more difficult.

Type of Radiation.—Although the effects on the body of alpha and beta particles, gamma rays, and neutrons are qualitatively alike, the various radiations differ in depth to which they penetrate and in the degree of injury.

Biological Result To Be Measured.—Although acute injury from ionizing radiation was noted soon after the discovery of x-ray and naturally radioactive elements, more

Colonel Marks presented this paper during the Stateline Course on Medical Aspects of Civil Defense held in Austin in February, 1963.

subtle, delayed effects were not detected until much later. These delayed effects are of prime importance in determining permissible human exposure and they should not be ignored even in wartime insofar as possible. Delayed effects include increased incidence of leukemia, increased incidence of malignant and benign tumors, cataracts, probable genetic effects, and possible shortening of life span.

The foregoing factors considerably influence the response in the irradiated victim. The variables are mentioned not to add confusion to a difficult problem, but rather to acknowledge their importance in arriving at a departure point for describing this syndrome.

The acute radiation syndrome can be defined as that series of clinical events which follows whole-body exposure to penetrating ionizing radiation received in significant dose in a short time. The syndrome is in three forms—hematopoietic, gastrointestinal, and cerebral, the names used to indicate the organ or organ systems involved. Since the doses required to produce the two latter forms are so high (over 1,000 rad) and the prognosis hopeless, only the hematopoietic form is discussed here. It is amenable to treatment and doses range from 200 to 1,000 rad.

Following a dose of several hundred rad, the clinical picture proceeds along a highly predictable, time-defined course of four phases, prodromal, latent, bone marrow depression, and recovery (Fig. 4).

Prodromal Phase.—Even when several hundred rads of penetrating radiation are accumulated in less than one hour, there are no sensations or reactions for one or two hours; then, rather suddenly, listlessness and fatigue appear. More and more, the patient becomes quiet, withdrawn, and asocial. He complains of a headache, dullness, and dizziness, and frequently becomes mentally depressed. At about this same time, he complains of upset stomach and loss of appetite, soon followed by vomiting, until he reaches a climax between five and eight hours after exposure. These symptoms subside on the second or third day, at which time most patients exhibit no symptoms.

On the third day, the patient not only feels well but routine laboratory findings disclose

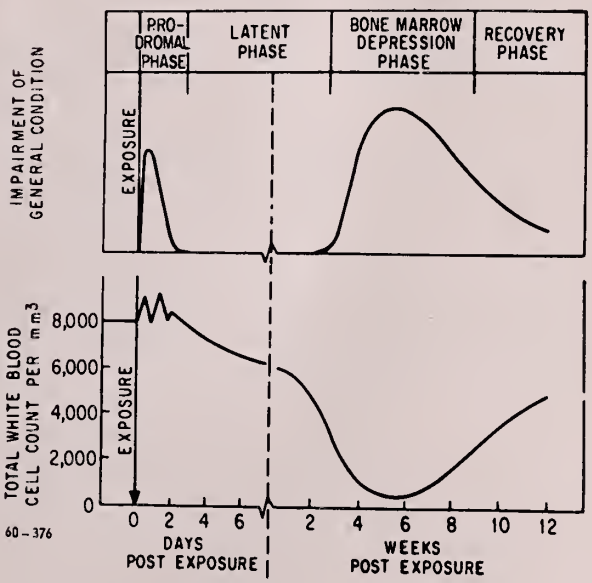

Fig. 4. This chart illustrates the four phases of the acute radiation syndrome.[1]

no abnormal data other than a decrease in the number of white blood cells. This is the beginning of the latent phase.

Latent Phase.—From about the third to the twentieth day after exposure the patient feels well, is ambulatory, and can even perform strenuous physical exercise. Between the 13th and 17th days, this innocuous picture is somewhat disturbed by an ominous sign—loss of hair. In the morning, the patient finds the bed sheet covered with body hair; on combing, he notices easy uprooting of hair in large bundles. Loss of hair usually is associated with exposure to 300 rad or more. To an informed person, this is alarming although he still may feel perfectly well. On about the 20th or 21st day, overt clinical symptoms begin to erupt once again. The patient now enters the bone marrow depression phase.

Bone Marrow Depression Phase.—The patient first experiences chills, malaise, a feverish feeling, headache, and fatigue. Soon his condition so deteriorates that he must stay in bed. The picture is soon aggravated by reddening and swelling of the throat, a tendency to bleed, high fever, and susceptibility to infection; this reaches a climax between four and six weeks after exposure. This phase is caused by the radiation damage to the blood-forming tissues, with the resultant decrease in blood cell production.

Recovery Phase.—Between eight and ten weeks after exposure, a vast majority of the patients are sufficiently recovered to require only convalescent care, to include nothing but rest and good food supplemented by vitamins, iron, and other measures helpful for complete restoration of the blood-forming system. After about one month of convalescence, the patient can resume his normal activities.

Summary

An understanding of the medical effects of nuclear weapons is a responsibility of all medical personnel in view of the important role they will play in a nuclear disaster. The types of injuries produced by blast and thermal radiation are nothing new to the medical service. The problem we face is the instantaneous production of tremendous numbers of injured people with the resultant disparity between needs and resources.

The only unique feature of nuclear weapons effects is ionizing radiation. To understand this hazard requires a clear appreciation of the types of radiation produced and their characteristics.

The most intelligent approach to the medical problem is prevention, and only by understanding the effects of nuclear weapons can we prevent injuries.

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Life Reports Hazards Of Junior League Football

Physical and psychological hazards of league football for preadolescents are reported in a recent *Life Magazine* article (Nov. 15).

While league football is said to be preferable to unsupervised sandlot football because rules can be enforced to keep the game as safe and sensible as possible, adult-run games are believed by many to cause serious psychological damage to the players.

In sandlot football, a kid can "pick up and go home," but in a league game he must remain in competition regardless of how bored or afraid he may be.

Of course, there are the parents who drive their children to high performance and "thus expose them to a rat's nest of psychological horror." In an extreme case, one child who felt he had disappointed his father by not making starring football plays, tried to attract attention otherwise by stealing and committing arson. He is now in a mental hospital.

The Westchester County (New York) Medical Society [among others] opposes football for preadolescents because these children are extremely vulnerable to injuries to the soft bone tips.

ANESTHESIA and Problems of Mass Casualties

CAPT. JOHN W. COURSEY, USAF, MC, and ROY D. WILSON, M.D.

SYMPOSIUM

THE SPECTER of nuclear explosion is constantly with us, and there is always the possibility of natural disasters and industrial accidents. Any of these could produce many casualties in a short period of time and greatly tax the medical facilities fortunate enough to escape destruction.

It has been estimated that, if a ten megaton nuclear bomb were exploded in the center of a city of 1,000,000 people, the area of destruction including damaged houses and blocked streets would extend for a radius of 15 miles. It is estimated that there would be approximately 210,000 immediate casualties excluding the dead. Of these 210,000 casualties, 60,000 persons would be only slightly injured and 150,000 would be seriously injured. Of the seriously injured persons, 100,000 would be trapped; some of these would be released quickly, dead or alive, and others would be discovered during a period of several days. The 50,000 untrapped casualties remaining would require immediate aid.[1]

The military disaster problem is more complex because of the importance of the military mission. Military triage under wartime conditions, in contrast to civilian or peacetime military handling of disaster victims, employs a strict method of sorting, aimed at returning to duty as quickly as possible the greatest number of injured personnel.[3, 11]

In civilian disasters, resulting from natural causes or industrial accidents, triage should also be employed. In past civilian disasters, casualties usually have been treated in the order they arrive at the medical facility. However, in 1956, the British Royal Army Medical Corps, working with civilian authorities, demonstrated that some system of casualty sorting forward of the hospital area is essential.[11] Otherwise, the medical facilities quickly become "swamped" and chaos results.

Anesthesia Problems

Types of Patients.—An anesthetist confronted with mass casualties will find several types of patients who require immediate aid. If there is no triage, the types of patients will range from physical status 1E to physical status 5E, based on the classification recently adopted by the American Society of Anesthesiologists. These patients will have injuries resulting from blast,[9] including shock, pulmonary edema, and pulmonary hemorrhage. The presence of pulmonary edema and pulmonary hemorrhage will complicate the anesthetist's problems considerably. In addition to the blast injuries, many casualties will have immediate radiation reactions similar to those from any early burn and probably will be in compensated shock. A tremendous number of persons will be subjected to traumatic injuries resulting from falling and flying debris. Others will have multiple areas of laceration, contusions, and fractures. Many will suffer intracranial insults in addition to injuries in other areas of their bodies.

A large number of the injured will suffer from hysteria[9] which will require immediate treatment before definitive measures can be taken to remedy their other injuries. This

Immediate postdisaster conditions are described. Logistical support and medical considerations regarding anesthetic apparatus and agents are outlined. Also reviewed are requirements, qualifications, and availability of personnel for administration of anesthesia to disaster victims.

will place an added strain on an already overtaxed medical staff because the use of sedatives may compound these patients' problems. Finally, it must be realized that many casualties presented for help will be suffering from combinations of injuries, including pre-existing medical problems.

Resuscitation Efforts.—Therefore, the anesthetist must first consider the physical status of his patient and then plan on preanesthetic treatment. Of great concern will be whether intramuscular or subcutaneous narcotics have been given before the patient reaches him. After shock has been overcome, subcutaneously injected narcotics will be picked up by the restored vascular system. This may be dangerous if repeated doses are given to patients continuing to suffer from pain.[9] The use of sedatives to combat hysteria may compound the anesthetist's problems, especially if excessive doses are given to a person who has been injured.

Of great concern to the anesthetist will be the volume and type of fluids given to the patients, that is, blood, plasma, plasma expanders, glucose, or sodium chloride.

The lack or availability of fluids may, in itself, prove to be a formidable problem. The availability of whole blood will be limited if not entirely lacking and the use of plasma expanders will become a necessity. In addition, the anesthetist must be wary of the hypovolemic patient to whom vasopressors have been administered; the patient may give the mistaken appearance of having no great cardiovascular problem.

Pre-existing Problems.—In addition to the problems already mentioned, the anesthetist at the scene of mass casualties must consider the usual pathophysiologic conditions common in emergency cases. These will include patients with full stomachs; patients with compromised airways resulting from fractures of the facial bones, mandible, or collapsed tracheae; unconscious patients of whom little or nothing is known; hysterical or otherwise unmanageable patients; and patients suffering from multiple injuries. Of grave concern will be those patients who have existing serious medical illnesses and who require an anesthetic. Consideration must be given to the effects of previous drug therapy when selecting an anesthetic technique for such patients. Female patients who are pregnant or in labor and present themselves for aid must be accepted and their presence will add to the confusion.

Patients with pulmonary disorders will constitute a tremendous problem. Blast injuries, including pulmonary edema, pneumothorax, hemothorax, hemopneumothorax, flail chest, and sucking wounds of the chest, added to such pre-existing diseases as pneumonia, asthma, chronic bronchitis, emphysema, or fibrosis will greatly complicate matters. In addition, the anesthetist will be confronted by disaster victims in the pediatric and geriatric groups, each one with particular problems.

Premedication.—Finally, after hurried but critical analysis, the anesthetist must consider premedicating drugs in his anesthetic management.

The seriousness of all this can be fully appreciated when one thinks of the great number of casualties to be treated in the limited amount of time by personnel working long hours.

Logistical Problems

Stockpiling.—In any consideration of mass casualties, the problem of logistical support is of paramount importance.[3, 6, 9] Perhaps the greatest single problem is the recognition that there is a problem. Next in importance is the follow-through with adequate presupply. In the civilian approach, the transportability of stored material is of less consequence because items can be stockpiled, even if they are bulky, heavy, and in great variety. Civilian and military authorities alike are confronted with the high initial cost of materials to be stockpiled.

The civilian planners can count on some immediate support from neighboring areas that escape damage if prior planning at a high enough level has provided for a dovetailing scheme of strategic placement of supplies.[8] Not only will material be available, but personnel from adjacent areas will be able to move into the disaster area to render aid.

Thus, a concerted effort must be made to (1) evaluate what is needed, (2) obtain it, and (3) stockpile it strategically. This necessitates close, top-level civilian and military planning. Inasmuch as many hospitals may be rendered ineffective, schools, meeting halls, or any large-area facilities may have to be utilized as disaster hospitals. With this

in mind, strategic stockpiling of material for possible use in these areas should be considered. Not to be forgotten is the necessity for constant inspection and turnover of the stockpiled material because some items deteriorate.[10]

Resupply.—Having solved the presupply problem, the planners must face the additional problem of resupply, especially if many areas of the nation are devastated. The existing stockpiles will vanish, and the problem of resupply will become vital. In this regard, transportation and communication obstacles will undoubtedly arise and magnify existing problems.[3] The only logical solution is stockpiling of material in relatively uninhabited areas that will probably be spared initial destruction. The Canadian government has already accomplished this in 200 remote areas. Provided adequate transport facilities are available, the devastated area will be able to call upon this regional stockpile for resupply. At best, resupply will prove to be formidable. Therefore, initial supply should consist of a maximum of essential items.

Agents and Equipment.—Perhaps of greatest importance to the anesthetist is the basic question of exactly what to stockpile. This question is dependent upon the final decision as to what agents and what techniques will be used. Work continues along these lines although no universal decisions are yet available.

The military services have endeavored to meet the disaster problems by the establishment of transportable hospital units. The Army has its Surgical Hospital, Mobile, Army (SHMA), and the Air Force has its Tactical (TAC) hospitals. These self-contained units are organized for quick air movement into required areas. By being air-transportable, these military hospitals demand small, light-weight, efficient equipment capable of withstanding rough handling. Some localities in the United States have organized, supplied, and dispersed civilian units, such as the 200-bed Federal Civil Defense Agency (FCDA) emergency hospital. Such is the Gulf Coast region, where the Texas City disaster and several hurricanes prodded the communities into action. As mentioned, Canada has designated, and is stockpiling, a number of widely separated stand-by emergency hospitals throughout that nation. The State of New York has purchased a number of ether-air drawover vaporizers, the Epstein, Macintosh, and Oxford (EMO) inhaler, and has distributed them throughout the state. The military problem is compounded by need for minimal weight and volume without sacrifice of effectiveness or safety.

USAF Captain Coursey and Dr. Wilson, at that time also in the Air Force, presented this paper at the TMA 1963 Annual Session. The two men were stationed then at Wilford Hall USAF Hospital where the paper was prepared after three and one-half years' research.

Medical Considerations

Equipment.—Having resolved the problem of logistics, the planners must evaluate the anesthetic apparatus with respect to the adaptability of the vaporizer to various agents and various techniques. Probably a simple non-breathing air-drawover vaporizer may prove to be the most feasible. Whatever its final design is, the vaporizer must be physiologically sound but simple enough to keep the cost low.[7, 10, 12]

Agents.—Much has been written concerning the agents or techniques to be used with mass casualties. Some advocate general anesthesia using cyclopropane, ethyl ether, or nitrous oxide and oxygen with muscle relaxants. Others advocate subarachnoid anesthesia or block anesthesia using the local drugs. Still others advocate the use of drug combinations.[2-5, 7-12] The need for a potent general anesthetic agent for use with limited oxygen supply or with air alone is apparent. The optimal agent should be nonexplosive, easily stored, easily packaged, and stable; it should also have a boiling point which will allow its use in temperate climates in the available vaporizers. For military use, that is, in the event of war, the agent selected must be manufactured in this country and stored in this country in large quantities at all times.

Personnel Problems

Trained Personnel.—Who will be the anesthetists in such a situation? Personnel

trained in anesthesia are in great demand under normal conditions. In the event of a disaster, these trained personnel must utilize all available sources of medical and nonmedical personnel, including physicians, nurses, and others with little or no training.[3, 8-10]

There may be relatively few fully trained anesthetists available at any one locality. The present number of 10,000 fully trained anesthesiologists will be an inadequate supply in a large scale emergency. Therefore, those who are on hand will have to supervise less trained personnel to extend their effectiveness. The nurse anesthetists may also have to serve as supervisors of less skilled individuals, often in disregard of normal nurse-physician relationships. If unusually fortunate, the hospital may have general practitioners, dentists, and veterinarians who have had prior anesthesia training.

Untrained Personnel.—Many relatively untrained personnel, such as medical corpsmen and practical nurses, could be pressed into service to allow trained anesthetists to conduct several cases at one time.

Discussion

The treatment of mass casualties may face any civilian community at any time. Hurricanes, typhoons, floods, fires, tornadoes, and earthquakes occur periodically. In addition, disasters secondary to transportation mishaps, industrial mishaps, and civil commotions arise occasionally. Unquestionably the most menacing threat to every community in the world is the ever constant danger of total nuclear assault, rendering whole nations disaster areas. The latter possibility serves to magnify the fact that plans should be made well in advance and should be adequate to meet any emergency. If a nuclear war occurs, those areas that are prepared will be the areas where survival will be the greatest.

Inseparable and vital problems of mass casualties are (1) the availability of anesthetists, (2) what they can use for the relief of pain, and (3) investigations of various anesthetic equipment to administer general or regional anesthesia. No ideal solution is available at this time.

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Dr. Wilson, University of Texas Medical Branch, Galveston.

Men in blue-collar work—operatives, craftsmen and laborers—number nearly half the working men in the country, life insurance statisticians report.

Keystone to Survival

Medical Self-Help Training Program

JAY P. SANFORD, M.D.

SYMPOSIUM

THE MEDICAL Self-Help Training program is more meaningful when considered in its appropriate position as one phase of expanded health resources to meet the disparity between medical means and casualty loads in a nuclear disaster.

There are two basic approaches to planning, first, prevention of casualties, and second, management after the impact. Planning must deal both with the immediate post-impact period as well as with delayed medical problems, inasmuch as any disaster sets up a chain of medical problems. Such was well-illustrated in Japan, with an initial post-impact problem dealing predominantly with mechanical and thermal injuries, subsequent radiation injuries and illnesses associated with breakdown in sanitation control, and now the late consequences including an increased frequency of leukemia.

The problem in planning does not stem from a lack of medical knowledge, but rather is the consequence of the tremendous disparity between medical means and the anticipated casualty load. A two megaton weapon exploded in the air over downtown Dallas would generate the maximal number of injured survivors, hence can be used to define the maximal medical work load. Larger weapons would be more destructive both of people and of property; however, there would be fewer survivors to comprise a medical load. The distance from ground zero to the point of partial damage would be an estimated 9.3 miles with a two megaton weapon. Assuming ground zero to be downtown Dallas, the 9.3 mile radius would envelope a population of 717,819. Approximately 200,000 persons would be injured; however, 40 per cent of the casualties are estimated to be only minimally injured. Thus, there would be about 120,000 individuals requiring medical attention.

At this time there are 1,700 physicians, which includes those in residency training and the osteopathic physicians, in Dallas County. Most of these people maintain offices within an area of severe destruction, that is, within a radius of 4.6 miles. Based upon standard casualty estimates, about 595 physicians would survive uninjured and an additional 204 with injuries minimal enough to permit continued activity. There are 3,000 to 4,000 hospital beds in Dallas. Virtually all of these fixed hospital beds would be rendered useless. Thus, in Dallas as in most cities, there would be a greater loss of hospital facilities and health personnel than casualties in general.

From these dismal statistics, it is apparent that the solution to health-medical planning rests primarily on prevention of casualties. Casualty estimates suggest that mere warning would contribute in a major way to increasing the number of uninjured as well

Their ranks and resources greatly diminished, physicians surviving nuclear attack would face overwhelming numbers of casualties. Medical Self-Help Training is one of several ways to expand resources to give the most people the best care possible in such sub-optimal situations.

Mr. Dawson T. Kilcrease, regional program consultant, Division of Health Mobilization, U. S. Public Health Service; Mr. Ralph M. Shannon, executive director, Dallas County Chapter, American Red Cross; and Mr. Boise B. Smith, director, Dallas City-County Civil Defense, gave assistance and guidance in the pilot program of Medical Self-Help Training; Dr. Sanford served as chairman.

as injured survivors, perhaps resulting in a 36 per cent decrease in casualties in Dallas. It has been estimated that the total casualties could be decreased to 10 per cent of the "unwarned" estimates with a nationwide shelter program which provided 10 psi or more blast protection as well as fall-out protection. However, even in Dallas there still would be approximately 20,000 casualties—a tremendous disparity. There are already at least four major approaches to help meet the problem of expanded health resources.

Expanded Hospital Facilities

The first of these is expanded hospital facilities. The heart of this program is the 200-bed Civil Defense Emergency Hospital. There are 1,818 of these hospitals now pre-positioned about critical target areas in the nation. These units would supply approximately 400,000 hospital beds, or perhaps 50 per cent of the available beds in the event of major disaster. These hospitals have been pre-positioned in a fashion such that there is one hospital for every 50,000 population in critical target areas. The hospitals with a few exceptions are placed at least 15 miles out from anticipated target areas and the anticipated loss is 9 per cent, in contrast to the 80 per cent loss for fixed hospital beds. These units are not plush, but are best termed "austere," and have the potential for definitive surgical care for a period of 36 to 96 hours. This year an additional 750 hospitals are to be pre-positioned and the capability of the existing units is being increased from 3 days to 30 days. There are currently in Texas 88 such pre-positioned hospitals and 5 training units.

Expanded Function Medical Care

The next major approach to the problem of expanded health resources is expanded function medical care. Again the problem of disparity between medical means and casualty load is emphasized. Korean experience showed that a physician could operate on a maximum of 10.5 patients per day and this could not be extended beyond three days. Consider this in light of the 120,000 Dallasites who might require medical attention from about 800 doctors.

A partial solution to this problem is the training of allied health personnel to work under the supervision of physicians. For example, veterinarians are well-trained in many techniques of surgery and anesthesia; dentists are skilled in use of instruments, and have much basic medical knowledge. Such a program would not constitute poor medical care, but could better be described as ideal therapy under suboptimal conditions.

Maximal Utilization

The third phase is planning for maximal utilization of surviving personnel, facilities, and resources. Such is going to require the development of coordinated regional support plans.

Medical Self-Help Training

The fourth approach is the Medical Self-Help Training program. What is the program? It includes not only first aid, but also teaches the individual what to do in the event that a physician does not and is not going to arrive for a reasonable period of time, for example, that interval during which individuals must remain in shelters if they are to survive.

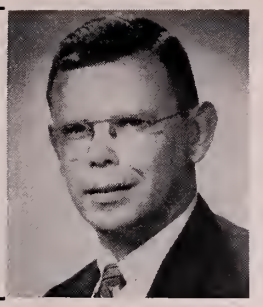
Here are illustrations of the type of material covered:

Under conditions of radioactive fall-out, which of the following sources of water would be safest for drinking purposes: (a) rainwater, (b) a fast-flowing stream, (c) an open reservoir, (d) a covered well?

Where there is a compound fracture of the limb, with heavy bleeding at the wound area, your first action should be: (a) Apply pressure at wound site with a pad or bare hand to attempt to stop bleeding. (b) Immediately apply a tourniquet on the limb above wound area. (c) Splint wound and apply pressure to wound area. (d) Immediately apply tourniquet directly over wound area.

Before training, the average score on such questions was 63 per cent, increasing to 84 per cent after completion of the course.

The course consists of 11 lessons, which are given in 16 hours. The subjects covered include radioactive fall-out and shelter. The student is taught the important concepts regarding radiation protection; that is, radioactivity decays with time. The decay is rapid,



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decreasing by a factor of ten with each seven-fold increase in time. Thus, after 49 hours the residual radioactivity represents only 1 per cent of the initial level. The next factor of seven is 343, hence within 343 hours, or roughly 14 days, the radioactivity has decayed to one-thousandth of its initial intensity. This is the basis for which the 14-day period of shelter habitation has been reached. The next lesson includes healthful living in emergencies. Subsequent lessons include food and water, shock, bleeding and bandaging, artificial respiration, fractures and splinting, transportation of the injured, burns, nursing care of sick and injured, infant and child care, and lastly, emergency childbirth.

The teaching materials for the entire course have been packaged into a suitcase-sized kit, which includes projector, filmstrips for each lesson, instructor's guide, student handbooks, screen, a pointer, and examination questions. The kit is designed to teach 100 students, preferably in classes of no more than 25 each. The initial cost of the kit is $48 and the handouts to replenish the kit are $20 per hundred, or 20 cents per student.

This program was approved by the American Medical Association, and was activated late in 1961. The kits were placed in the states late in March, 1962, under a pilot program. Under the pilot program, courses were to be given and as of June 30, 1962, the results of the courses would be compiled and modifications of the program made prior to widescale introduction. In each state, the actual distribution and design of the program was at the state level. Approval by the state and local medical societies was mandatory prior to implementing the program. On Feb. 11, 1963, a mutual agreement was reached between the American Red Cross, the Public Health Service, and the Office of Civil Defense, whereby the Red Cross would offer its nationwide set-up of chapters and first-aid instructors to the various state Self-Help steering committees to assist in teaching Medical Self-Help. In Texas, the pilot program was carried out in nine areas and by the end of December, 1962, about 10,000 students had participated in courses presented by 72 local civil defense groups, including many taught by Red Cross first-aid instructors (Table 1). The nationwide totals are

TABLE 1.—*Medical Self-Help Program, Oct. 31, 1963.*

	Estimated No. Students	No. Classes	No. Questionnaires
Texas	10,000	246	6,555
U. S.		7,958	204,376

based on questionnaires received by the United States Public Health Service through Oct. 31, 1963. These figures are considerably less than the number of individuals who have participated in the course. Texas initially trained more students than any other state.

An analysis was made of the improvement in pre- and post-course test scores, depending on the type of instructor (Table 2). The

TABLE 2.—*Relationship Between Type of Instructor and Improvement.*

Instructor	No. Students	% Improvement*
M.D.	1220	25
Allied health (nurses)	6460	35
School teachers	3751	36
Laymen	2237	32

*Improvement judged by pre- and post-course test scores.

training plans actually may be read verbatim by laymen, and include information such as "turn on slide," "point to such-and-such," and so forth. As a physician, the author finds this type of presentation quite difficult; however, it does insure that all material has been covered. The percentage of improvement is approximately the same regardless of whether the instructors have been physicians, allied health personnel such as nurses, school teachers, or laymen.

Analysis of improvement according to type of student illustrates that even medical and allied health personnel have gained some information, or at least learned how to answer a quiz, in that a 26.5 per cent improvement was noted in this group (Table 3). Of particular note is that 97 per cent of the students who have taken the course thus far have felt it to be worthwhile.

The anticipated future of this program is

TABLE 3.—*Relationship Between Type of Student and Improvement.*

Type Student	% Improvement*
Medical and allied health	26.5
Professional	26.5
Non-professional	48.3

*Improvement judged by pre- and post-course test scores.

that sets will be given to the states to train an additional 5,000,000 people by June 30, 1964. Training kits are now available and are being distributed in Texas. Information regarding the establishment of programs is available from Mr. Philip W. Gauss, Jr., Texas State Department of Health, Austin.

Such programs seem overdue, in that between 1955 and 1956, 110,000,000 persons within the Soviet Union completed a ten-hour course in the fundamentals of atomic defense, and approximately 22,000,000 civil defense workers have received further training.

Summary

In summary, civil defense medical problems must be approached with the realization that nuclear attack would mean great losses in trained medical personnel and facilities; at the same time, overwhelming numbers of people would need medical attention. This disparity can be minimized by preventing casualties through shelter programs, by knowledge of medical self-help, and by planning for expanded hospital facilities and expanded function medical care.

Dr. Sanford, University of Texas Southwestern Medical School, 5323 Harry Hines Blvd., Dallas 35.

Mental Illness in U.S.S.R. Compared With That in U.S.

An attempt to compare Soviet and American approaches to mental illness has been made by a Boston University professor, a sociological authority on Soviet medicine.

Dr. Mark G. Field, who has traveled extensively in Russia in recent years, cites some major obstacles to making any blanket comparisons. A lack of common terms and definitions, the absence of standardized terms, differences in diagnostic procedures and therapeutic orientations may indicate differences that do not exist in reality.

Russian medical attention appears to be focused on somatic problems that cause loss of working capacity, Dr. Fields believes. He continues by saying, "The Soviets claim time and time again that their type of society which has a clear-cut, well-defined goal and collectivistic orientation in its culture leads to a lesser degree of mental illness than a capitalistic society with its antagonistic classes, its violent competition, its irresponsible individualism, and its multiplicity of goals."

Dr. Field's analysis of more accurate Soviet data shows, however, that the proportion of people listed as having "psychic disturbances" and "diseases of the nervous system" amounts to a figure roughly comparable to that of the United States.

He was informed that the center of the Soviet psychiatric arch is not the mental hospital but the community neuropsychiatric clinic or dispensary, a comprehensive facility located in the community which provides a variety of services, preventive, clinical, and rehabilitational. Such a clinic is well in line with some of the most advanced psychiatric thinking in the West, but Dr. Field cannot be sure how many such clinics actually exist in Russia.

Dr. Field's findings are soon to be published in the first issue of *The Review of Soviet Medical Sciences* in Munich, Germany, by the Institute for the Study of the U.S.S.R.

Radiological Defense in Texas

CHARLES E. BARDEN, P.E.

SYMPOSIUM

RADIOLOGICAL DEFENSE is a part of the total emergency medical, health, and special weapons organization, which plans for the provision of essential services to the population in the event of nuclear attack.

The surviving population, both injured and uninjured, could be exposed to radiation levels which could kill, cause illness, shorten life span, and probably produce genetic effects.

Texas' Radiological Defense Program (RADEF) is concerned with increasing the actual survival rate after a nuclear attack. The extent and degree of radioactive fallout, the promptness and adequacy of its measurement and evaluation, a knowledge of protective measures for people and materials, and a knowledge of radiation control including shielding and decontamination will affect survival.

Broadly stated, the mission of RADEF in Texas is

1. To plan and direct those actions which can be taken to decrease the number and degree of radiation casualties.

2. To reduce or eliminate the harmful effects of radiation contamination and exposure after survival.

3. To reduce or eliminate contamination in later periods of local and national rehabilitation.

The organization has established these statewide capability objectives:

1. To organize, train, equip, and direct a force capable of using effective measures against radiation hazards created by nuclear explosions.

2. To predict areas of fallout with corresponding fallout zone radiation levels, and to inform the people within each area of existing dangers and how they can best protect themselves.

3. To monitor areas to determine radioactive contamination.

4. To establish ways to protect food and water, and to decontaminate and dispose of radioactive materials.

Organization

Serving in the Texas State Department of Health, the director for radiological defense acts for and reports to the director of emergency medical and health services, and is responsible for the general planning, initiation, direction, coordination, and supervision of the total statewide organization for radiological defense. Assisting this officer are his deputy and the directors of monitoring services, plotting services, evaluation services, and decontamination services.

Local leadership and participation by the people who will be affected is an absolute necessity in carrying out any civil defense program. To insure this, 17 radiological defense districts have been established, with a RADEF officer in each district. The officer's job is to organize training programs, to initiate an operational program, and to serve as liaison and reporter between city and county civil defense organizations and the state RADEF control center. These officers have done an outstanding job in establishing radiological monitoring capabilities in their districts (Fig. 1). For example, as of Dec. 31,

In most areas of Texas, trained persons stand ready to assess the situation and take action where needed if radiation becomes a hazard. Their knowledge would be vital in increasing the survival rate after nuclear attack.

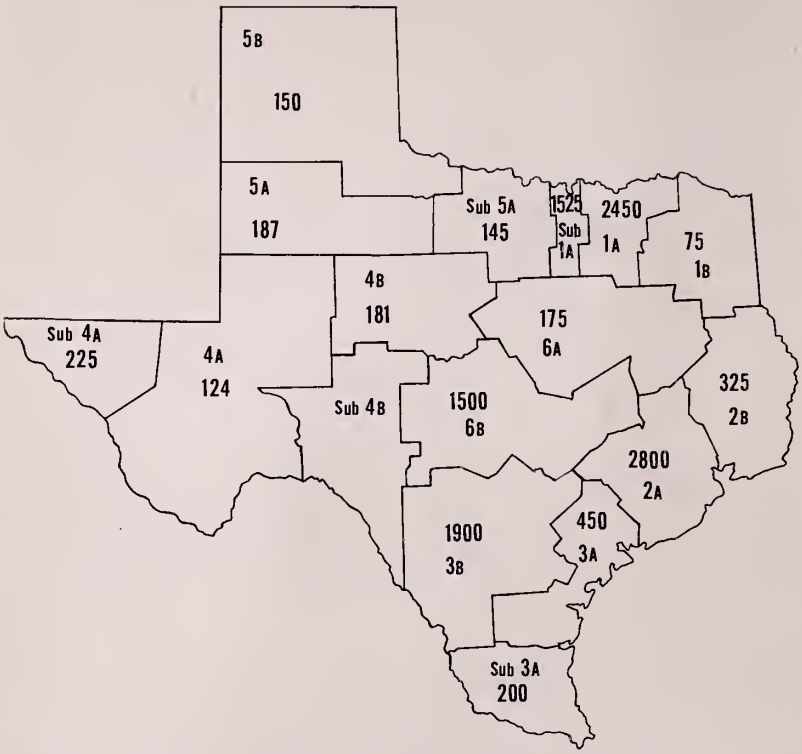

Fig. 1. Shown are the number of trained RADEF monitors in each of the 17 disaster districts of Texas as of Dec. 31, 1963.

1963, 12,437 people had been trained as radiation detection monitors in Texas.

The state is fairly well covered with trained monitors who are capable of obtaining radiological information for local and state medical officers. In the few areas which are not adequately covered, the fault lies not with the local personnel but is the result of a shortage of instructors. With high interest in radiological defense, all areas of the state

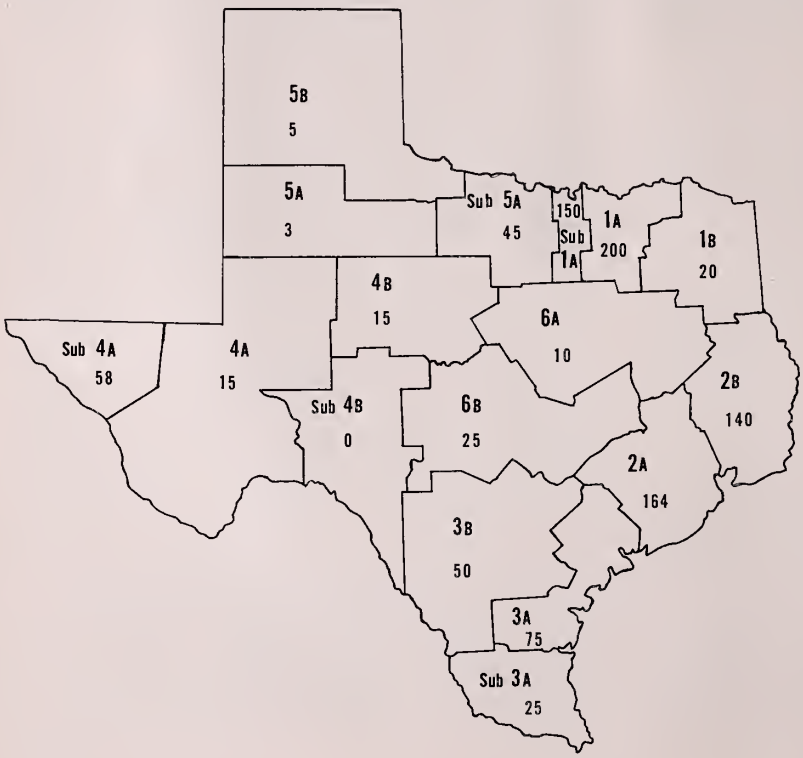

Fig. 2. Shown are the number of operational kits for radiation detection distributed in each of Texas' 17 disaster districts. These figures do not include the kits for use in public shelters.

will soon have trained personnel for this important activity.

Instruments

After an individual has received the required training and has agreed to serve as a radiation monitor in an emergency, instruments are shipped to him. Fig. 2 shows the current distribution of radiation detection instruments which have been provided by the Office of Civil Defense.

The radiological defense operational set, which is used for both public shelter and outlying monitoring stations, includes the following detection instruments:

1. One Geiger counter with earphones to detect low-level contamination of food, water, and personnel. The range of this instrument is 0-50 milliroentgens per hour.

2. One ionization chamber survey meter for detection of high range radiation. This instrument is capable of measuring radiation intensities from 0-500 roentgens per hour. It covers a range which formerly required two meters.

These instruments are designed to measure the dose rate levels of radiation and not total dosage.

To measure the total dosage received by monitoring personnel, the set includes three pocket chambers (direct reading dosimeters).

Mr. Barden is Director, Division of Occupational Health and Radiation Control, Texas State Department of Health, Austin. He presented this material at the Stateline Course on Medical Aspects of Civil Defense held in Austin in February, 1963.

These have a range of 0-200 roentgens. There is also a charger to reset the dosimeter after each use.

The work that has been done at state level, such as the training of instructors, monitors, and radiological defense officers, as well as the issuance of equipment including the operational kits, radiation source sets, and radiation detection training sets, has been accomplished by personnel of the Division of Occupational Health and Radiation Control of the Texas State Department of Health. These activities have been performed in addition to regular duties, and with the cooperation of other state and local agencies.

Division personnel stand ready to help in any way to expedite local radiological defense programs, and requests for aid may be sent to that office.

Mr. Barden, Division of Occupational Health and Radiation Control, State Department of Health, 1100 West 49th, Austin.

Fitter than at First

Today's kids are stronger and better conditioned than the school children of a few years ago. They are spending more time in physical fitness programs.

This is the report of the President's Council on Physical Fitness which based its findings on a U.S. Office of Education survey and pilot studies on 119,904 American school boys and girls.

The Council also announced its next plan which is to get adults in shape. An illustrated 64-page exercise book is now on sale at a cost of 35 cents.

Despite the improvements, the United States still has a long way to go to match many foreign countries in fitness programs, the Council maintains.

Ulcerative Colitis

Recognition and Management

CLINICAL MEDICINE

A DIAGNOSIS OF idiopathic ulcerative colitis hinges on the symptoms, the proctoscopic appearance of the rectosigmoid, the roentgenographic appearance of the colon, and the elimination of specific causes of colitis.

Recognition

The symptoms vary and are not diagnostic, but can be classified by their onset and course as acute fulminating colitis, intermittent or recurrent colitis, and continuous colitis. At our university,* about 40 per cent of cases are of the intermittent or recurrent type with moderate symptoms of fever, bloody diarrhea, anemia, weight loss, and anorexia, while the symptoms of cases observed by our colleagues in practice are usually milder. Often, constipation and rectal bleeding are the main or only complaints. Fortunately, the acute fulminating case is rare. Frequently, the patient with continuous colitis symptoms travels from doctor to clinic searching for a cure.

Usually, ulcerative colitis begins in the rectum and spreads toward the cecum. It might be anticipated that symptoms would correlate with the extent of involvement. Sometimes this is true, but some patients with disease limited to the rectum or rectosigmoid have been the sickest, and others with total colon involvement have had minimal symptoms. We are unable to correlate roentgenographic appearance and proctoscopic appearance closely with the severity of symptoms.

Patients can be classified by the extent of anatomical involvement of the colon as having rectum or rectosigmoid (proctitis), left-sided colitis, total or universal colitis, segmental colitis, and enterocolitis. For some time, arguments about ulcerative proctitis have flourished. Is this the same disease as idiopathic ulcerative colitis or is it a distinct entity? A growing body of experience suggests that it is the same disease as ulcerative colitis. We, as well as others,[9] have seen the inflammatory change spread to involve the whole colon. Fortunately, the symptoms of ulcerative proctitis are usually mild and tend to be self-limited to distinct attacks of four to six weeks; recurrences can be severe and difficult to control.

Segmental or right-sided colitis presents a special problem. The systemic symptoms or complication may dominate the clinical picture and the condition go unrecognized for several months to years. Aside from the traditional symptoms of rectal bleeding, cramps, and diarrhea, chronic ulcerative colitis should be looked for in all patients with perirectal disease such as fistulas, perirectal abscesses, cryptitis, or anal ulcers; in patients with unexplained fever; in patients with arthralgias and arthritis; in patients with erythema nodosum, and in patients with unexplained anemias, recurrent iritis, or conjunctivitis.

Few conditions seen through the proctoscope can be confused with chronic ulcerative colitis. Characteristically, early, there is marked edema of the mucosa with severe hyperemia. Often, the mucosa is a raw bleeding surface, or where bleeding is not present, brushing with a cotton swab will produce multiple pinpoint ulcers from which blood wells. The exudate may be creamy, profuse, and may have a foul odor.

Later, as the process subsides, the mucosa becomes dull, granular, and lusterless or a chronic state supervenes and the mucosa be-

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Chronic ulcerative colitis is a disease of unknown cause. Treatment is challenging, but with new agents available, results of management continue to improve.

MARCEL PATTERSON, M.D.

comes studded with multiple, friable, gray, bleeding pseudopolyps.

In our experience, the most confusion regarding diagnosis arises from acute bacillary dysentery. In this instance, the lesions appear identical to acute ulcerative colitis, but stool cultures and the rapid healing following sulfonamides or broad spectrum antibiotics provide a diagnosis.

Other conditions that might confuse are lymphopathia venereum, factitial proctitis from radiation, and trauma from enemas. Lymphopathia venereum must be on the wane. In our clinic, the patient with rectal stricture and a positive Frei and complement fixation test is seen less and less commonly. The changes from enemas as well as radiation injury are self-limiting. The former disappears in a few days, the latter in weeks to months.[2]

Almost every patient with ulcerative colitis referred to us has once had a diagnosis of amebic colitis. Usually, prior amebicide therapy did not help and the laboratory diagnosis of amebiasis was tenuous. Amebic colitis is not rare in our clinic, but usually appears distinctive by proctoscopy. Often, material aspirated directly from the discrete ulcers contains active trophozoites. Except for a few disturbing exceptions, amebic colitis responds promptly to treatment. A rare amebic colitis case under treatment has progressed to a "full-blown" severe ulcerative colitis case even when the parasites disappeared. When this occurs, the colitis has been difficult and frustrating to treat and the mortality rate has been high.

Tuberculous ulcers are large and discrete with ragged edges and gray bases. We have seen them only in patients with known pulmonary lesions. Rare conditions reported to produce lesions similar to chronic ulcerative colitis that we have not seen are scleroderma, amyloidosis, acute vasculitis, histoplasmosis, and diffuse infiltrating malignancy.[4] These lesions should be detected earlier with suction rectal biopsies. The shortened Rubin biopsy tube works well, and may be used with ease and safety.[3] A biopsy from all patients with idiopathic colitis will uncover more specific causes of the process. Furthermore, these tissue samples give the best information regarding response to therapy and the degree of healing.

The barium enema changes in ulcerative colitis are well known. They vary with the stage and anatomic location of the disease. Early, these changes can be slight and are primarily fine serrations of the colon wall, particularly the sigmoid and left colon. After evacuation, the mucosa may be granular-appearing. Sometimes, ulcerations can be giant-like resembling diverticula. Other times, multiple pseudopolyps occur producing large filling defects. Ultimately, the colon becomes shortened and tubular as the haustra disappear. Occasionally, one is surprised to see extensive x-ray changes with a very short history of disease.

As many as one-third of patients with ulcerative colitis, in some reported[12] series, have a negative or normal barium enema. On occasions, we have seen a patient with ulcerative colitis go unrecognized for months because the physician relied on the barium enema to make the diagnosis. Barium enema best defines the extent of involvement, whereas the proctoscopic examination and a suction biopsy best determines the state or stage of involvement. Neither examination alone adequately evaluates these patients.

In summary, the recognition of idiopathic ulcerative colitis should not be difficult if the physician uses the proctoscope often and

on the slightest provocation, if he gets films when colon symptoms dominate, or when he believes an occult process lurks, and if he takes the necessary steps to rule out specific etiologic causes of colitis.

Management

Ulcerative colitis management must be tailored to the individual patient. It cannot be standardized and must be modified by the amount and severity of involvement of the colon, and the complications that are present.

It should have as its goals[7] (1) the reestablishment of normal bowel function, (2) formed stools free of blood, (3) restoration of nutrition, and (4) resumption of an effective or normal life.

Therapy can be considered under the following headings: (1) psychotherapy, (2) diet, (3) correction of anemia and electrolyte deficits, (4) antibiotics, (5) control of symptoms, and (6) steroids.

Psychotherapy.—Often, ulcerative colitis has its onset during a period of severe emotional stress. The stress varies from a straightforward situation to a complex one concealed by embarrassment or shame which prevents the patient from talking of it without great pain. Sometimes months and occasionally years elapse before the patient can confide his troubles. Some patients never can, and in general, they have the poorest response to treatment.

The physician, rather than probing, should reiterate to the patient his belief in the importance of emotional problems in this disease and indicate his willingness to listen. Usually, this approach gets these patients to air their difficulties. In the meantime, one must be patient, unhurried, and sympathetic even when the demands appear unreasonable.

In most of these people, emotional support is critical. If, for some reason, such support cannot be provided by the attending physician, formal psychiatric help may be needed, particularly for children, since children, even more than adults, seem unable to talk about their problems. In our experience, the psychiatrist's discoveries and help with patients and their family situations could make the difference between therapeutic failure or success.

Diet.—The diet should be high in protein, attractive to the eye, and planned for squeamish and fastidious taste. If diarrhea is severe, the diet should be non- or low-residue. Frequent small feedings may be better tolerated than three regular meals.

For some time, clinicians suspected that milk was poorly tolerated and many would restrict milk, often with considerable improvement. This improvement may have resulted from a decrease in residue, but recent work[14] has demonstrated that some patients with chronic ulcerative colitis have high titers for milk antibodies in their blood and the reaction to milk is apparently on an allergenic basis. Denaturing milk by cooking may modify the protein enough to permit tolerance, but every patient with chronic ulcerative colitis should be given a trial on a milk-free diet. The significance of other food allergies is less clear-cut and, in our experience, strict elimination diets have not been too rewarding.

As soon as possible, one adds foods to the diet, at first cooked vegetables and later fruits, always encouraging a high intake of meat, eggs, cheese, and fish. Eventually, the patient in remission may tolerate a free diet. Unfortunately, too many of these patients latch onto a diet with great tenacity and much of the office visit is spent in a never-ending series of queries about what can and cannot be eaten, rather than in more helpful supportive measures. A printed diet sheet seems preferable and timesaving.

Anemia.—Most patients with chronic ulcerative colitis suffer with mild to moderate anemias of the iron deficiency type. Iron, because of its irritating effect, is often poorly tolerated by mouth;[1] however, if some effort is not made to replace the iron stores constantly drained by fecal blood loss, these patients tend to slip into severe anemias. Occasionally, the anemias are so severe that blood transfusions are required.

No one iron product has been completely satisfactory, but Simron has been an acceptable iron preparation for most of our patients. Other products can be explored when this drug is not tolerated and on occasions, intramuscular iron will be required. Ordinarily, electrolytes, except in the acute and fulminating colitis, are readily controlled by replacement of measured deficits. Therapeutic vitamins are desirable particularly with severe diet restrictions.

Antibiotics.—In general, broad-spectrum antibiotics are not well-tolerated by these patients.[6] Apparently, some[8] cases of ulcera-

tive colitis have arisen from them and many times antibiotics appeared to aggravate diarrhea and bleeding. In the presence of secondary infections, penicillin and streptomycin, intramuscularly, are the drugs of choice.

Salicylazosulfapyridine (Azulfadine) is a useful drug. We give 1 gm. four times a day to most patients, although others[5] recommend 2 to 3 gm. every four to six hours around the clock. The drug has produced dramatic remissions in some patients, while in others, it has not been helpful and in some

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rare instances, severe reactions have occurred. We have tended to restrict the use of Azulfadine to those patients with moderate symptoms and with involvement beyond the rectum where topical steroid therapy was not feasible or where response to topical and systemic steroid treatment was less than satisfactory. These patients should be followed closely during the first weeks of treatment for the development of leukopenias, anemias, drug fevers, vascular headaches, and rashes. Generally, these reactions occur early in treatment and if they do not appear, one can maintain Azulfadine therapy for months to years without untoward reactions.

The dosage of the drug is tapered gradually over the ensuing weeks at a speed dictated by the patient's response. Eventually, the drug may be discontinued completely.

Control of Symptoms.—These patients need a tranquilizing agent and something to control diarrhea. We have not found any tranquilizing agent superior to another in this disease, but have not done a planned or controlled study. Anticholinergics are not recommended and have the disadvantage of causing dryness, occasional urinary retention and, on rare occasions, toxic megacolon.[13]

Tincture of opium, Lomotil, and codeine have been useful in controlling diarrhea, but also have been incriminated as a cause of toxic megacolon and must be used with caution.[13]

Steroids.—Several years ago we[10, 11] began the use of topical steroids in ulcerative colitis. Early, we were impressed with the dramatic effects in some patients, particularly the rapid improvement of the proctoscopic appearance of the rectum. Since that time, we have continued to try this method on almost every patient with ulcerative colitis, partly to evaluate the place of this modality in ulcerative colitis, but mainly because it was the most useful method we had ever tried.

For patients with disease limited to the rectum or rectosigmoid, suppositories of hydrocortisone, 10 mg. (Wyanoid HC) are used. Rectal instillations of 50 mg. hydrocortisone acetate suspended in 100 ml. of water as retention enemas are given for disease limited to the left half of the colon. In patients in whom the disease extends to involve more colon, a solution of hydrocortisone sodium succinate (100 or 200 mg.) added to 250 or 500 cc. of saline is dripped daily into the rectum for two to three weeks, as a retention proctolysis.

If a response to topical therapy is not noted within a week, a systemic steroid, preferably a different one than the one given by mouth, should be added.

Recently, we have evaluated our efforts with this treatment. Since 1953, 62 patients with chronic ulcerative colitis have been treated with topical steroids. Eight had, in addition, systemic steroids plus topical steroids. Immediately, 38 had clinical and proctoscopic improvement, 12 were failures, and 12 had clinical improvement but no proctoscopic change.

Failure of response could not be related to duration, type of involvement or a history of lack of response to prior systemic steroid therapy (only 4 failures occurred in 15 prior steroid-treated patients). Poorest response was noted with enterocolitis (3 failures in 5 patients).

At follow-up the first year, 27 out of 41 were asymptomatic; second year, 22 out of 38; third year, 18 out of 26; fourth year, 17 out of 25; fifth year, 9 out of 15; and, over five years, 8 out of 11. Nineteen patients relapsed within the first year and 17 responded to a second course of treatment, but in general, response was poorer with each relapse.

Of the 12 initial failures, six had colectomy with one death within four years. Two are in remission, two continue with symptoms, one has progressed, and one responded partially to topical steroids later.

Perhaps the best summary of the usefulness of steroids in chronic ulcerative colitis is to be found in the extensive control studies reported by the Medical Research Council of England.[15] These findings are as follows:

1. Patients treated with cortisone fare significantly better than those receiving inert therapy.

2. Patients with first attacks do better than relapsing cases.

3. When the response of corticotropin (ACTH) and cortisone was compared, ACTH had a significant advantage over cortisone, particularly in relapses.

4. The results with prednisone and triamcinolone are not as good as with ACTH and cortisone.

5. Rectal complications are higher in the ACTH-treated group.

6. Combined steroid treatment (systemic and topical) gives the best results.

Summary

Chronic ulcerative colitis is a disease of unknown cause, recognized by its symptoms and its appearance and by elimination of specific causes of the colitis.

The treatment is difficult and challenging, but the number of agents to control the illness has increased and the results of management steadily improve.

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Credit Card Holders May Charge Hospital Bills

A guaranteed in-patient accounts program, the first national one sponsored by a credit card company, went into effect Jan. 1.

It guarantees the first $200 of hospital bills incurred by patients and provides them with immediate credit recognition at other associate hospitals.

More than 1,200 hospitals in 50 states are participating in the program sponsored by the Hilton Credit Corporation, whose cards, Carte Blanche, provide credit privileges on a worldwide basis including such establishments as restaurants, hotels, motels, airlines, and retail stores. They are also honored for automotive repairs and accessories, in major oil company service stations, and for many other services.

The Injured Diaphragm

WILLIAM KLINGENSMITH, M.D., and PATRICK OLES, M.D.

CLINICAL MEDICINE

INJURIES OF the diaphragm are not found commonly, but when they do occur they may produce immediate catastrophic effects if not recognized. Even if they are initially asymptomatic, they will eventually produce distressing symptoms, sometimes after many years. If the surgeon is alert to the possibilities of diaphragmatic hernia, many of the severe or even fatal complications may be avoided.

In certain socioeconomic groups the most frequent injury to the diaphragm is from stab wounds which usually pass across the pleural space and into the peritoneal cavity through the diaphragm. About 15 per cent of penetrating abdominal wounds enter through the diaphragm,[6] and the intimate anatomic relationship of the liver, stomach, spleen, and colon with the left hemidiaphragm must be kept in mind; these organs are actually within the thoracic cage, although not in the pleural space (Fig. 1). Any penetrating wound of the chest may involve the diaphragm and one of these organs, and the surgeon must be alert to the possibility of peritonitis or intraperitoneal hemorrhage developing in addition to the pneumothorax or bleeding within the pleural space that is often seen. Fortunately, the history of the stab usually alerts the surgeon; injuries of the diaphragm produced by indirect force may go unrecognized.

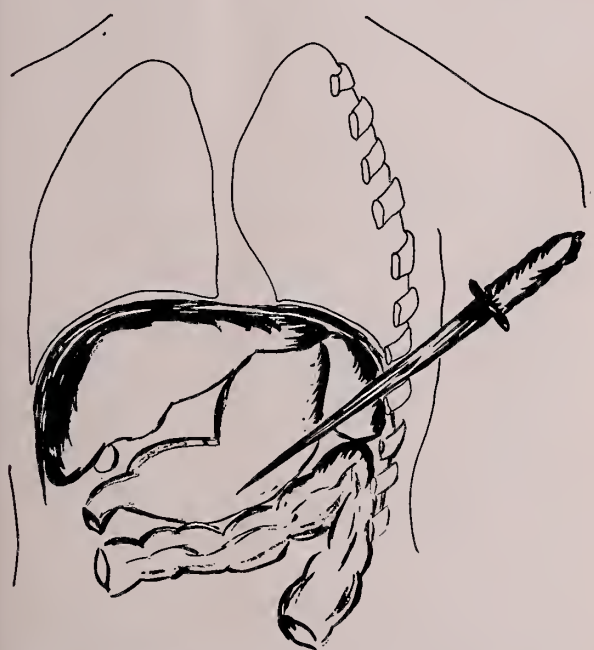


Fig. 1. The liver, spleen, stomach, and colon are intrathoracic organs, though subdiaphragmatic. They are in intimate contact with the diaphragm and may be involved in chest wounds that penetrate the diaphragm.

Stab wounds of the diaphragm do not ordinarily produce immediate herniation unless the diaphragm is extensively lacerated such as from a butcher knife wound. The most trivial laceration may lead to herniation months or years later as the omentum gradually works its way through the rent, to be followed by the bowel. When the peritoneal cavity is explored, the diaphragm must be carefully searched and these defects closed if the later serious complications are to be avoided.

During recent years, the most spectacular injury to the diaphragm has been the bursting injury associated with automobile accidents. These massive injuries are sometimes seen after falls or some other violent trauma, but most all of them are the result of high speed automobile collisions.[3] Such a bursting of the diaphragm must be the result of a violent trauma, and associated injuries to the long bones, pelvis, or head occur in most cases.[2] These associated injuries are obvious to the examining surgeon and may draw his attention away from the injury to the diaphragm. These patients are often comatose or in shock from fractures and unless there is respiratory distress due to massive herniation through the diaphragmatic defect, the chest may not be thoroughly examined or x-rayed. Immediate herniation is common in

The authors review injuries which result from penetrating wounds and from the bursting force of indirect trauma. They recommend early surgical intervention.

these injuries due to the large size of the burst defect, the extreme force applied to the abdomen in the production of the bursting injury, and the relatively negative pressure in the chest. If this herniation is not recognized, it may produce immediate serious results in the form of compression of the lung and shift of the mediastinum (Fig. 2), bowel obstruction, and eventual strangulation

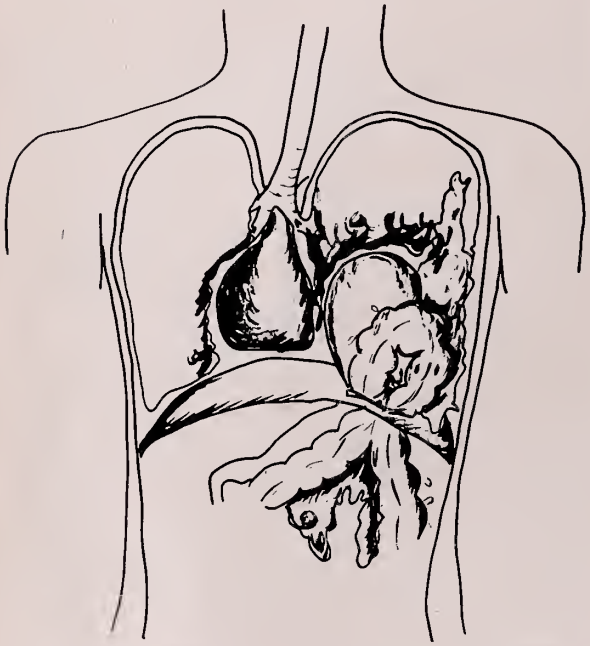


Fig. 2. Massive herniation of stomach, colon, and omentum into the left chest through a burst diaphragm. The left lung is collapsed partially and the mediastinum is shifted to the right, producing compression of the right lung. Severe dyspnea is the result.

and gangrene of the herniated viscera. Many deaths from these causes have been reported.[5]

The mechanism of these bursting injuries is of interest. About 90 per cent[4] of the injuries occur through the left hemidiaphragm because it is relatively unprotected by the liver and more exposed to the force transmitted through the stomach, spleen, and left lobe of the liver (Fig. 3). It has been found that this force is transmitted through the *intact* viscera to the left hemidiaphragm; if the liver or spleen ruptures, then the force is dissipated and the diaphragm may be spared (Fig. 4). Rupture most commonly occurs through the weak central dome of the diaphragm or along the pericardium (Fig. 5). The lacerations may be somewhat stellate and are often of large size, permitting the immediate evisceration of stomach, liver, colon, or spleen into the left chest.

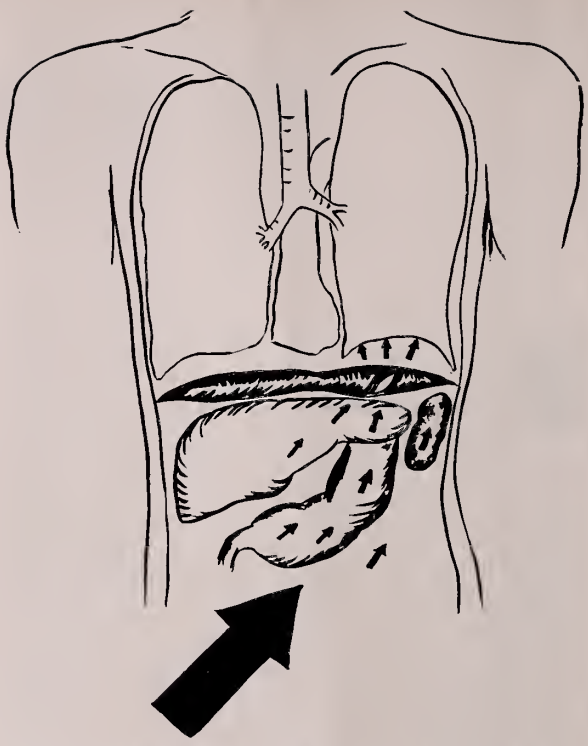


Fig. 3. Force transmitted through the viscera to the left hemidiaphragm. Rupture is much more common on the left because it is not protected by the liver.

The diaphragm may also be torn during the force of a crushing injury applied directly to the thoracic cage. This may occur with or without rib fracture and can also result in immediate herniation with massive evisceration into the left chest. Crushing injuries are more prone to involve the right chest than those produced by a bursting force applied to the abdomen. These crushing injuries are also the result of a violent force, such as that received by being run over by a car or other machinery. Rupture of the intrathoracic but subdiaphragmatic organs is more likely to occur.

The symptoms produced by the injured diaphragm are minimal unless herniation occurs, and even then they may be benign for long periods of time. In one large group of cases, an average of four and one-half years elapsed from the time of injury to diagnosis.[1] Unless the herniation produces respiratory distress, obstruction, strangulation, or other symptoms, the diagnosis is not made (Fig. 6). In most cases of burst injury due to indirect violence there is immediate and massive herniation, and the alert surgeon will detect the dullness in the left chest and make the diagnosis from a chest roentgenogram.

The diagnosis can be suspected from the

Fig. 4. A force applied to the abdomen is transmitted through the viscera to the diaphragm. If the liver or spleen ruptures, the force may dissipate, and sometimes the diaphragm is spared.

Fig. 5. Rupture commonly occurs at sites along the left pericardium or through the relatively weak central portion of the diaphragm.

violence of the original injury and the nature of the patient's symptoms, but ordinarily it is necessary to obtain a plain film of

Fig. 6. A 20-year-old man was involved in a collision one year earlier with a result of fracture of the femur and severe head injury with coma for several days. He had never recovered his strength or appetite, remained emaciated, and complained of vague pain and indigestion. With a sudden onset of abdominal cramping and nausea, the abdomen was explored in a community hospital, and an acute small bowel obstruction due to incarceration in left chest was discovered. Small bowel was reduced, and the patient was referred for treatment of chronic incarceration of the stomach and colon. Transthoracic approach was used; recovery was swift.

the chest to substantiate this impression. Sometimes the roentgenogram shows a huge gas-filled viscus (Fig. 7) almost filling the chest or multiple loops of bowel (Fig. 8), but sometimes the mass is not so definite and may be confused with a pleural effusion or hemothorax (Fig. 9). In rare cases the passage of a Levin tube or a barium swallow may help, particularly in the silent cases of long-standing where the eventration of the diaphragm may be confusing.

The treatment of these acute injuries may be and often is complicated by other injuries such as fractures of the long bones or pelvis, or cerebral injury. Often it is necessary to reduce the herniation without delay in the massive transdiaphragmatic herniations because of respiratory distress and the danger of strangulation (Fig. 10). In the acute injury, a peritoneal approach is of great advantage because the abdomen may be explored for other damage and there is as yet no adherence of the herniated viscera to the

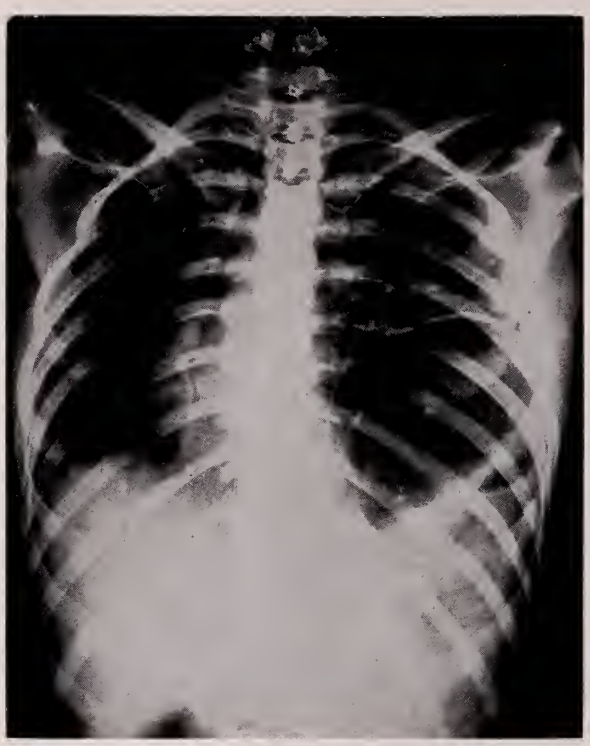
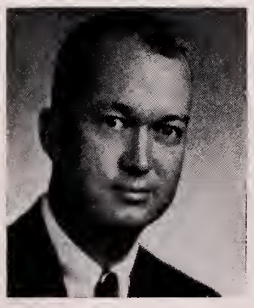


Fig. 7. This young woman had been in an automobile accident one hour before this film was made. She had a compound fracture of the tibia, multiple lacerations, and severe dyspnea. Note the huge gas-filled stomach in the left chest with shift of the mediastinum.

lung or pleura. In some instances, the rent in the diaphragm must be enlarged before the hernia can be reduced. In chronic herniations secondary to trauma, it is often impossible to remove the viscera from the chest cavity through the abdominal approach because of the amount of adherence in the chest. A transthoracic approach is much preferred and is often mandatory. The diaphragm can usually be closed with mattress sutures of silk or some other nonabsorbable suture.

Summary

Injuries of the diaphragm may result from penetrating wounds or from the bursting force of indirect trauma. Such injuries may lead to immediate severe symptoms, or they may be delayed for months or years. The diagnosis of bursting injuries often is obscured by associated severe trauma. The mechanism of these injuries is discussed, and early surgical intervention is recommended.



Dr. Klingensmith, pictured, and Dr. Oles practice surgery in Amarillo.

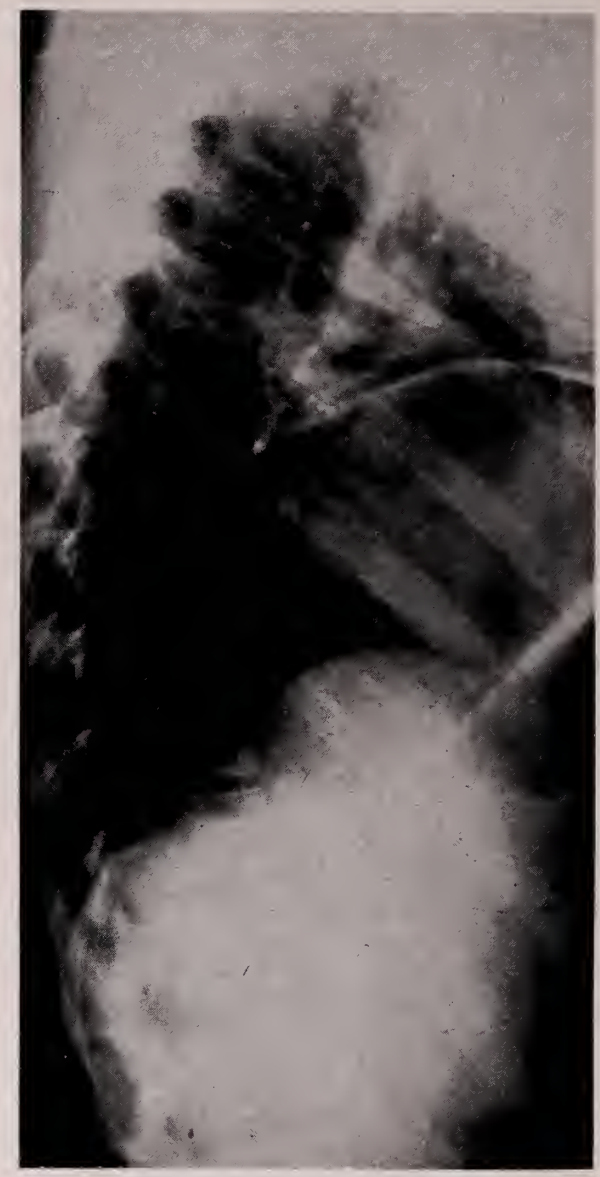


Fig. 8. Lateral view of patient in Fig. 7. An immediate transthoracic approach was utilized.

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Fig. 9. A 35-year-old man with a stab wound of the left chest four years previously complained of nausea, indigestion, cramps, weakness, and recurring "pneumonia" for one year. Incarceration of stomach, colon, and omentum had occurred.

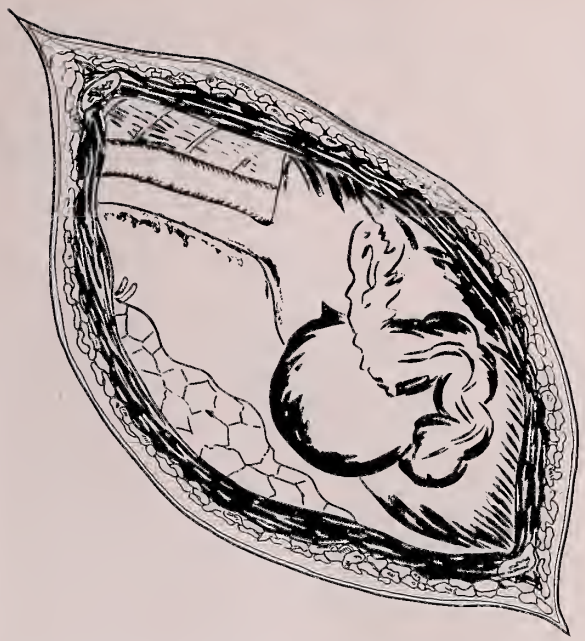

Fig. 10. A transthoracic view of a central rupture of the diaphragm with stomach, colon, and omentum protruding into the left chest.

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Blood Transfusion "Standards" Revised, Expanded in New Edition

A revised and expanded edition of "Standards for a Blood Transfusion Service," the fourth to appear, has just been published by the American Association of Blood Banks. A Spanish language translation of the new edition will be available shortly. The work details the Association's requirements for accreditation of hospital and community blood banks. The recommended standards have been accepted by other organizations in the field.

Greatest expansion in requirements appear to be for preparation of blood components. These include fresh frozen plasma for hemophiliac patients, stored liquid plasma for treatment of shock and severely burned patients, and packed cells to correct anemias.

The publication has been expanded to include a section on plasmapheresis, and also places great emphasis on protection of the donor, increased evaluation of the donor by qualified physicians, and makes a determined effort to exclude possible donors who may be carriers of viral hepatitis. There are also new and rigid requirements designed to discourage promiscuous use of group O blood. These outline the conditions under which group O blood may be used in recipients of other blood groups.

Copies of the new edition are available from the American Association of Blood Banks, 30 North Michigan Avenue, Chicago 2.

Carcinoma of the Prostate

CASE REPORTS

ALMOST BY tradition carcinoma of the prostate is considered a disease that begins in the fifth decade and increases in incidence in the older age groups. The literature makes few references to carcinoma of the prostate at earlier ages, and when a case occurs, its rarity makes it worth attention.

Chute[2] estimates that there are 195,000 to 1,500,000 cases of carcinoma of the prostate which, if diagnosed, would be operable. These figures are based on the percentages reported by investigators in combination with the 1950 census. With an increasing aged population these figures are rising. Chute also estimated that there are from 2,500,000 to 7,500,000 cases of carcinoma of the prostate in all stages. Usually it is first found as a small nodule in the prostate. It is asymptomatic and must be differentiated from prostatic calculi, prostatic granuloma, prostatic tuberculosis, and benign prostatic hypertrophy with condensation of the fibromuscular stroma. Cases that have metastasized are usually differentiated by the pyelogram and/or acid phosphatase. Those that have not metastasized can only be differentiated by either needle or open biopsy. Since surgery offers the only cure at present, the complications of radical prostatectomy such as urinary incontinence, impotency, and recto-urethral fistulas are not severe enough to justify withholding operation in cases that are operable. In comparing the surgical versus nonsurgical management of carcinoma of the prostate, Young,[17] Colston,[3] Hinman,[6, 7] and Jewett[9] give figures varying from 44 to 51 per cent of five-year cures, with no evidence of residual disease in their cases of radical prostatectomy. This compares more than favorably with the 43 per cent five-year survival in those patients who did not have metastasis in which the disease was treated by palliative therapy and in whom residual disease is still present. These latter figures were reported by Nesbitt and Baum.[12] To summarize, carcinoma of the prostate can only be cured by radical surgery and is only operable when discovered early. No one knows how long a carcinoma of the prostate remains in situ before it becomes active and spreads to the seminal vesicles, lymphatics, and bone.

Case Report

The patient, a well-developed and nourished white male, age 37, was seen in the office on March 31, 1961. During treatment for an acute pyelonephritis, an intravenous pyelogram had revealed a possible deformity of the renal contour with some elongation of the calyces of the left kidney. The patient had no relevant previous history. Examination revealed a .2 cm. nodule in the right lobe of the prostate. This nodule was small, firm, and discrete, but there was no fixation of the prostate. Needle biopsy was recommended, as well as retrograde pyelographic studies of the left kidney. The patient was hospitalized and laboratory data were within normal limits except for urinalysis, which revealed 1 to 4 WBC's per high power field.

Cystoscopy and retrograde pyelography revealed that the left kidney was normal. Transperineal needle biopsy was carried out using the Melick needle.

The biopsy specimen consisted of a 10 by 2 by 2 mm. moderately firm piece of gray tissue (Figs. 1 and 2). One-fourth of the specimen showed an infiltrating, exceedingly well-differentiated adenocarcinoma, forming small glandular structures which had well-differentiated cells. A number of the nuclei were hyperchromatic; mitotic activity was not prominent. Some of the glands showed duplication of the epithelium. Bone survey and acid phosphatase were normal.

A radical retropubic prostatectomy and seminal vesiculectomy were done, using the technique of Chute.[2] The patient's postoperative course was uneventful. His catheter was removed on the 13th postoperative day. The patient was continent and voiding well, and he was discharged from the hospital the next day.

The pathologic specimen (Figs. 3 and 4) consisted of a radically resected prostate gland with the vas deferens and seminal vesicles attached to the capsule of the gland. The prostate measured 50 by 30 by 35 mm. A previous needle tract could be identified in the right side of the posterior lobe. In this area there was a light tan, poorly circumscribed, very firm gritty area which blended into the prostatic tissue. It measured approximately 15 by 10 mm. The prostatic gland was not nodular nor was lymphoid

The 14th case of carcinoma of the prostate in a male under the age of 40 is presented, with a review of the literature. The value of regular prostate examination in individuals under the age of 50 is emphasized.

S. HEINEMANN, M.D.

tissue demonstrated. Microscopic examination of multiple sections taken throughout the prostate gland revealed carcinoma only in the posterior lobe on the right just beneath the seminal vesicles. This was located approximately one low power field beneath the capsule and the tumor was relatively small, covering approximately 3 to 4 mm. The tumor was diffuse, exceedingly well-differentiated, and infiltrated the stroma. In the center of the tumor a single nerve had been invaded. The perineural lymphatics in this area also contained tumor. Many sections taken through the capsule of the prostate and adjacent areas failed to reveal any tumor in the perineural lymphatics of the capsule, although there were a few areas in which there was considerable lymphocytic infiltrate in the perineural lymphatics. The tumor was confined to the posterior lobe and the line of surgical section was well around the tumor. Sections through the epididymis showed no evidence of invasion. Diagnosis was adenocarcinoma of the prostate, grade I.

With chemotherapy, the patient's urine quickly returned to normal. About six weeks after surgery his urinary stream slowed, and cystoscopic examination revealed what appeared to be a vesicle neck contracture. A transurethral resection of the bladder neck contracture was done, removing approximately .5 gm. of tissue. The patient was placed on catheter drainage. The pathologic diagnosis was resected hypertrophied and scarred bladder neck tissue. The patient was discharged on the sixth postoperative day.

The patient has been seen regularly. After his resection his urine cleared promptly and he was voiding well. About six weeks following surgery he reported that he had only slight difficulty in main-

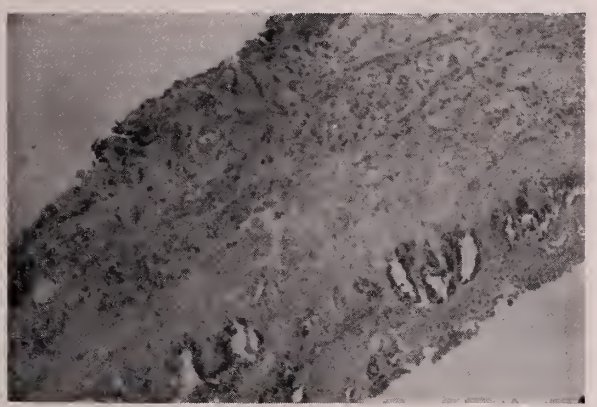


Fig. 1. Photomicrograph at low power shows entire width of needle biopsy. Note normal glands at upper margin and infiltrative cancer below.

Fig. 2. Photomicrograph at higher power shows infiltration of muscle and stroma.

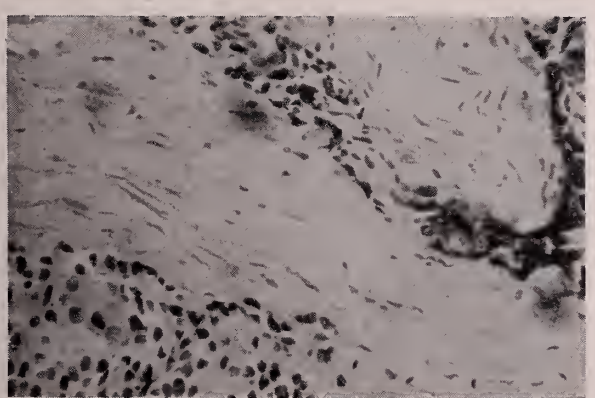


Fig. 3. Photomicrograph at low power shows perineural lymphatic invasion.

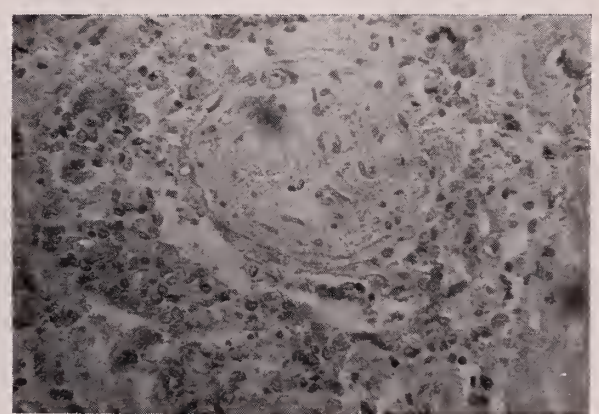
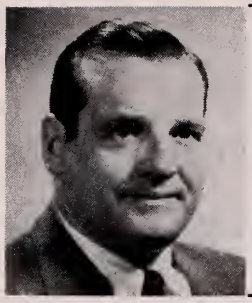


Fig. 4. Photomicrograph at slightly higher power shows nerve surrounded by tumor cells.



Dr. Heinemann, a diplomate of the American Board of Urology, is in private practice in El Paso.

taining an erection and was able to have satisfactory sexual intercourse. On his return visits to the office a 24 F. sound was passed with ease. The patient has been checked every three months with kidney, ureter, and bladder films, and acid phosphatase has been taken every six months. Thus far, these studies have been normal. The patient is continent and able to maintain his sex life.

Review of Literature

There have been 13 cases of carcinoma of the prostate reported in patients between puberty and age 40. I believe this to be the 14th published case within this age group, although I am sure there are many unpublished. Kimbrough and Lewis[11] have reported two cases, one aged 20 and one aged 18. Wolff[4] has reported six cases. Gardner and Cummins,[5] Moore,[15] Gaynor,[4] Wear and Schoenenberger,[16] and Nicholson[13] have reported one case each in this age group.

There have been four cases of carcinoma of the prostate reported in patients under the age of puberty. Aleksejew and Dunaevsky[1] reported a case in a 1-year-old child; Spaeth[14] reported a patient aged 3; and Hunata[8] reported a carcinoma of the prostate in a 20-month-old infant found at autopsy. The first American case in this age group was in a 3-year-old child, reported by Kagan and Fox.[10]

How long will a carcinoma of the prostate remain in situ? Following the usual rule of thumb that these carcinomas occur after the age of 50, one wonders how many of those seen after the age of 50 have been present for years, to be discovered only after they increased their rate of growth. Hinman claims that only 5 per cent of the carcinomas of the prostate are found early enough to be cured by surgery. Colson gives a higher figure of 20 per cent. However, these are still very low figures. One must consider whether males should have routine rectal examinations at a much earlier age than 50. With the development of the Papanicolaou stain, our fellow workers in the field of gynecology are expanding their examinations in the female to all age groups in an effort to discover carcinoma of the cervix much earlier.

Conclusion

Urologists have been the leading advocates of routine rectal examination and educational programs for all patients in an effort to find carcinoma of the prostate. Usually, they have advocated that this examination should begin at about the age of 50 and should be done annually. Here is reported a carcinoma of the prostate in a 37-year-old man. It is hoped that this report will encourage physicians to advocate more frequent rectal examinations in earlier age groups in the hope of discovering more cases of carcinoma of the prostate that will be amenable to surgery. The literature on carcinoma of the prostate in patients under the age of 40 has been summarized and reviewed.

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Mammography in Carcinoma of the Male Breast

THOMAS S. ALLEN, M.D.

CASE REPORTS

MAMMOGRAPHY, SOFT tissue radiography of the breast, is used usually as an aid to physical diagnosis in women with palpable nodules or as a screening procedure. There are occasional instances in which roentgenographic examination of the male breast is of value. It is the purpose of this report to describe the radiographic findings in a case of carcinoma of the male breast, which are thought to be of some interest because of the infrequency of this type of tumor.

Case Report

A 75-year-old white male saw his physician at the insistence of his family about six months after he first noted a lump in his right breast. During that time the mass had grown from about the size of a marble to that of an egg. There was no history of previous trauma to the breast or pain or tenderness of the area. There was no past history or family history of cancer of any type.

Physical examination of the right breast revealed a 4 by 5 cm. nontender mass beneath the nipple which was attached to the overlying skin. It was questionably fixed to the underlying tissues. The left breast was normal. There was a small, firm node palpable in the right axilla.

Mammography revealed two rounded, lobulated densities beneath the right nipple, each approximately 2 cm. in diameter. The border of the superior mass was smooth, but the border of the lower one was ill-defined and spiculated. Skin thickening and retraction were noted. The lower mass was partially superimposed on the other and could have arisen from it. The vessels in the right breast were increased in number and size in comparison to those in the normal left breast. The radiographic impression was carcinoma of the right breast.

Biopsy of the mass revealed adenocarcinoma, and a radical mastectomy was performed.

The specimen of the tumor measured 3.5 cm. in diameter and was of a woody hard consistency. It appeared grossly to be well encapsulated. The cut surface was gray-white in appearance. Microscopic sections of the tumor revealed a poorly differentiated adenocarcinoma which infiltrated the fibroglandular tissue and the surrounding adipose tissue of the breast. Sections of multiple lymph nodes from levels one through three of the internal mammary chain revealed only reactive hyperplasia.

Discussion

Carcinoma of the male breast is relatively rare. It is said to occur in approximately 1 per cent of all tumors of the breast in both sexes.[1] In the largest published series, Treves found the average age of the patients to be 52.1 years in 146 cases. Although the etiology is unknown, a history of antecedent trauma is not infrequently obtained.

In general, men tend to seek the advice of their physicians considerably later than women. In Wainwright's collection of cases, the average duration of the tumor was 2.4 years,[2] and every series contains a number of cases in which the lesions were present for many years prior to surgery. The fact that carcinoma of the male breast is generally considered difficult to cure may be due to the advanced nature of the disease when first treated.

Physical examination usually reveals a hard mass beneath the nipple, often fixed to the overlying skin and underlying chest wall. Enlarged axillary lymph nodes are frequently present.

In an older patient, the differential diagnosis includes a senile type of hypertrophy. In a younger patient, breast enlargement may be due to gynecomastia. Hypertrophy of the breast occurs frequently secondary to liver or thyroid disease.

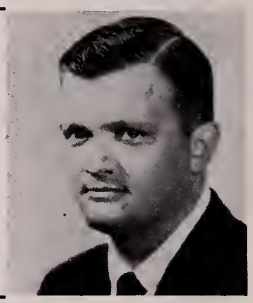
The use of mammography in diagnosing carcinoma in the case of a 75-year-old man is discussed.

Fig. 1. Two superimposed rounded, lobulated densities with distinct borders are revealed. The lesion was situated behind the nipple.

Fig. 2. The single arrow points to thickened overlying skin, while the two adjacent arrows show the ragged, invasive border of the tumor.

Mammography has been in use at the University of Texas Medical Center for about two years and has been found of assistance in the clinical evaluation of breast lesions. The radiographic findings in this case were striking. Soft tissue radiography of the breast may help persuade the male patient to consent to surgical examination and treatment earlier than he may otherwise.

Dr. Thomas S. Allen, now practicing in Beaumont, was a resident in radiology at the University of Texas Medical Center in Galveston at the time that he prepared this article.



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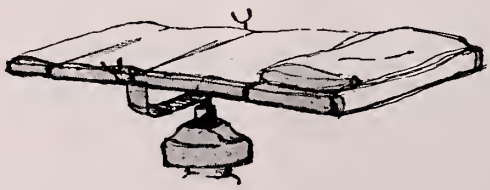


Clinicopathologic Conference

F. LAMONT JENNINGS, M.D.; C. WILLIAM DAESCHNER, M.D.;
and JOE B. NASH, Ph.D.

This is the fourth in a series of clinicopathologic conferences from the University of Texas Medical Branch, Galveston. Guest editor for this series is Dr. Howard C. Hopps, Department of Pathology. Dr. F. Lamont Jennings, professor and chairman in the Department of Pathology, serves as moderator. Dr. C. William Daeschner, professor and chairman in the Department of Pediatrics, discusses the clinical aspects of the case. Dr. Joe B. Nash, associate professor in the Department of Pharmacology and Toxicology, reviews the toxicological problems presented by the case.

THIS NEGRO BOY was born in John Sealy Hospital on Nov. 11, 1959, the fourth child of a 38-year-old mother. The pregnancy and delivery were normal, and birth weight was 6 lb. 4 oz. At the time of his delivery, two siblings were living and well; one an 18-year-old sister and the other a 3-year-old brother. One child had died at 16 months of age of pneumonia. Our patient was seen as an outpatient at 1 month of age, when he was suffering from an upper respiratory infection. It resolved in about ten days with symptomatic therapy. At 4 months of age he developed rubella and was again followed as an outpatient. He was next seen in the clinic at 15 months of age when he had an upper respiratory infection. He was not seen again until he was 3 years old when he had a routine physical examination in the clinic. At that time his height was 34½ in. and his weight 21½ lb. The possibility of dwarfism was suggested at this physical examination. His mother indicated that the boy had suffered from numerous respiratory infections during the previous 15 months. He was not seen again at John Sealy Hospital until he was dead on arrival in the emergency room at 9:20 a.m. on April 12, 1963. His family indicated that the lad had been irritable for the past few days, and they felt he was suffering from a respiratory infection. On this morning he was somewhat more irritable but at about 8:10 a.m. he had walked to the neighborhood grocery store and had purchased ten unpackaged "butter cookies" for five cents and had returned home at about 8:30 a.m., having consumed seven or eight cookies en route. A short time later his mother left to go to work on the bus. The lad fussed when his mother left home and he was spanked mildly by his 18-year-old sister and was taken to the breakfast table. The boy sat at the table but refused to eat his breakfast. His sister asked if he would prefer a pork chop for breakfast and the lad indicated that he would prefer this to his regular breakfast. However, when served the pork chop he refused to eat it and sat quietly in the chair. A few minutes after being served the pork chop, the boy fell from the chair for no apparent reason. His older sister, who was sitting with him at the table at the time, rushed to his side but felt that he was dead when she got to him; she rushed him immediately to the hospital where he was pronounced dead on arrival at 9:20 a.m.

Discussion

DR. DAESCHNER: Sudden and unexpected death of infants and children has occupied the thinking of many physicians in the past 50 years. Clinicians and pathologists alike have described these patients in detail, with some of the most comprehensive studies ema-

Sudden death in which a 3-year-old Negro boy fell from his chair, apparently dead, was the problem confronting the attending physicians. While the child had had a recent upper respiratory infection, he showed no prodromata except some irritability on the morning of death.

nating from the medical examiners offices of large metropolitan areas.[2, 3, 11] Children, unlike adults, show no "normal" pathology with which sudden death may be reconciled. The histories are often strikingly similar.[14] The infant is usually 2 to 4 months of age—uncommonly a neonate or child over 6 months of age. It is a male child by a 2 to 1 or 3 to 1 ratio, and there is a distinct predilection for Negro as compared to white children. The home environment includes almost every socioeconomic strata of society. Though a mild upper respiratory infection often is mentioned (sometimes only in retrospect), it is rarely of a severity sufficient to encourage even the most anxious parent to seek medical advice. More than a few children have been seen for a routine health check a few hours to a day before death. The peak seasonal incidence of these deaths is in the spring, though no month is free of them. This child had a mild upper respiratory infection and died in April. Immediately prior to death he appeared well, and certainly no one anticipated as they began breakfast that he would die within a few minutes. In contrast to the usual situation, however, he was almost 4 years old, a time when sudden unexpected death is uncommon and unexplained death is much less likely. His age then encourages us to believe that the pathologist will find a cause of death.

Some of these deaths will be the result of previously unrecognized congenital malformations—particularly those of the heart and central nervous system (and occasionally, genitourinary defects). The fact that this child at age 3 was more the size of a 1- to 2-year-old suggests that there existed some unrecognized factor interfering with growth, such as acyanotic congenital heart disease (e.g. pulmonic stenosis or large ventricular septal defect). Anomalous great vessels creating a ring about the trachea should also be mentioned, however, the absence of symptoms and findings at the time of his earlier clinic visit and in the interim makes these unlikely. Although he is said to have had frequent "colds," these were apparently mild and well-tolerated so I believe we can avoid further consideration of chronic lung disease.

Sickle cell anemia may cause rapid death,[8] especially in infancy, but these patients usually go into shock with tachypnea, dyspnea, and extreme pallor and die on the way to the hospital or shortly after admission. This course was not described in our patient. In those who live long enough to have one, the blood hemoglobin determination may be in the 1-3 gm. per 100 cc. range. Brain tumors, though characteristically producing pathognomonic signs, may exist silently until edema or hemorrhage suddenly expands them to symptomatic proportions. Here, again, one would expect some evidence of central nervous system disturbance in the hours before death.

Accidental deaths are still a major killer of children. It is important, therefore, to our discussion that this patient was observed at the time of death. There was no struggle, no seizure, no cyanosis, and no evidence of mechanical suffocation. Thus, we can put aside the possibility of a plastic bag or wrapper over his head, or aspiration of a foreign body. It cannot be ignored that immediately before death he consumed some unpackaged cookies. Very few poisons he might have obtained would have killed as quickly and as silently as this child died. However, I will leave this to Dr. Nash's discussion and simply say this does not look like poisoning to me. Injury seems unlikely in this patient though there was a period when he was away from home and unobserved 20 to 30 minutes before his death. Blunt trauma to the precordial area by the fist or head in children at play has been the source of rapid death with the findings at postmortem of massive contusion of the myocardium. These patients usually experience severe chest pain and dyspnea. Our patient was not described as being uncomfortable or dyspneic so this event seems unlikely.

Fulminating infectious processes are often suspected in the death of small infants.[5, 15] These may be bacterial or viral sepsis with secondary involvement of the lung or heart. In other patients primary pneumonic[1, 6] and myocardial disease is described. The bacterial infections in small infants are most often caused by gram-negative enteric organisms. It is interesting to remember that the neonate loses his passively transferred maternal immunity by about the sixth week, and must then begin to rebuild, by his own immune response, a portfolio of antibodies. The peak incidence of sudden deaths in the 2 to 4 months age group seems more than coincidence. Some authors have noted, in individual cases, low levels of gamma globulin[13] but in large series no significant deficiency

of gamma globulin or total protein has been discovered, so this seems an unlikely etiologic factor.[17] Others have shown that these infants also have normal serum complement levels.[4] Sepsis due to *Hemophilus influenzae, Diplococcus pneumoniae,* and *Neisseria meningitidis* are other plausible causes of sepsis and sudden demise in infants and young children. These are usually preceded by fever and a period of illness before death. Several authors studying these perplexing cases have noted a tendency to mononuclear cell infiltrates in the lung producing the histologic appearance of an interstitial pneumonia.[1, 6] Experimentally, this picture with sudden death of the animal may be produced by the administration of large doses of virus to the unimmune or only partially immune subject. Death occurs so rapidly there is little alveolar exudate, and characteristic inclusion bodies do not form. Recent studies,[3] however, cast some doubt on this entity for similar histologic changes in the lung are sometimes present in sudden deaths from acute trauma.

Primary, isolated, or interstitial myocarditis has been emphasized by several groups [9, 12, 16] as a cause of sudden death. For our discussion it is a particularly pertinent consideration since it is one cause that may occur at any period of infancy and childhood and seems to show no age predilection. Death may follow evidence of congestive failure or it may come suddenly and without warning. Case histories identical to that of our patient are to be found in the literature showing interstitial myocarditis as the cause of death. The histologic changes are characterized by extensive mononuclear cell infiltration of the myocardium with some changes extending to the endocardium and pericardium. There is a striking absence of polymorphonuclear leukocytes.

Viral studies are sometimes rewarding—the Coxsackie viruses being those most often recovered. In this regard it should be emphasized that facilities are not usually available to the examiner for identification of all of the possible viruses which might cause sudden death. Most studies so far have been best suited to the identification of the enteroviruses and for this reason they are more frequently reported as the agent involved. Though virologic investigations have not produced a high yield of information thus far, many possibilities remain unexplored and most investigators still regard this as a fertile field for investigation.

Bacteria cultured from the blood of the deceased at the time of postmortem examination must be evaluated cautiously and this evaluation has been generally unrewarding albeit occasionally very valuable. Postmortem bacterial contamination of blood and viscera are known to increase as the time between death and examination increases. Therefore, if our report today describes a sepsis, we will have to weigh that evidence against the organism's known pathogenicity, the manner of death, and the age of the subject, in coming to a decision as to its etiologic significance.

At some point in this discussion there are a group of diagnoses which I should like to mention only to condemn and eliminate them from our thinking: (1) status thymicolymphaticus, and (2) suffocation, except where mechanical means are available or the subject is extremely debilitated.

The thymus in the healthy infant and small child is characteristically quite a large organ, and all lymphoid tissue is present in substantial quantities. With the usual illness of sufficient severity and duration to produce death, this lymphoid tissue (including the thymus) rapidly decreases in size so that at the time of necropsy the thymic and lymph node tissue appear small. As might be anticipated when death occurs suddenly (trauma or disease), the thymus and lymph nodes are of normal size for a healthy individual. The latter finding was at one time interpreted as abnormal and termed status thymicolymphaticus. Investigators now agree that such a diagnosis is untenable and that suffocation due to tracheal compression by a large thymus does not occur.

There was also a time when physicians and mass news media made much of suffocation as the cause of death in previously well infants found dead in bed. Such a diagnosis implied neglect on the part of the parents and often created a serious emotional problem superimposed upon their grief. Little evidence was ever produced to support such an etiology. Investigations by Wooley[18] and others have clearly demonstrated that this is not a cause of death in previously well children when plastic bags, tracheal foreign bodies, ropes, and so forth can be excluded.

DR. NASH: Those toxic agents which are capable of producing rapid death are included in the following:

1. Some caustic substances upon ingestion may produce shock by inducing hemorrhage

or vasomotor collapse. The mechanisms underlying vasomotor collapse are still ill-defined. Among this group of substances producing shock are strong acids, alkalies, certain phenolic compounds, and a few heavy metals in high dosage such as mercuric chloride and, more commonly, ferrous sulfate.

2. Substances stimulating the central nervous system may result in rapid death by the production of convulsive seizures. In this class of compounds, those most commonly found in the household medicine cabinet are the antihistaminic agents. Poisoning by the antihistamines is difficult to antidote; supportive therapy is best employed. Other convulsant agents are more effectively treated by selection of a suitable barbiturate. Phenol and sodium fluoroacetate, terminally, may be convulsive agents.

3. Certain central nervous system depressants such as alcohol, barbiturates, and opiates may produce death, however, it is rare that this action would be rapid except in overwhelming doses.

4. Certain volatile compounds may produce quick lethal action. Carbon monoxide in high concentrations is an example. Inhalation of fumes from gasoline may result in ventricular fibrillation under certain circumstances.

DR. DAESCHNER: There are other interesting, but as yet unconfirmed (or denied) theories of sudden unexpected death. Parish and associates[10] noted that if they placed milk in the trachea of an unsensitized guinea pig he tolerated it well. If the animal were first sensitized to milk, he rapidly developed severe anaphylactic shock and died (a picture readily distinguishable from infant sudden deaths). However, if the sensitized guinea pig was lightly anesthetized before being challenged with intratracheal milk he experienced no anaphylactic symptoms, death being "sudden and peaceful." They had previously demonstrated that most infants' sera contained cows' milk antibodies and postulated that in the course of normal feedings and eructations the unnoticed aspiration of small amounts of milk probably was not unusual. In addition, they found the sera of infants who died suddenly and unexpectedly consistently contained higher milk antibody levels than the general population of infants. On these data they hypothesize that sudden death may be due to milk hypersensitivity precipitated by aspiration during sleep. Similar studies have not been reported by others and the final judgment as to the significance of milk antibodies to milk allergy remains uncertain.

Stowens[15] studied large numbers of sudden, unexpected infant deaths, through the Armed Forces Institute of Pathology, and was unable to find a satisfactory explanation of death in 82 per cent of the cases. The only consistent finding at autopsy in his studies (and also in those of most other workers) was an increased lung weight attributed to edema. He noted that many vomited and/or passed a large stool terminally. Efforts to isolate bacterial and viral agents from the lungs were relatively unsuccessful except in patients who showed symptoms before death and who had inflammatory (polymorphonuclear) cells by microscopic examination of lung tissue. He proposes that these patients died of a fulminating pulmonary edema due to autonomic hyper-reactivity. The latter represents an uninhibited viscero-visceral (vago-vagal) reflex set off by minor aspiration in a subject whose cerebral inhibitory centers for such reflexes are immature. While this is an interesting speculation, proof of such a mechanism will be hard to provide.

I have wandered rather freely over the many possibilities suggested by others to explain this perplexing and tragic problem. The age of our patient makes it likely that the pathologist has found an explanation. As I reviewed the medical literature, the experience of others suggests that most often we will find either (1) generalized viremia, (2) an extensive viral pneumonitis, or (3) an interstitial myocarditis. Since the patient was observed at death and no mention was made of dyspnea or tachypnea, I will choose the latter—an interstitial myocarditis.

Autopsy Findings

DR. JENNINGS: This autopsy was done at the request of the appropriate authorities at approximately 3½ hours after death. The external examination showed the lad to be rather slight but well-developed. The body measured 36 in. in length and was estimated to weigh 35 lb. The pupils were round, regular, and equal measuring 3 mm. in diameter. A few superficial bruises over the anterior aspects of the shins were thought to be sec-

ondary to incidental trauma and not significant in the demise of the patient. No evidence of other trauma was seen. Examination of the viscera showed no striking findings. The lungs were not increased in weight and did not appear to be either edematous or congested. The heart was somewhat heavy, weighing 78 gm. in contrast to a normal weight of 59 gm. The left ventricle was globular and firmly contracted while the right ventricle was rather flabby. No abnormalities could be distinguished in the heart, and though the myocardium was pale, it was uniformly firm. A superficial gastritis was apparent and the stomach contained about 200 gm. of semisolid, light tan, recently ingested food material. A single adult ascaris worm was found in the upper portion of the jejunum. The kidneys were slightly smaller than the normal weight but were not otherwise remarkable. The endocrine system and central nervous system appeared to be well within normal limits on gross examination.

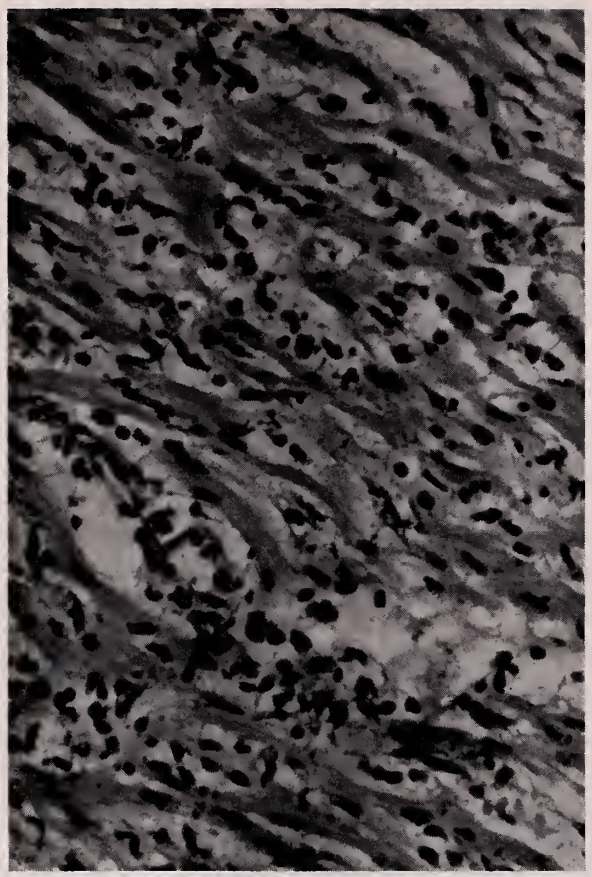
In view of the abrupt death of this patient and a history of reasonably good health until the morning of death, the possibility of toxic substance from the unpackaged cookies was considered and accordingly the remainder of these were confiscated from the seller and submitted to Dr. Nash for toxicologic examination.

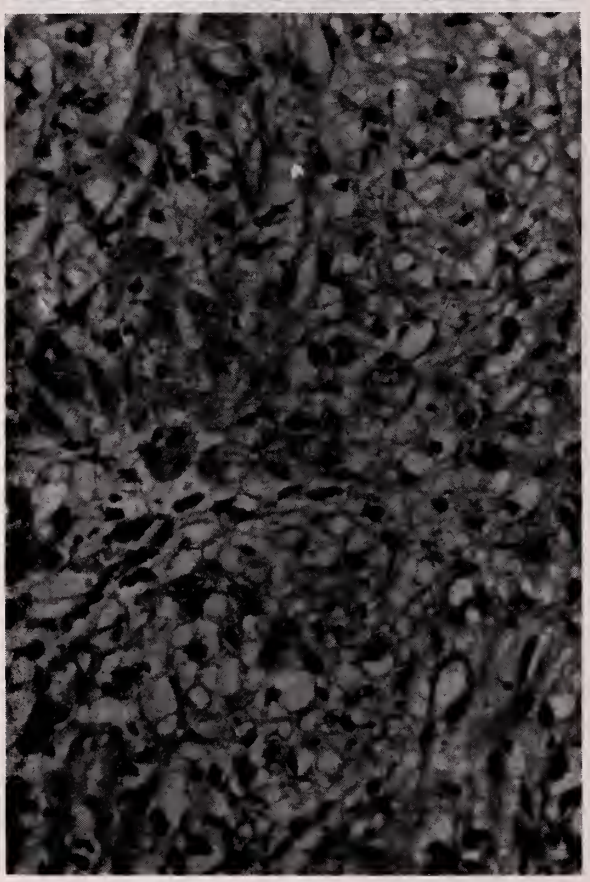
DR. NASH: On the morning following the gross autopsy, Dr. Jennings indicated that gross pathologic findings did not indicate a cause of death in this patient and accordingly brought a sample of the suspected cookies to the Department of Pharmacology and Toxicology for toxicologic evaluation, maintaining the proper chain of custody in delivering this sample. The cookies were fed ad libitum to several mice, none of which died until the third day, at which time one mouse was found dead. To exclude the possibility that the death of the mouse was caused by an inadequate nutritional state, more carefully controlled studies employing the addition of standard laboratory chow as a supplement to the diet of cookies resulted in no evidence of toxic substances in the suspected cookies. These latter feeding studies were continued for approximately two weeks. Only a very limited number of toxic agents will produce the fulminating death as exhibited in this case without prodromal signs or symptoms.

DR. JENNINGS: Bacteriologic studies obtained at the time of the autopsy showed a few small gram-positive rods isolated from the fluid at the base of the brain, after 72 hours' incubation. These were believed to represent contaminants, and a blood culture also demonstrated contamination with the isolation of *Paracolobactrum coliforme.* Virus studies performed by the State Department of Health failed to reveal any virus in either kidney or stomach wall samples which were submitted.

Only in the microscopic examination were we able to substantiate the cause of death in this patient. The entire myocardium was involved in a spectacular interstitial myocarditis. This myocarditis, as shown in Figs. 1 and 2, was about equal in all areas of the heart and consisted primarily of a heavy interstitial infiltration of lymphocytes and a few plasma cells. Large tissue mononuclear cells were seen and there were focal areas of degeneration of the myocardial fibers, as shown in Fig. 3. It was noted that while the necrosis was fairly marked in some areas it involved primarily individual muscle fibers rather than groups of myocardial fibers.

Fig. 1. Interstitial myocarditis with diffuse infiltration of lymphocytes and a few plasma cells is seen. The infiltrate is of about equal severity throughout the myocardium. X 150





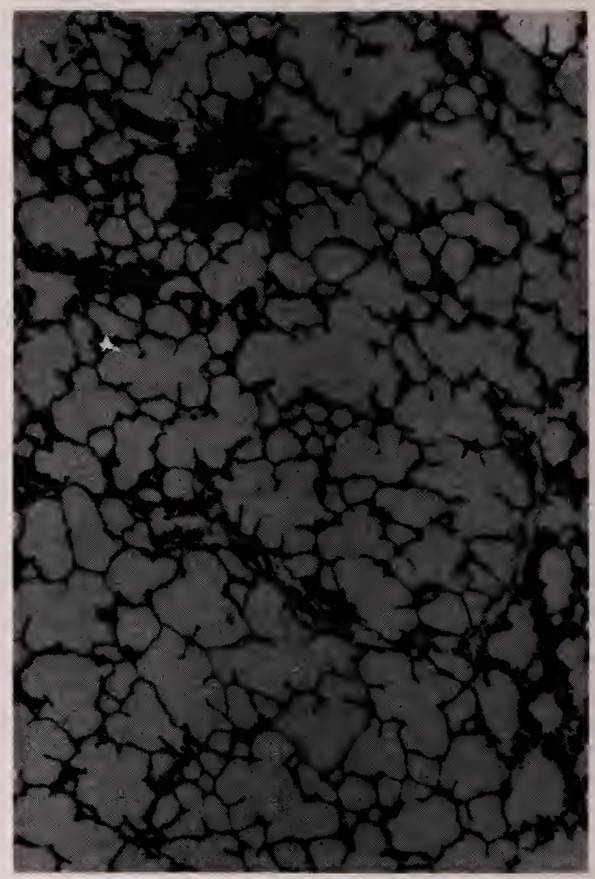


Fig. 2. (top left) The cellular infiltration consists primarily of lymphocytes with a few plasma cells. Little myocardial degeneration is seen in most areas. X 500

Fig. 3. (bottom left) Focal areas of degenerative changes in myocardium showed vacuolization of cytoplasm with loss of striations. Large mononuclear cell infiltration is present in these focal areas. X 500

Fig. 4. (top right) No edema or congestion was present in the lungs; the lungs appeared normal on gross and microscopic examination. X 150

Neither the lungs, as seen in Fig. 4, nor the liver, Fig. 5, showed any significant evidence of congestion. This lack of congestion in either of these areas indicated that the mechanism of death was rapid without a significant prodromal period of congestive heart failure . It was felt that this case represented an isolated myocarditis of the Fiedler type.

Comment

DR. JENNINGS: While it is difficult to ascertain the etiology of the severe myocarditis observed in this patient, it is probably a viral interstitial myocarditis. The fact that the inflammation is limited to the heart suggests rather strongly that this is indeed a

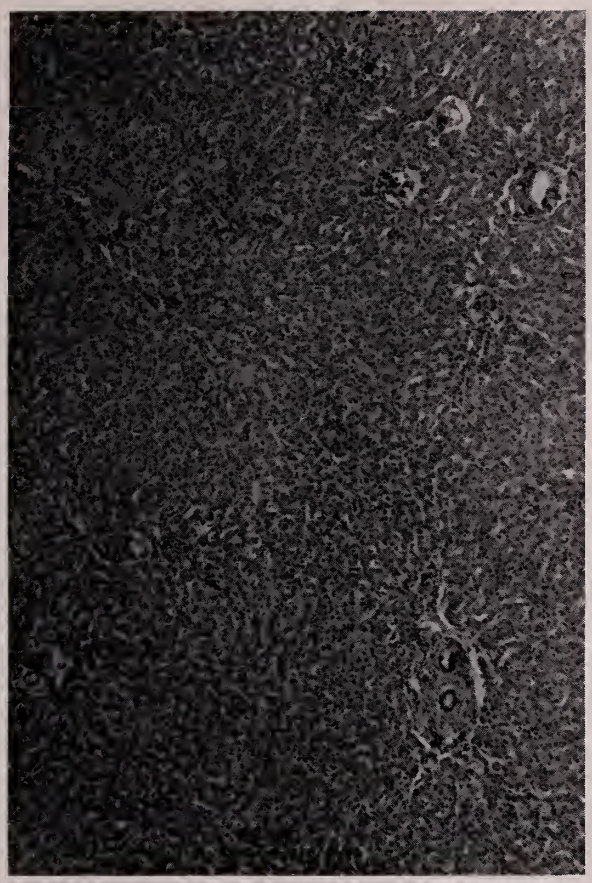


Fig. 5. The liver showed no edema or congestion and appeared to be within normal limits. X 150

Fiedler's type of isolated myocarditis rather than a Coxsackie myocarditis as described by Hosier and Newton.[7] While it is realized that necrosis is rare in Fiedler's myocarditis, it must be emphasized that necrosis as seen in this case is focal and is not extensive, involving primarily individual myocardial fibers.

DR. DAESCHNER: Our responsibilities in this conference would be incomplete if we neglect to mention the influence of sudden, unexpected death on the family. The parents, particularly the mother or the member present when the child dies, often develop a severe and enduring sense of guilt. Terms such as smothering or suffocation only tend to reinforce their fears. Postmortem examination is important, but permission must be sought with care lest we imply parental neglect. Similarly, our interviews and home visits (which are essential to the history) must be conducted in such a manner that a guilt complex is not created. Our responsibility as physicians is to leave the parents with a clear conscience so that their grief will not be compounded.

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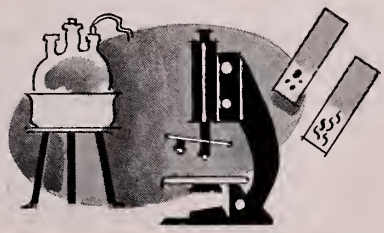


NEWS

Physicians Have Vital Part In Medical Self-Help Training

When disaster strikes, families and individuals must be self-sufficient in order to survive. They may be deprived of physicians' services for hours, days, or weeks. Physicians who recognize the physical impossibility of properly caring for their patients under disaster conditions have initiated Medical Self-Help as a means of filling the needs.

If America is to survive, it will be because individuals have survived; therefore, every individual and family has an essential role in increasing his chances of survival by preparing himself for such an emergency. This can best be done by taking instruction in Medical Self-Help. To relieve the decreased numbers of the medical profession from impossible tasks, it is needful that they promote self-help throughout the state and local areas. This can be done by promoting interest through their local medical society and by interesting other organizations in such training. Information has been sent to all physicians in the state, and further information will be provided by the Texas State Department of Health, or by the local medical society.

—J. E. PEAVY, M.D., Austin,
Commissioner of Health.

WHEN DISASTER strikes . . . what then? Casualties, pestilences, illnesses increase and professional and medical resources are severely overtaxed. This is the situation that the Medical Self-Help Program of the Texas Medical Association and the State Department of Health is intended to meet.

Medical Self-Help is the training program designed by the AMA in cooperation with the Office of Civil Defense Mobilization to cope with the inevitable emergencies of natural phenomena or nuclear attack. It is a part of the national plan for civil defense.

The program is locally organized and sponsored by the county medical society, civil defense office, health unit, or Red Cross. Instructors are appointed by the local group. The training may be given as an extension or addition to the first-aid courses taught by American Red Cross personnel, the additions including subjects that go beyond first-aid, such as fallout protection, nursing care, infant care, and emergency childbirth. The last is of great importance in total disaster. It is highly desirable that physicians take part or be in attendance at most of the sessions.

Training in Medical Self-Help is not started in a community until the medical society of that community has given its approval. This has been and will continue to be the policy of the Texas State Department of Health.

Progress to Date

In Texas, the program to date has received valuable support from the Texas Medical Association, along with various county and local societies and their appointed committees. Many courses have been given by the local American Red Cross personnel with the supplemental Medical Self-Help lessons. By direction of the Governor, the Texas State Department of Health is assigned the primary responsibility for Medical Self-Help in the state.

Courses have been held throughout the state, and some 10,625 have been registered and certificates issued to 8,497. Some 100 teachers have been used, among them a num-

ber of physicians who have served as instructors, advisors, and consultants. The presence of a physician adds greatly to the interest and lends authenticity to the training. Attendance is increased when a physician is present; this has been the observation of teachers in all parts of the state. The mere fact that a physician considers the course important enough to spend his time adds credence to its importance.

Physician interest is increasing rapidly. In four days, an offer of waiting room folders about Medical Self-Help brought requests from physicians for 11,500 copies. The demand continues as more physicians become aware of the folder and the program.

Medical Self-Help Training courses have been conducted in communities of all sizes and under all conditions. In several instances the program has already been established in the school system, and many hundreds of high school students have been graduated. A number of larger industries have undertaken one or more training sessions for their employees, and it is expected that many others will follow as the availability of Medical Self-Help becomes widely known. The newest school system added is Beaumont, where 6,500 are enrolled in Medical Self-Help courses for January and February, 1964. The Gulf States Utilities Company has already trained 2,000 of its employees and will train 2,000 more. Other groups which have completed the training are city, federal, and state personnel. The training set-up depends on the size of the community and the extent of training needs.

Normal procedure consists of a meeting of local medical society and civil defense personnel to discuss programming, facilities, and instructors. When these matters are settled, publicity and recruiting are planned, and the kits are requested from the State Department of Health.

Although the Medical Self-Help Program has been in existence only a few months, it has already proved its value in numerous instances in Texas. For example, in Big Bend National Park a forest ranger who had taken the course revived a drowning victim by use of mouth-to-mouth resuscitation, and an Austin physician who sustained a broken leg with lacerations and bleeding received help from companions who had studied Medical Self-Help.

Scope of the Course

The Medical Self-Help Training Program consists of 11 lessons totaling 16 hours, usually given in sessions of two hours each week. Lessons are divided as follows:

1. *Radioactive Fallout and Shelter* deals with the problem of nuclear blast and radioactive fallout and describes protective measures.
2. *Healthful Living in Emergencies* provides instruction for personal and environmental sanitation in shelters and at home under disaster conditions. Instruction is given on food and water problems.
3. *Artificial Respiration* gives instruction and technique in mouth-to-mouth resuscitation and the back-pressure arm-lift method.
4. *Bleeding and Bandaging* teaches methods of controlling bleeding and develops skills in bandaging different types of wounds.
5. *Fractures and Splinting* teaches an understanding of fractures, how to recognize them, and the complications of fractures. It teaches splinting and immobilization of fractures. Stress is placed on improvisation in splinting.
6. *Transportation of the Wounded* develops knowledge of the methods used in transporting the sick and injured. It also emphasizes errors which, when made, may accentuate the existing injury.
7. *Burns* develops an understanding of the types and severity of burns, and the early treatment of burns. Burns will constitute perhaps the greatest health problem in nuclear disasters.
8. *Shock* provides an understanding of shock, its causes, seriousness, and prevention and treatment.
9. *Nursing Care of the Sick and Injured* provides knowledge of the care of general illness with emphasis on symptomatic treatment.
10. *Infant and Child Care* develops the ability to care for the newborn, the premature infant, infants, and preschool, preadolescent, and adolescent children under emergency shelter conditions. Their emotional and psychological problems and specific problems of disease are discussed.
11. *Emergency Childbirth* demonstrates the physiology of childbirth and the fundamental techniques in assisting the mother with a normal delivery.

The complete training kit for instructors includes a projector and screen, films, leader's instructions, student handbooks, quiz sets, and graduation certificates for groups of 25. The kit is available free of charge to course leaders, and replacement material is furnished.

How Self-Help Began

The project originated with a contract between the AMA and the federal government. The AMA appointed a Commission on National Emergency Medical Care to conduct a study of that subject. Its report, approved in 1959, was submitted to the Office of Civil Defense.

Recommendations included, first, establishment of a national medical planning group to prepare a complete national medical plan for the care of mass casualties and for the medical support of surviving noncasualties, and to prepare programs to train persons normally engaged in medical and health activities, and the general population, for mass casualty conditions.

The second recommendation was to establish a Medical Advisory Council, the purpose of which, among other things, was to train all segments of the population in appropriate fields.

Finally, the Commission recommended establishment of a National Medical Directorate to succeed the planning group when its work was finished.

"It is imperative that the general public receive training and become proficient in the application of first-aid and self-aid procedures," the Commission stated in its summary report.

The training responsibility of the medical profession, as pointed out by the Commission, states: "The general public be systematically trained in the appropriate aspects of all-out war, sanitation and first-aid and self-aid procedures, through the media of newspaper and magazine articles, radio and television facilities, classroom instruction in the case of school children, college and university students, and organized classes for adults (such classes taught by physicians, other professional health personnel or qualified lay personnel, as appropriate), as will permit the application of proper self-aid and first-aid measures and the institution of adequate sanitation precautions under mass casualty conditions."

The Medical Self-Help Program is a part of the plan worked out, as a result of this report, through the U. S. Public Health Service and the Office of Civil Defense. Intensive research revealed information that an individual should know about medical care in an emergency; and the medical self-help training was designed specifically to meet these needs.

For those who desire material for their

waiting rooms, or information on initiating Medical Self-Help Programs in their communities, the following order blanks are provided.

Mr. Phil Gauss
State Department of Health
1100 West 49th Street
Austin, Texas

Please send me_____copies of the pamphlet, "Disaster Strikes," for my waiting room.

Name (Please Print)_____

Address _____

_____

Mr. Phil Gauss
State Department of Health
1100 West 49th Street
Austin, Texas

Our county medical society wishes to sponsor a Medical Self-Help Training Course. Please send complete information to:

Name (Please Print)_____

Address _____

_____

Name of County Society_____

Physicians Benevolent Fund Contributions Received

During the first month of solicitation for the Physicians Benevolent Fund of the Texas Medical Association, donations totaling $3,872.50 were received.

The Fund, started in 1962 by Dr. May Owen, Past President of TMA, with her donation of $2,500, is to help needy, destitute physicians and their families.

Administered by the Board of Trustees, the Physicians Benevolent Fund will be supported by voluntary donations from physicians, gifts from other private sources, and bequests.

The solicitation campaign at the county society level has resulted in 100 per cent contributions from the members of the Howard-Martin-Glasscock Counties Medical Society, Johnson County Medical Society, Nolan-Fisher-Mitchell-Borden-Kent-Scurry-Stonewall Counties Medical Society, Coryell County Medical Society, and Denton County Medical Society. Several county societies are nearing the 100 per cent mark, and others have voted to donate to the Fund at the time dues are paid.

Interested physicians may send their contributions to the Physicians Benevolent Fund, Texas Medical Association, 1801 North Lamar Blvd., Austin, Tex. 78701.

Football Players Health Guarded in Hot Game

In spite of 114-119 degree heat, players in a Los Angeles football game between the University of Southern California and the University of Oklahoma suffered no ill effects.

Many medical precautions were initiated during the Sept. 28 contest, reports the *AMA News* in its Oct. 28 issue.

Some of the precautions included placing canopies over the players' benches to ward off the sun, maintaining supplies of ice water and iced towels to cool the players during rest periods, keeping available emergency intravenous therapy and oxygen (it was not used), instructing referees to call frequent time outs, and setting up a giant wind fan to stir the air on the playing field (this was not used).

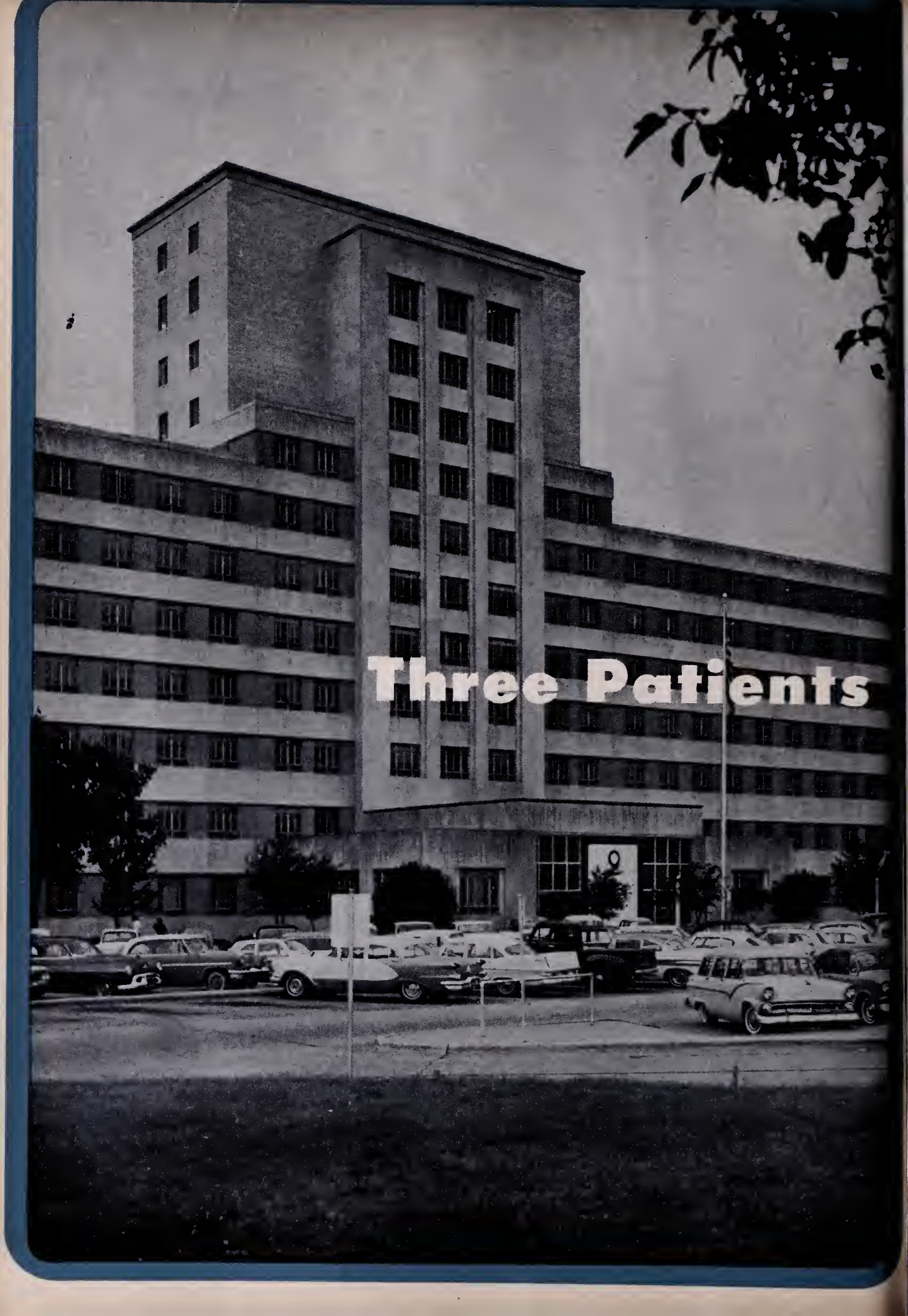
Members of both teams took either salt tablets or salt solution, and physicians were on hand to take the temperatures of players as they came off the field, making certain no player was in danger of heat prostration.

Some 39,300 perspiring fans watched Oklahoma win, 17-12.

Application Cards Prepared For AMA Affiliate Members

The Judicial Council has prepared application cards for persons seeking affiliate membership in the AMA, reports the *AMA News* Oct. 28. As a result of changes made in the AMA Constitution and Bylaws at the annual meeting in Atlantic City last June, the Judicial Council has to approve and nominate affiliate members.

Application forms have been prepared for medical missionaries, dentists and pharmacists, teachers of medicine or of sciences allied to medicine, and for physician members of national medical societies of foreign countries adjacent to the United States.


Three Patients

at Parkland

PARKLAND Memorial Hospital, Dallas, treats an average of 272 emergency cases a day. It is adjacent to and is the major teaching hospital for the University of Texas Southwestern Medical School. It is staffed by the faculty of the medical school and has 150 interns and residents in all medical specialties. It is a modern hospital, well equipped, one of which any community might be proud. Today—and for none of these reasons—Parkland has a new reputation all over the world, and historians are typing its name into manuscripts that will be textbooks for generations to come. This has happened because three particular gunshot victims were carried there out of the bright November sunlight, two to die and the third to leave by wheel chair almost two weeks later, his arm in a sling.

Many Texas physicians have visited Parkland hospital; many have worked or trained there. Members of the Parkland staff are their acquaintances and friends. Many Texas physicians know personally the surviving gunshot victim, Gov. John Connally; some personally knew President John F. Kennedy who died in Trauma Room 1; perhaps a few even knew Lee Harvey Oswald, the man charged by Dallas authorities with the assassination of the President and who was himself shot two days later.

The assassination of President Kennedy, the wounding of Governor Connally, and the fatal shooting of Oswald are events of profound import to people everywhere, but they have special, personal meaning for Texans. So because a Texas hospital and Texas physicians figured prominently in this tragedy, the *Texas State Journal of Medicine* records for its readers of the medical profession a full account of treatment given a never-to-be-forgotten trio.

When President John F. Kennedy in a moribund condition entered Parkland on Nov. 22, there was never opportunity for medical history taking. Such a history, had it been taken, would have shown that the patient "had survived several illnesses, the dangers of war, the rigor of exposure in icy waters, and . . . had waged grueling electoral campaigns in spite of a serious and painful back injury."*

Parkland records show that the President arrived at the emergency room sometime after 12:30 p.m. (There is conflict as to the exact moment.) At 1 p.m. Dr. William Kemp Clark, associate professor and chairman of the Division of Neurosurgery of the University of Texas Southwestern Medical School, declared him dead. During the interim of less than 30 minutes, continuous resuscitative efforts were made.

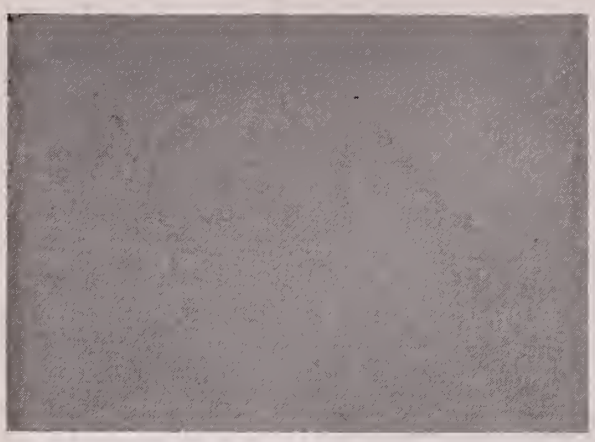
Later that day, several attending physicians filed reports. The following identifies these physicians and gives the gist of their reports:

Charles J. Carrico.—Dr. Carrico was the first physician to see the President. A 1961 graduate of Southwestern Medical School, he is 28 and a resident in surgery at Parkland.

He reported that when the patient entered the emergency room on an ambulance carriage he had slow agonal respiratory efforts and occasional cardiac beats detectable by auscultation. Two external wounds were noted; one a small wound of the anterior neck in the lower one third. The other wound had caused avulsion of the occipitoparietal calvarium and shredded brain tissue was present with profuse oozing. No pulse or blood pressure were present. Pupils were bilaterally dilated and fixed. A cuffed endotracheal tube was inserted through the laryngoscope. A ragged wound of the trachea was seen immediately below the larynx. The tube was advanced past the laceration and the cuff inflated. Respiration was instituted using a respirator assistor on automatic cycling. Concurrently, an intravenous infusion of lactated Ringer's solution was begun via catheter placed in the right leg. Blood was drawn for typing and crossmatching. Type O Rh negative blood was obtained immediately.

In view of the tracheal injury and diminished breath sounds in the right chest, tracheostomy was performed by Dr. Malcolm O. Perry and bilateral chest tubes inserted. A second intravenous infusion was begun in the left arm. In addition, Dr. M. T. Jenkins began respiration with the anesthesia machine, cardiac monitor and stimulator attached. Solu-Cortef (300 mg.) was given intravenously. Despite those measures, blood

*Profile in Courage, *MD* Medical Newsmagazine 7:91 (Dec.) 1963.



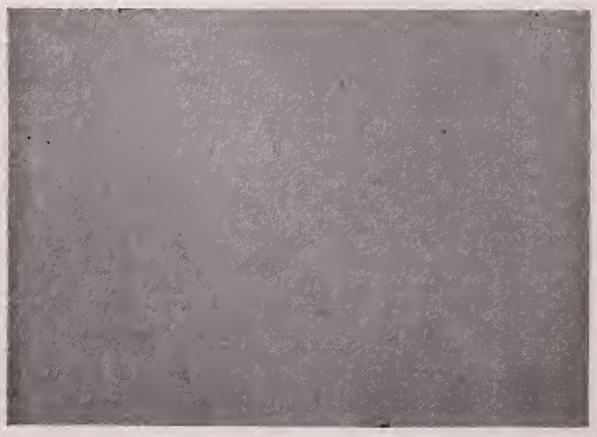
Editor's Note.—One of the purposes of the *Texas State Journal of Medicine* has been to serve as a historical record of events affecting Texas medicine.

Although the advisors and editors of the *Journal* believe that this record of the medical treatment of President John F. Kennedy, Gov. John Connally, and Lee Harvey Oswald is also of current interest, it is their sincere belief that the historical importance of the record is of even greater value.

The record of medical treatment of three earlier assassinated Presidents of the United States (page 74) undoubtedly has provided students of gunshot wounds a significant record of the treatment available during the particular points in history at which these men died. A review of their treatment may also have aided in the search for better treatment of other victims.

There were several forms in which this record could have been carried, some of which might have provided easier reading, usually an important goal of this *Journal*. The advisors to the *Journal* considered providing a composite review or a reconstructed scene of the events in the operating rooms at Parkland, but to some extent other publications have done this and it was felt that the impressions of the individual participating physicians, written in a period of strain, without consideration for rhetoric, was perhaps of greatest value to medical history.

Therefore, the *Journal* presents the reports showing the separate views of the physicians in charge to let the reader see for himself part of history as it was recorded a few hours after the events of Nov. 22 and Nov. 24. Like the various aspects of the four gospels, as reported by Matthew, Mark, Luke, and John, it is hoped that this form will provide an insight not possible in a more concise, composite presentation.



pressure never returned. Only brief electrocardiographic evidence of cardiac activity was obtained.

Malcolm O. Perry.—Dr. Perry is an assistant professor of surgery at Southwestern Medical School from which he received his degree in 1955. He is 34 years old and was certified by the American Board of Surgery in 1963.

At the time of initial examination of the President, Dr. Perry has stated, the patient was noted to be nonresponsive. His eyes were deviated and the pupils dilated. A considerable quantity of blood was noted on the patient, the carriage, and the floor. A small wound was noted in the midline of the neck, in the lower third anteriorly. It was exuding blood slowly. A large wound of the right posterior cranium was noted, exposing severely lacerated brain. Brain tissue was noted in the blood at the head of the carriage.

Pulse or heart beat were not detectable, but slow spasmodic respiration was noted. An endotracheal tube was in place and respiration was being controlled. An intravenous infusion was being placed in the leg. While additional venesections were done to administer fluids and blood, a tracheostomy was effected. A right lateral injury to the trachea was noted. The cuffed tracheostomy tube was put in place as the endotracheal tube was withdrawn and respirations continued. Closed chest cardiac massage was instituted after placement of sealed-drainage chest tubes, but without benefit. When electrocardiogram evaluation revealed that no detectable electrical activity existed in the heart, resuscitative attempts were abandoned. The team of physicians determined that the patient had expired.

Charles R. Baxter.—Dr. Baxter is an assistant professor of surgery at Southwestern Medical School where he first arrived as a medical student in 1950. Except for two years away in the Army he has been at Southwestern and Parkland ever since, moving up from student to intern to resident to faculty member. He is 34 and was certified by the American Board of Surgery in 1963.

Recalling his attendance to President Kennedy, he says he learned at approximately 12:35 that the President was on the way to the emergency room and that he had been shot. When Dr. Baxter arrived in the emergency room, he found an endotracheal tube

in place and respirations being assisted. A left chest tube was being inserted and cutdowns were functioning in one leg and in the left arm. The President had a wound in the midline of the neck. On first observation of the other wounds, portions of the right temporal and occipital bones were missing and some of the brain was lying on the table. The rest of the brain was extensively macerated and contused. The pupils were fixed and deviated laterally and were dilated. No pulse was detectable and ineffectual respirations were being assisted. A tracheostomy was performed by Dr. Perry and Dr. Baxter and a chest tube was inserted into the right chest (second interspace anteriorly). Meanwhile one pint of O negative blood was administered without response. When all of these measures were complete, no heart beat could be detected. Closed chest massage was performed until a cardioscope could be attached. Brief cardiac activity was obtained followed by no activity. Due to the extensive and irreparable brain damage which existed and since there were no signs of life, no further attempts were made at resuscitation.

Robert N. McClelland.—Dr. McClelland, 34, assistant professor of surgery at Southwestern Medical School, is a graduate of the University of Texas Medical Branch in Galveston. He has served with the Air Force in Germany and was certified by the American Board of Surgery in 1963.

Regarding the assassination of President Kennedy, Dr. McClelland says that at approximately 12:35 p.m. he was called from the second floor of the hospital to the emergency room. When he arrived, President Kennedy was being attended by Drs. Perry, Baxter, Carrico, and Ronald Jones, chief resident in surgery. The President was at that time comatose from a massive gunshot wound of the head with a fragment wound of the trachea. An endotracheal tube had been placed and assisted respiration started by Dr. Carrico who was on duty in the emergency room when the President arrived. Drs. Perry, Baxter, and McClelland performed a tracheostomy for respiratory distress and tracheal injury. Dr. Jones and Dr. Paul Peters, assistant professor of surgery, inserted bilateral anterior chest tubes for pneumothoraces secondary to the tracheomediastinal injury. Dr. Jones and assistants had started three cutdowns, giving blood and fluids immediately. In spite of this, the President was pronounced dead at 1:00 p.m. by Dr. Clark, the neurosurgeon, who arrived immediately after Dr. McClelland. The cause of death, according to Dr. McClelland was the massive head and brain injury from a gunshot wound of the right side of the head. The President was pronounced dead after external cardiac massage failed and electrocardiographic activity was gone.

Fouad A. Bashour.—Dr. Bashour received his medical education at the University of Beirut School of Medicine in Lebanon. He is 39 and an associate professor of medicine in cardiology at Southwestern Medical School.

At 12:50 p.m. Dr. Bashour was called from the first floor of the hospital and told that President Kennedy had been shot. He and Dr. Donald Seldin, professor and chairman of the Department of Internal Medicine, went to the emergency room. Upon examination, they found that the President had no pulsations, no heart beats, no blood pressure. The oscilloscope showed a complete standstill. The President was declared dead at 1:00 p.m.

William Kemp Clark.—Dr. Clark is associate professor and chairman of the Division of Neurosurgery at Southwestern Medical School. The 38-year-old physician has done research on head injuries and has been at Southwestern since 1956.

He reports this account of the President's treatment:

The President arrived at the emergency room entrance in the back seat of his limousine. Governor Connally of Texas was also in this car. The first physician to see the President was Dr. Carrico.

Dr. Carrico noted the President to have slow, agonal respiratory efforts. He could hear a heart beat but found no pulse or blood pressure. Two external wounds, one in the lower third of the anterior neck, the other in the occipital region of the skull, were noted. Through the head wound, blood and brain were extruding. Dr. Carrico inserted a cuffed endotracheal tube and while doing so, he noted a ragged wound of the trachea immediately below the larynx.

At this time, Drs. Perry, Baxter, and Jones arrived. Immediately thereafter, Dr. Jenkins and Drs. A. H. Giesecke, Jr., and Jackie H. Hunt, two other staff anesthesiologists, arrived. The endotracheal tube had been connected to a respirator to assist the President's breathing. An anesthesia machine was substituted for this by Dr. Jen-

kins. Only 100 per cent oxygen was administered.

A cutdown was performed in the right ankle, and a polyethylene catheter inserted in the vein. An infusion of lactated Ringer's solution was begun. Blood was drawn for typing and crossmatching, but unmatched type O Rh negative blood was immediately obtained and begun. Hydrocortisone (300 mg.) was added to the intravenous fluids.

Dr. McClelland arrived to help in the President's care. Drs. Perry, Baxter, and McClelland did a tracheostomy. Considerable quantities of blood were present in the President's oral pharynx. At this time, Dr. Peters and Dr. Clark arrived.

Dr. Clark noted that the President had bled profusely from the back of the head. There was a large (3 by 3 cm.) amount of cerebral tissue present on the cart. There was a smaller amount of cerebellar tissue present also.

The tracheostomy was completed and the endotracheal tube was withdrawn. Suction was used to remove blood in the oral pharynx. A nasogastric tube was passed into the stomach. Because of the likelihood of mediastinal injury, anterior chest tubes were placed in both pleural spaces. These were connected to sealed underwater drainage.

Neurological examination revealed the President's pupils to be widely dilated and fixed to light. His eyes were divergent, being deviated outward; a skew deviation from the horizontal was present. No deep tendon reflexes or spontaneous movements were found.

When Dr. Clark noted that there was no carotid pulse, he began closed chest massage. A pulse was obtained at the carotid and femoral levels.

Dr. Perry then took over the cardiac massage so that Dr. Clark could evaluate the head wound.

There was a large wound beginning in the right occiput extending into the parietal region. Much of the right posterior skull, at brief examination, appeared gone. The previously described extruding brain was present. Profuse bleeding had occurred and 1500 cc. of blood was estimated to be on the drapes and floor of the emergency operating room. Both cerebral and cerebellar tissue were extruding from the wound.

By this time an electrocardiograph was hooked up. There was brief electrical activity of the heart which soon stopped.

The President was pronounced dead at 1:00 p.m. by Dr. Clark.

M. T. Jenkins.—Dr. Jenkins is professor and chairman of the Department of Anesthesiology at Southwestern Medical School. He is 46, a graduate of the University of Texas Medical Branch in Galveston, and was certified by the American Board of Anesthesiology in 1952. During World War II he served in the Navy as a lieutenant commander.

When Dr. Jenkins was notified that the President was being brought to the emergency room at Parkland, he dispatched Drs. Giesecke and Hunt with an anesthesia machine and resuscitative equipment to the major surgical emergency room area. He ran downstairs to find upon his arrival in the emergency operating room that Dr. Carrico had begun resuscitative efforts by introducing an orotracheal tube, connecting it for controlled ventilation to a Bennett intermittent positive pressure breathing apparatus. Drs. Baxter, Perry, and McClelland arrived at the same time and began a tracheostomy and started the insertion of a right chest tube, since there was also obvious tracheal and chest damage. Drs. Peters and Clark arrived simultaneously and immediately thereafter assisted respectively with the insertion of the right chest tube and with manual closed chest cardiac compression to assure circulation. Dr. Jenkins believes it evidence of the clear thinking of the resuscitative team that the patient received 300 mg. hydrocortisone intravenously in the first few minutes.

For better control of artificial ventilation, Dr. Jenkins exchanged the intermittent positive pressure breathing apparatus for an anesthesia machine and continued artificial ventilation. Dr. Gene Akin, a resident in anesthesiology, and Dr. Giesecke connected a cardioscope to determine cardiac activity.

During the progress of these activities, the emergency room cart was elevated at the feet in order to provide a Trendelenburg position, a venous cutdown was performed on the right saphenous vein, and additional fluids were begun in a vein in the left forearm while blood was ordered from the blood bank. All of these activities were completed by approximately 12:50 at which time external cardiac massage was still being carried out effectively by Dr. Clark as judged

by a palpable peripheral pulse. Despite these measures there was only brief electrocardiographic evidence of cardiac activity.

These described resuscitative activities were indicated as of first importance, and after they were carried out, attention was turned to other evidences of injury. There was a great laceration on the right side of the head (temporal and occipital), causing a great defect in the skull plate so that there was herniation and laceration of great areas of the brain, even to the extent that part of the right cerebellum had protruded from the wound. There were also fragmented sections of brain on the drapes of the emergency room cart. With the institution of adequate cardiac compression, there was a great flow of blood from the cranial cavity, indicating that there was much vascular damage as well as brain tissue damage. President Kennedy was pronounced dead at 1 p.m.

It is Dr. Jenkins' personal feeling that all methods of resuscitation were instituted expeditiously and efficiently. However, he says, the cranial and intracranial damage was of such magnitude as to cause irreversible damage.

John Connally

The health of the Governor of Texas has been news on at least two occasions before his serious injury on Nov. 22. As Secretary of the Navy, while on a visit to Austin, John Connally was accidentally stabbed in the eye by a bayonet as he reviewed ROTC troops; and shortly after his inauguration as Governor, he underwent surgery for hernia.

When he arrived with the President at Parkland's emergency entrance, the Governor had been wounded by a bullet that had pierced his chest, arm, and thigh. In a recent interview with editors of the *Journal*, he recalled raising himself up in the back seat of the limousine but was unable to raise himself onto the stretcher. Mrs. Connally, the Governor's wife, also present at the interview, explained that her husband, still conscious, was then aided in getting onto the stretcher. He was then taken into Trauma Room 2, across the hall from Trauma Room 1 where the President had been carried.

Soon afterwards he was taken to an operating room. Later, four Parkland physicians described the treatment given the Governor.

Text of Note to Hospital From Governor

November 30, 1963

TO THE STAFF AND PERSONNEL
OF PARKLAND HOSPITAL:

Words cannot adequately express my persanal appreciation for the care, treatment and supreme thoughtfulness my family and I have received from all of you.

These have been trying days far everyane cannected with this great hospital, but you have risen to the occasion with a dedication to duty which merits the highest confidence and praise. Parkland has proved again that it is ane of the finest institutions af its kind in the world.

I will probably nat have the opportunity to thank each of you individually, so I hape yau will accept this message as an expression af my heartfelt gratitude. I speak also for Mrs. Connally, the children, other members of my family, my staff and the officers of the Department af Public Safety who have received so many courtesies from Parkland emplayees. We will always remember yaur help and consideration.

Sincerely,
(Signed)
JOHN CONNALLY

Robert R. Shaw.—Dr. Shaw, professor of thoracic surgery at Southwestern Medical School, returned to Dallas last summer after spending a year and a half in Kabul, Afghanistan, as head of the Medico team there. A graduate of the University of Michigan Medical School at Ann Arbor, he is 58. He was certified by The Board of Thoracic Surgery in 1948, and has practiced in Dallas since 1938.

Dr. Shaw performed a thoracotomy, removed rib fragments, and debrided the chest wound. Diagnosis of the chest condition was gunshot wound of the chest with comminuted fracture of the fifth rib, laceration of the middle lobe, and hematoma of the lower lobe of the right lung.

The Governor was brought to the operating room from the emergency operating room where a sucking wound of the right chest had been partially controlled by an occlusive dressing supported by manual pressure. A tube had been placed through the second interspace of the right chest in the mid-clavicular line and connected to a water-seal bottle to evacuate the hemopneumothorax. An intravenous infusion of lactated Ringer's solution had already been started. As soon as the patient was positioned on the operating table the anesthesia was induced by Dr. Giesecke and an endotracheal tube was put in place.

As soon as it was possible to control respiration with positive pressure, the occlu-

sive dressing was taken from the right chest and the extent of the wound more carefully determined. It was found that the bullet had made a wound of entrance just lateral to the right scapula, close to the axilla, had passed through the latissimus dorsi muscle, shattered approximately 10 cm. of the lateral and anterior portion of the right fifth rib, and emerged below the right nipple. The wound of entrance was approximately 3 cm. in its longest diameter and the wound of exit was a ragged wound approximately 5 cm. in its greatest diameter. The skin and subcutaneous tissue over the path of the missile moved in a paradoxical manner with respiration indicating softening of the chest.

The skin of the whole area was carefully cleansed with Phisohex and iodine. The entire area, including the wound of entrance and wound of exit, was draped partially excluding the wound of entrance for the first part of the operation.

An elliptical incision was made around the wound of exit removing the torn edges of the skin and the damaged subcutaneous tissue. The incision was then carried in a downward curve up toward the right axilla so as not to have the skin incision over the actual path of the missile through the chest wall. This incision was carried down through the subcutaneous tissue to expose the serratus anterior muscle and the anterior border of the latissimus dorsi muscle.

The fragmented and damaged portions of the serratus anterior muscle were excised. Small rib fragments that were adhering to periosteal tags were carefully removed preserving as much periosteum as possible. The fourth and fifth intercostal muscle bundles were not appreciably damaged. The ragged ends of the damaged fifth rib were cleaned with the rongeur. The pleura had been torn open by the secondary missiles created by the fragmented fifth rib.

The wound was widely opened and exposure was maintained with a self-retaining retractor. Approximately 200 cc. of clot and liquid blood was removed from the pleural cavity. The middle lobe had a linear rent starting at its peripheral edge, going down toward the hilum separating the lobe into two segments. There was an open bronchus in the depth of this laceration. Since the vascularity and the bronchial connections to the lobe were intact it was decided to repair the lobe rather than to remove it. The repair was accomplished with a running suture of #000 chromic gut on an atraumatic needle closing both pleural surfaces as well as two running sutures approximating the tissue of the central portion of the lobe. This almost completely sealed off the air leaks which were evident in the torn portion of the lobe. The lower lobe was next examined and found to be engorged with blood and at one point a laceration allowed the oozing of blood from the lobe. This laceration had undoubtedly been caused by a rib fragment. The laceration was closed with a single suture of #000 chromic gut on an atraumatic needle. The right pleural cavity was now carefully examined. Small rib fragments were removed. The diaphragm was found to be uninjured. There was no evidence of injury to the mediastinum and its contents. Hemostasis had been accomplished within the pleural cavity with the repair of the middle lobe and the suturing of the laceration in the lower lobe. The upper lobe was found to be uninjured.

The drain which had previously been placed in the second interspace in the midclavicular line was found to be longer than necessary so approximately 10 cm. of it was cut away and the remaining portion was fenestrated with two additional openings. An additional drain was placed through a stab wound in the eighth interspace in the posterior axillary line. Both these drains were then connected to a waterseal bottle.

The fourth and fifth intercostal muscles were then approximated with interrupted sutures of #0 chromic gut. The remaining portion of the serratus anterior muscle was then approximated across the closure of the intercostal muscle. The laceration of the latissimus dorsi muscle on its inner surface was then closed with several interrupted sutures of #0 chromic gut.

Before closing the subcutaneous tissue, 1,000,000 units of penicillin and 1 gm. of streptomycin in 100 cc. normal saline were instilled into the wound.

A small wound was then made in the most dependent portion of the wound coming out near the angle of the scapula. A large Penrose drain was drawn out through this stab wound to allow drainage of the wound of the chest wall. The subcutaneous tissue was then closed with interrupted #0 chromic gut, inverting the knots. The skin was closed with interrupted vertical sutures of black silk.

Attention was next turned to the wound of entrance. It was excised with an elliptical incision. It was found that the latissimus

Parkland Memorial Hospital

dorsi muscle, although lacerated, was not badly damaged so that the opening was closed with sutures of #0 chromic gut in the fascia of the muscle. Before closing this incision, the Penrose drain could be felt, by palpation with the index finger, immediately below in the space beneath the latissimus dorsi muscle. The skin was closed with interrupted vertical mattress sutures of black silk. Drainage tubes were secured with safety pins and adhesive tape, and dressings were applied.

As soon as the operation on the chest had been concluded, Drs. Gregory and Shires started the surgery that was necessary for the wounds of the right wrist and left thigh.

Charles F. Gregory.—Dr. Gregory is an orthopedic surgeon certified in 1953 by the American Board of Orthopaedic Surgery. He is 44, professor and chairman of orthopedic surgery at Southwestern Medical School, and a graduate of the Indiana University School of Medicine, Indianapolis. He is a veteran of both World War II and the Korean War.

There was comminuted fracture of the Governor's right distal radius which occurred when the bullet passed through the chest and struck the arm. Dr. Gregory debrided the wound and reduced the fracture.

This took place while the patient was still under general anesthesia following a thoracotomy and repair of the chest injury which was done by Dr. Shaw.

The right upper extremity was thoroughly shaved and prepped in the routine fashion. The patient was draped in the routine fashion using stockinette. An addition was the use of a debridement pan.

The wound of entry on the dorsal aspect of the distal right forearm at the junction of the distal fourth of the radius and its shaft was approximately 2 cm. in length and rather oblique, with a loss of tissue and with considerable contusion at its margins. There was a wound of exit along the volar surface of the wrist about 2 cm. above the flexion crease of the wrist in the midline.

The wound of entrance was carefully ex-

JOHN CONNALLY
GOVERNOR OF TEXAS

January 8, 1964

Dear Mrs. Baker:

When a person literally owes his life to a doctor or a team of doctors, it would seemingly be easy to express his thanks. This must not be the case, however, for it is extremely difficult for me to express in words my appreciation to the medical profession for the treatment I have received at Parkland and since my return to Austin.

The severity of my wounds probably would have resulted in death had there been any delay in medical treatment. Fortunately, a hospital such as Parkland Memorial was available, staffed with personnel trained to react swiftly to any circumstances. I was saved because men and women of great skill and experience were on hand to perform their duty.

Medicine is a profession with lofty traditions. As long as there are doctors like those with whom I have been associated during these weeks of treatment and recovery, there is no doubt in my mind that American medicine will continue its great advances in service to the people.

Sincerely,

John Connally

Mrs. Marilyn Baker, Managing Editor
Texas State Journal of Medicine
1801 North Lamar Boulevard
Austin, Texas

cised and developed through the muscles and tendons from the radial side of that bone to the bone itself where the fracture was encountered. It was noted that the tendon of the abductor pollicis longus was transected. Only two small fragments of bone were removed; one approximately 1 cm. in length consisted of lateral cortex which lay free in the wound and had no soft tissue connections, and another much smaller fragment 3 mm. in length. Small bits of metal were encountered at various levels throughout the wound. Wherever they were identified and could be picked up, they were submitted to the Pathology Department. Throughout the wound there were noted fine bits of cloth like mohair. Dr. Gregory was told that the patient was wearing a mohair suit at the time of the injury thus accounting for the deposition of such organic material within the wound.

After as careful and complete a debridement of the volar wound as possible and the integrity of the flexor tendons and the median nerve on the volar side established, the wound of exit on the volar surface of the wrist was closed primarily with wire sutures. The wound of entrance on the radial side of the forearm was only partially closed, being left open for the purpose of drainage. This was in deference to the presence of mohair and organic material deep in the wound.

In view of the urgency of the Governor's original chest injury, it was impossible to definitely ascertain the status of the circulation or the nerve supply to the hand and wrist on the right side on admission. Accordingly, it was determined as best it could be at the time of the operation. The radial artery was found to be intact and pulsating normally. The integrity of the median nerve and the ulnar nerve over a long distance was not established by Dr. Gregory. These would be repaired at a later date if necessary. Following closure of the volar wound and partial closure of the radial wound, dry sterile dressings were applied and a long arm cast was then applied with skin traction, rubber band variety, attached to the thumb and index finger of the right hand. An attitude of flexion was arranged at the right elbow, and postoperatively the limb was suspended from an overhead frame.

George Thomas Shires.—Dr. Shires, 38, is professor and chairman of the Department of Surgery at Southwestern Medical School. He is a 1948 graduate of Southwestern, a former Navy officer, and was certified by the American Board of Surgery in 1956.

Dr. Shires performed the surgery for exploration and debridement of the gunshot wound of the Governor's left thigh. The operation lasted 20 minutes. He reported that there was a 1 cm. punctate missile wound over the juncture of the middle and lower third, medial aspect, of the left thigh. X-rays of the thigh and leg revealed a bullet fragment which was imbedded in the body of the femur in the distal third. The leg was prepared with Phisohex and iodine and was draped in the usual fashion.

Following this, the missile wound was excised and the bullet tract was explored. The missile wound was seen to course through the subcutaneous fat and into the vastus medialis. The necrotic fat and muscle were debrided down to the region of the femur. The direction of the missile wound was judged not to be in the course of the femoral vessel, since the wound was distal and anterior to Hunter's canal. Following complete debridement of the wound and irrigation with saline, the wound was felt to be adequately debrided enough so that three simple through-and-through, stainless steel alloy #28 wire sutures were used encompassing skin, subcutaneous tissue, and muscle fascia on both sides. Following this, a sterile dressing was applied. The dorsalis pedes and posterior tibial pulses in both legs were good. The thoracic procedure had been completed at this time, the debridement of the compound fracture in the arm was still in progress at the time this soft tissue injury repair was completed.

The Governor's postoperative convalescence has been good, according to Dr. Shires. On the sixth postoperative day, the cast on his right arm was windowed and a routine delayed primary closure, with through-and-through stainless steel alloy wire, was performed. On the 14th postoperative day, the arm was x-rayed and a new cast was applied. On the 19th postoperative day, after ambulation, the Governor developed a superficial saphenous thrombophlebitis in the right ankle. This was the site of a sterile cutdown done in the operating room at the time of the initial injury for the administration of blood, fluids, and antibiotics. For this he was placed on a regimen of bed rest, elevation, heat, and heparin for a period of two weeks.

A. H. Giesecke, Jr.—Dr. Giesecke is an as-

sociate anesthesiologist in the Department of Anesthesiology at Parkland and an assistant professor at the University of Texas Southwestern Medical School. He is 31 years old and a 1957 graduate of the University of Texas Medical Branch.

Dr. Giesecke had learned from Dr. Jenkins that the President had been shot. He went to Trauma Room 1 to assist with the President, but was soon called to attend Governor Connally in Operating Room No. 5 in the main operating suite on the second floor where the Governor had been taken. On the way, Dr. Hunt briefed him, saying that she had examined the Governor and had found his color to be ashen, his pulse was of normal rate and volume, but he was dyspneic and tachypneic, grunting as he exhaled. She recalled having passed a cuff link to Mrs. Connally while the Governor was having a chest tube placed.

Upon arrival in the operating room, Dr. Giesecke was brought an anesthesia machine which he hurriedly checked for safe operation. He then introduced himself to the Governor, determined that he had not eaten since early morning, had not had any serious medical illnesses, and had not been in shock. At this time, he had 150 ml. of blood in the chest bottle, his color was as Dr. Hunt had described, his nail beds were cyanotic, his pulse was 100 and full, and he was alert and had not been premedicated.

Dr. Giesecke checked his mouth for foreign bodies and started 10 liters per minute oxygen by mask from the anesthesia machine. At this time he was having a cutdown performed in his right ankle and his trunk shaved from the clavicles down, including the right axilla. A Foley catheter was being placed in his bladder and 200 cc. urine was recorded.

Because of his poor color, respiratory distress, and probable large blood loss, Dr. Giesecke decided to omit thiopental and to use cyclopropane and oxygen. Accordingly, he asked for quiet and for the Governor to be covered with a clean cotton blanket. At 1 p.m., 20 minutes after his arrival in the emergency room, Dr. Giesecke started slowly with 800 cc. cyclopropane per minute plus 2 liters of oxygen per minute. His color had improved but his respirations were still rapid at 40 with grunting exhalations. The Governor lost consciousness without excitement at 1:07 p.m. and was given 80 mg. succinylcholine chloride very slowly intravenously to prevent hard fasciculations and passive regurgitation. Laryngoscopy was atraumatic and easy and no abnormalities were noted. The pharynx and trachea were sprayed with 4 per cent cocaine and intubated with a 34 Fr. endotracheal tube with a Knight-Grimm-Sanders cuff which was inflated to provide a good fit.

During the induction, Dr. Hunt connected the leads to the electrocardiograph monitor. Dr. Hunt reported a very transient bradycardia during the intubation. The pulse rapidly returned to 100 and the electrocardiogram looked normal. A blood pressure cuff and stethoscope was applied to the left arm and blood pressure was noted at 100/70. The explosion-proof x-ray machine was moved in and x-rays taken of the chest, right arm, and left thigh and leg. Blood was drawn for typing and crossmatching, and the hemoglobin was reported at 15.2 gm. per 100 cc.; urine was normal. Respirations were controlled, the position of the endotracheal tube was checked by auscultation of the chest and reference to the x-rays. The Governor was placed in a semi-lateral position with the wounded side up. The right arm, by a sling over the chest, was supported from the operating table. The skin incision was made at 1:35, 55 minutes after the Governor arrived in the emergency room.

Dr. Shaw, Dr. James Boland, resident in thoracic surgery, and Dr. James Duke, resident in surgery, operated for 1 hour and 45 minutes. The patient's position was changed to supine, and Drs. Gregory and William Osborne operated on the arm and Drs. Shires, Baxter, and McClelland operated on the left thigh simultaneously.

The cyclopropane was turned off at 4:45 p.m. and 50 mg. meperidine was given intravenously. The Governor regained consciousness during the application of the cast to the right arm and forearm. The endotracheal tube was irrigated with 50 ml. normal saline in 10 ml. increments, followed by suctioning, which yielded moderate amounts of bloody mucus. The oropharynx was cleaned. The estimated blood loss at surgery was 1296 cc. in the chest bottle, suction bottle, and weighed sponges. Urine output was 450 cc. He received 3 liters of lactated Ringer's solution, 2 liters of which contained 5 per cent dextrose; 2000 ml. whole blood; and 125 ml. 5 per cent dextrose in water. His color was pink, pulse 110, blood pressure 120/70, and his extremities were warm and dry. He was

awake, could open his eyes, and nod his head on command; therefore he was extubated.

Total anesthetic time was 3 hours and 50 minutes; total operating time was 3 hours and 15 minutes.

Upon extubation, Governor Connally spoke immediately, saying he felt well, but he was somewhat restless.

The immediate postoperative course was satisfactory, without hypotension, and with only a hint of cyanosis, which resolved over the following three to four hours, during which time he complained of soreness of his right shoulder and a sensation of needing to urinate, caused by the urethral catheter.

During surgery he received 1,000,000 units of penicillin after it was determined that he was not sensitive following a discussion with his wife and a call to Dr. W. B. Swift of Fort Worth. In addition, he received 500 mg. tetracycline. He had received 0.5 cc. tetanus toxoid in the emergency room prior to transfer to the main operating suite.

Lee Harvey Oswald

On Nov. 24, two days after the shooting of President Kennedy and Governor Connally, an ironic event brought to Parkland the man whom Dallas police had charged with the murder of the President. Lee Harvey Oswald, 24, had been shot.

Initial care and surgery were handled by Parkland physicians, including some of those who had cared for the President and the Governor. Oswald was brought into the emergency room at 11:32 a.m., to the operating room at 11:42 a.m. and at 1:07 p.m. he was pronounced dead in spite of all efforts.

Dr. Shires.—The surgery performed on Oswald, who had been shot in the upper abdomen and chest, was done by Drs. Shires, Perry, McClelland, and Jones, and included an exploratory laparotomy, thoracotomy, and efforts to repair the aorta, vena cava, and multiple organ injuries. Dr. Shires has said that on previous inspection an entrance wound over the left lower lateral chest edge was revealed and an exit was identified by subcutaneous palpation of the bullet over the right lower lateral chest cage. At the time he was seen preoperatively, Oswald was without perceptible blood pressure, his heart beat was heard intermittently at 130 beats per minute, he had an endotracheal tube in place and was receiving only oxygen by anesthesia at the time he was moved to the operating room.

Under endotracheal oxygen anesthesia, a long midline abdominal incision was made. Bleeders were not apparent and none were clamped or tied. Upon opening the peritoneal cavity, approximately 2 to 3 liters of blood, both liquid and in clots, was encountered. This was removed. The bullet pathway was then identified as having shattered the upper medial surface of the spleen, then entered the retroperitoneal area where there was a large retroperitoneal hematoma in the area of the pancreas. Following this, bleeding was seen to be coming from the right side, and upon inspection, there was seen to be an exit to the right through the inferior vena cava, thence through the superior pole of the right kidney, the lower portion of the right lobe of the liver, and into the right lateral body wall.

First the right kidney, which was bleeding, was identified, dissected free, retracted medially, and the inferior vena cava hole was clamped with a partial occlusion clamp of the Satinsky type. Following this immobilization, packing controlled the bleeding from the right kidney.

Attention was then turned to the left, as bleeding was massive from the left side. The inspection of the retroperitoneal area revealed a huge hematoma in the midline. The stomach had been penetrated by the bullet. The spleen was then mobilized, as was the left colon, and the retroperitoneal approach was made to the midline structures. The pancreas appeared to be shattered in its midportion; bleeding was seen to be coming from the aorta. This was dissected free. Bleeding was controlled with finger pressure by Dr. Perry. Upon identification of this injury, it was seen that the superior mesenteric artery had been sheared off of the aorta; there was some back bleeding from the artery. This was cross-clamped with a small, curved DeBakey clamp. The aorta was then occluded with a straight DeBakey clamp above and a Potts clamp below. At this point all major bleeding was controlled, blood pressure was reported to be in the neighborhood of 100 systolic. Shortly thereafter, however, the pulse rate, which had been in the 80 to 90 range, was found to be 40, and a few seconds later found to be zero. No pulse was felt in the aorta at this time. Consequently, the left chest was opened by Dr. Perry through an intercostal incision in approximately the fourth intercostal space. A Finochietto retractor was inserted, and the heart was seen to be flabby and not beating at all. There

was no hemopericardium. There was a hole in the diaphragm but no hemothorax. A left closed suction chest tube had been introduced while the patient was in the emergency room prior to surgery, so that there was no significant pneumothorax on the left side. The pericardium was opened, cardiac massage was started, and a pulse was obtainable with massage. The heart was flabby, consequently calcium chloride followed by 1 mg. epinephrine hydrochloride and 90 mg. Xylocaine Hydrochloride were injected into the left ventricle. The standstill converted to fibrillation. Following this defibrillation was done, using 240, 360, 500, and 750 volts, and finally successful defibrillation was accomplished after a second attempt with the defibrillator. However, no effective heart beat could be instituted. A pacemaker was inserted into the wall of the right ventricle and grounded on skin, and pacemaking was started. A very feeble, small, localized, muscular response was obtained with the pacemaker, but still no effective heart beat. At this time Dr. Shires was informed by Dr. Jenkins that there were no signs of life in that the pupils were fixed and dilated, there was no retinal blood flow, no respiratory effort, and no effective pulse could be maintained even with cardiac massage. The patient was pronounced dead at 1:07 p.m.

Anesthesia consisted entirely of oxygen. The patient was never conscious from the time of his arrival in the emergency room until his death at 1:07 p.m. The subcutaneous bullet was extracted from the side during the attempts at defibrillation which were rotated among the surgeons. The cardiac massage and defibrillation attempts were carried out by Drs. McClelland, Perry, and Jones. Assistance was obtained from the cardiologist, Dr. Bashour.

Dr. Jenkins.—In a statement concerning resuscitative efforts for Oswald, Dr. Jenkins reported that Dr. Jones, after being notified through the office of the administrator of the hospital, informed a surgical and anesthesiology team that Lee Harvey Oswald had sustained a gunshot wound and was being brought to the emergency operating room for emergency and definitive treatment. By the time that the patient, Oswald, was registered into the emergency operating room, there was assembled a resuscitative team in the emergency operating room surgical room.

Dr. Jenkins recalls that the following physicians were members of the resuscitation team: Drs. Jenkins and Akin, with an anesthesia machine and full resuscitative equipment for the maintenance of ventilation; Drs. Gerry Gustafson, Dale Coln, and Charles Crenshaw, all residents in surgery, who were prepared to introduce cannulae into the veins via cutdowns or percutaneous puncture; Dr. Jones with chest drainage equipment; Dr. William R. Osborne, resident in orthopedics, for necessary orthopedic services; and Dr. William Risk, resident in urology, for evaluation of possible urological damage. Dr. Perry was present to direct the surgical approach. There were many other medical personnel present in addition to these, but the physicians named figured importantly in the initial resuscitative experience, Dr. Jenkins said.

As the patient, Oswald, was brought into the operating room, Dr. Akin introduced a #36 cuffed endotracheal tube and connected it to an anesthesia machine for assisted ventilation or controlled respiration with oxygen. It was obvious that the patient was in extremis as judged by his general pallor, the cold extremities, the dusky or ashen gray color of his nail beds, his gasping respiration, and his dilated pupils and dry conjunctiva. There was a small, oval, traumatized area in approximately his left anterior axillary line at approximately the sixth intercostal space, and a foreign object, thought to be a bullet, could be palpated in his right posterior axillary line at about thoracic dermatome ten.

No time was expended in making these observations and evaluation of the patient's status, for at the time the endotracheal tube was being inserted, three members of the staff were performing venous cutdowns, one in each lower extremity and one in the left forearm. These were performed by Drs. Coln, Crenshaw, and Gustafson. Because of the obvious chest wound and appearance of pneumothorax on the left, Dr. Jones inserted a chest tube and connected it to a closed waterseal drainage bottle. The head of the emergency room cart was lowered into a Trendelenburg position. There was no perceptible peripheral arterial pulsation. However, the cardioscope tracing showed electrical cardiac activity with a heart rate of approximately 130 per minute. Blood was sent to the blood bank for immediate typing and crossmatching, and two units of uncrossmatched type O, Rh negative blood was started by pressure infusion from plastic

blood containers.

It was obvious that this patient had sustained such an injury that he was continuing to lose blood internally very rapidly. Drs. Shires, McClelland, and Perry collaborated in the decision to move the patient immediately to the main operating suite for emergency laparotomy, since the suspected path of the bullet would seem to traverse the left leaf of the diaphragm, the aorta and inferior vena cava, and perhaps the right kidney and part of the liver. (Dr. Risk had inserted a Foley catheter into the urinary bladder, obtaining only a scant quantity of urine which was not blood tinged.)

With the anesthesia machine still connected to the patient, he was transported to the elevator and into the operating room which had already been prepared for emergency surgery. The abdominal incision was made at 11:44 a.m., 12 minutes from the time the patient was first admitted to the emergency operating room.

The operating team consisted of Drs. Shires, McClelland, Perry, and Jones. The anesthesia team consisted of Drs. Akin, Jenkins, and Dr. Harlan Pollock, resident on anesthesiology.

In describing the patient's condition and the parasurgical considerations, Dr. Jenkins says that by the time of the beginning of surgery, type-correct blood (A-1, Rh negative) was available and was administered under pressure through the three venous cutdowns. Dr. Curtis Spier, fellow in anesthesiology, cannulated a vein in the right forearm to aid in fluid replacement.

Under the influence of blood administration and pulmonary ventilation with 100 per cent oxygen, the patient's pulse rate slowed from 130-150 to 80 per minute, and by 12 o'clock he had a discernible peripheral blood pressure, recorded at about 60 systolic, and by 12:10 p.m. his blood pressure was 90/60 and his pulse rate remained regular at 80 per minute.

By 12:15 p.m. he had received 3000 ml. of blood and 800 ml. of 5 per cent dextrose in lactated Ringer's solution. Estimated and measured blood loss at this time was 4000 ml.

By 12:30 p.m. he had received 6000 ml. of blood and 1 gm. of calcium gluconate intravenously. His measured blood loss at this time was 5000 ml., and it was also obvious that an additional quantity was sequestered in his bowel lumen and bowel wall.

At this time the surgical and anesthesia teams consulted about the patient's fluid status and decided that he needed a quantity of balanced salt solution; therefore, in two of the cutdown veins, 5 per cent dextrose in lactated Ringer's solution was begun. (Despite this rapid blood and fluid replacement, the patient's pulmonary status seemed satisfactory in that there was no perceptible change in compliance, as judged by the resistance to ventilation by compressing the reservoir breathing bag.)

At 12:37, Dr. Akin, who was monitoring the heart sounds with a chest stethoscope, reported that the cardiac tones were becoming weaker and the pulse rate was slowing from the previous rate of 80, to 60, to 40, to 30, and then became imperceptible. (These changes in rate were verified by a change in electrical activity as shown on the cardioscope.) Palpation of the heart through the diaphragm from the abdominal operating site was performed by Dr. Shires, who reported that he could not feel cardiac activity and he noted that the aorta had now ceased to pulsate. Dr. Perry opened the left chest with an incision at approximately the fourth intercostal space, extending from the sternum laterally to the left anterior axillary line. Under direct vision it was verified that rhythmic cardiac activity had ceased, the heart was dilated. Ten milliliters of 10 per cent calcium chloride were injected into the chamber of the left ventricle. The heart, which had been flaccid prior to this injection, showed an increase in muscular tone and was not dilated. One mg. of epinephrine hydrochloride in 90 mg. of 1 per cent lidocaine was injected into the left ventricular chamber, reducing the heart in overall size.

Ventricular fibrillation ensued. Manual cardiac systole (cardiac massage) was begun by Dr. Perry while the internal defibrillation apparatus was readied. Four attempts at ventricular defibrillation were made, with Dr. McClelland applying the defibrillation paddles to the heart, utilizing successively voltages of 240, 360, 500, and 750 without successfully effecting defibrillation. Between the applications of the defibrillation paddles, manual cardiac systole was continued alternately by Drs. Perry and McClelland.

At 12:55 p.m., the internal pacemaker, provided by Dr. Bashour, was attached to the heart, but the electrical stimulus provided by this pacemaker was not effective in producing visible cardiac systole. Two other attempts at internal defibrillation were made. The second defibrillating current produced asystole, but the internal pacemaker still did not stimulate effective cardiac activity.

Manual cardiac systole was re-started, causing palpable carotid pulse, but the patient's obvious external appearance was that circulation was ineffective as judged by the development of an ashen gray cyanosis. With an ophthalmoscope, Dr. Jenkins had periodically checked the retina for circulation during the resuscitative processes, and the retina could be visualized until 1:05 p.m., when it was apparent that the lens had become opaque, and retinal circulation was not observed. The patient was pronounced dead at 1:07 p.m. The bullet which was palpable in the right posterior axillary line was removed and sent out by the operating room supervisor, Miss Audrey Bell, to be turned over to the legal authorities.

As a summary of fluid replacement, this patient received 15 and ½ units of blood and 4200 ml. of 5 per cent dextrose in lactated Ringer's solution.

It is Dr. Jenkins personal feeling that all methods of resuscitation were instituted expeditiously and efficiently. Having observed this patient from the time he was wheeled into the emergency operating room, Dr. Jenkins felt that Oswald sustained a period of cerebral hypoxia or anoxia for the period of time lapsing between the gunshot wound which he received and the time that effective ventilation with oxygen was started in the emergency operating room. Considering the cerebral changes which would begin at the time of initial anoxia, notably cerebral edema, Dr. Jenkins felt that many vital centers, including the cardiovascular center, were irreparably damaged, despite all resuscitative measures, introducing the final cardiac asystole. The trauma which patient Oswald had sustained was too great for resuscitation.

Medical History

Gunshot Wounds of Four Presidents

As long as man tries to understand his environment, historians will probably compare one great event to similar preceding ones. Physicians, too, will evaluate significant medical events and try to put them in perspective.

Up until Nov. 22, 1963, physicians who studied gunshot wounds pondered over the aspects of medical care given to three assassinated Presidents of the United States. Now, the type of injury and the treatment of a fourth President has been entered into journals for comparison. In addition, the treatment of Governor John Connally of Texas may be studied and compared in the same books because of the event that linked him with President Kennedy.

Dr. S. B. Harper, reporting in the *Proceedings of the Staff Meetings of the Mayo Clinic* in January, 1944, described the injuries and treatment of Presidents Lincoln, Garfield, and McKinley.

Lincoln

The fatal bullet which struck Abraham Lincoln entered the occipital bone at the level of the transverse sinus about an inch to the left of the midline. "The path of the bullet," reports Dr. Harper, "was then obliquely forward across the brain, ending in the right frontal lobe. Extensive comminuted fractures of both orbital plates occurred, these apparently were the result of contrecoup since the dura over the frontal lobe was

not penetrated. Reconstruction of the path of the bullet on anatomical charts brings it across the upper surface of the pons and through the aqueduct of Sylvius."

President Lincoln lived about nine hours after the fatal shooting, the author comments, but adds that in view of the central location of the wound near vital centers, "it is surprising that the wound was not instantly fatal."

Signs noted before death were a deep coma, pulse rate of 48 beats per minute, unequal pupils finally becoming widely dilated, bilateral jacksonian convulsions and shallow respirations becoming Cheyne-Stokes in character. Also marked exophthalmos with extensive ecchymosis of the sclera and periorbital tissue were associated with the fractures of the orbital plates.

"With such an extensive wound across the base of the brain, it seems doubtful if the outcome would have been altered by any measures which are available today," Dr. Harper recorded in 1944.

Garfield

The bullet which led to the death of President James A. Garfield in 1881 entered the tenth intercostal space about three inches to the right of the midline and fractured the 11th rib.

"Because of its low velocity," Dr. Harper reports, "the lead ball was then deflected down and medially across the body of the first lumbar vertebra; it passed completely through the body but missed the spinal canal. It then buried itself under the peritoneum near the spleen after grazing the edge of the splenic artery."

President Garfield died two and a half months later, reportedly from sepsis occurring along the course of the bullet wound and rupture of an aneurysm of the splenic artery which developed at the site where the bullet grazed the vessel.

"Few cases in medical history have brought forth so much discussion and comment as did this one," notes Dr. Harper. "As was customary at that time, attention was centered on determining the location of the bullet. The roentgen rays were yet to come and reliance had to be placed on reconstruction of the relative positions of the President and his assassin at the time of the shooting, together with probing of the tract. Reconstruction of the scene and the shooting of bullets into cadavers was done in an attempt to reproduce the wound. Alexander Graham Bell was requested to conduct electrical experiments to determine the location of the bullet. . . . Everyone, including the general public, became concerned over the location of the bullet although it was apparent to a few of the more experienced military surgeons of the time that the presence of a bullet in the body was entirely compatible with life."

The physicians, Dr. Harper reports, were under tremendous criticism and pressure from physicians and individuals all over the world.

About a month after the shooting several subcutaneous abscesses appeared around the wound of entry. Twice the sinus tract was incised to promote better drainage, and later severe suppurative parotitis developed requiring incision and drainage.

The President was moved from the heat of Washington to the New Jersey seashore after which he became weaker and died from sudden hemorrhage from the aneurysm of the splenic artery.

Dr. Harper reports that "several things appear to have been done, which are now avoided if possible. The wound was explored digitally by unprotected fingers three times in the first twenty-four hours and then repeatedly during a collective examination of the patient by a large group of prominent doctors. Probes which were not sterile were used repeatedly during the first five days in an attempt to follow the course of the bullet. It seems likely that this procedure was associated with the production of the subhepatic abscess and the abscess in the right iliac fossa found at necropsy. It was recorded that a catheter could be passed into the abscess cavity in the right iliac region. The fractured vertebra was not suspected during life since apparently there was no displacement. It probably is fortunate that the probes were not able to follow the path of the bullet through the vertebra. Penetration of the spinal cord or perforation of the already damaged splenic artery might have been the result."

Speculating on what modern techniques might have accomplished, Dr. Harper mentioned the use of chemotherapy, better drainage for the subhepatic and iliac abscesses, blood transfusions and intravenous administration of fluids.

Shot twice on Sept. 6, 1901, President William McKinley received one superficial skin wound over the upper part of the sternum. Another bullet entered the left upper quadrant of the abdomen, perforating both walls of the stomach at the greater curvature and then, after grazing the upper pole of the left kidney, apparently buried itself in the deep muscles of the back.

Taken to a small emergency hospital on the grounds of the Pan-American Exposition at Buffalo, the President received an exploratory laparotomy within 90 minutes of the shooting.

"Light for the operation was provided in part by reflecting the rays of the setting sun into the wound with a hand mirror," Dr. Harper reports. "The instruments were those carried in a small pocket case by a local physician. Drop ether anesthesia was employed. The President was a large man and exposure of the abdominal viscera under the conditions of the operation must have been difficult. The perforations on both walls of the stomach were closed with a double row of silk sutures. Fortunately, the shooting occurred several hours after the President had eaten and there was little contamination of the peritoneal cavity from gastric contents. The path of the bullet into the retroperitoneal tissue was found, but, because of the shock attending the operation, the abdomen was closed without further exploration. No drainage was employed."

Because the first successful operation for a gunshot wound of the stomach had been performed only about 15 years before and it was customary to treat such wounds conservatively without laparotomy the doctor's decision to operate was "remarkable" notes Dr. Harper.

"During the first week following the operation, the President made satisfactory progress. He apparently recovered from the initial shock of the operation and was beginning to take a little solid food in addition to small amounts of liquids. Fluid balance had been maintained as much as possible by nutrient enemas and on several occasions the injection of small amounts of physiologic salt solution under the skin. Urinary outputs of 270 cc. for the second day and 420 cc. for the third day were recorded. The urine was concentrated (specific gravity of 1.026) and it seems likely that dehydration was present. Frequent irritating enemas and castor oil were given to bring 'elimination.' These did not aid in maintaining a positive fluid balance."

At noon on the seventh day after the operation, Dr. Harper reports, something happened and within 36 hours, the President was dead. No anatomic explanation for his sudden death was found at necropsy. It was officially attributed to the "presence of a localized region of necrosis near the pancreas in the retroperitoneal tissue along the track of the bullet."

Reports Dr. Harper, "There was no general peritonitis. The sutured holes in the stomach were still closed and the slight wound to the upper pole of the left kidney was healing. Although no embolus was reported to have been found on examination of the lungs, the sequence of the events suggests a pulmonary embolism. This seems to fit in with the meager available reports of the clinical findings during the last thirty-six hours. One other possible explanation would be the occurrence of a dehydration type of shock. The frequent purgings and enemas, the continued fever and the small oral intake of fluids all promoted dehydration. At the time it was suggested that the bullet had been poisoned with curare or smeared with virulent bacteria. These suggestions were officially refuted."

Dr. Harper noted that speculating on the use of modern therapeutic measures is difficult in President McKinley's case since the cause of death is unknown. He mentions, however, the use of anticoagulant drugs if there had been pulmonary embolism and intravenous fluids if there had been dehydration shock.

"While some surgeons might employ a drain into the lesser omental cavity, it was apparent from the report of the necropsy that drainage would not have altered the outcome. Dr. Matthew Mann, a prominent Buffalo surgeon who performed the operation on the President, stated that his reason for not draining the abdomen was that there was nothing to drain. This is still sound reasoning. It is doubtful whether failure to remove the bullet had anything to do with the President's death. Although necropsy was continued for four hours, the bullet was never found. This would indicate the further search at the time of operation was not justified."

Dr. Harper concludes that in the case of each of the wounded men the treatment ad-

ministered appears to have been entirely consistent with the best medical opinions of the time.

"While physicians now may not agree with some of the measures taken, it would seem that in each instance, the surgical judgment was sound and that it was lack of some of the modern therapeutic measures rather than faulty surgical judgment which made adequate treatment impossible."

He further concludes that Lincoln probably could not be saved today any more than he could have been in 1865, but Garfield and McKinley could have been helped by modern therapy.

Tarrant Heart Group Sets Symposium

The Tarrant County Heart Association will hold its 11th annual postgraduate symposium on heart disease on Sunday, Feb. 16, at the Fort Worth Academy of Medicine, 3855 Tulsa Way, Fort Worth.

Speakers and their topics include Dr. Thomas N. James, chairman, section on cardiovascular research, Henry Ford Hospital, Detroit, "Anatomy of the Coronary Arteries" and "Fact and Fiction about the Effects of Certain Cardiac Drugs"; Dr. Dwight C. McGoon, consultant, section of surgery, Mayo Foundation, Graduate School, University of Minnesota, Rochester, "Progress in Surgery for Congenital Heart Disease" and "Progress in Surgery for Acquired Heart Disease"; and Dr. S. Gilbert Blount, Jr., professor of medicine, head, Division of Cardiology, University of Colorado Medical Center, Denver, "Constrictive Pericarditis" and "Primary Myocardial Disease."

For additional information, physicians may write to Dr. Albert M. Goggans, chairman, symposium program committee, 1500 South Main St., Fort Worth.

Need for Loan Program Continues, Smith Says

Congress recently authorized a government loan program for students of medicine, osteopathy, and dentistry. The law provides for a total of $30,700,000 in loans over a three-year period. For as long as seven years (four years of school and three years after graduation), no interest is charged. When repayment of loans begins three years after graduation, the interest rate is 3 per cent.

Lyman J. Smith, director of the Medical Education Loan Guarantee Program, which is a part of the American Medical Association Education and Research Foundation, has recently commented on this law.

"A private loan program, such as AMA-ERF's, could not possibly set this low an interest rate. Banks, for example, must pay 3½ to 4 per cent on savings accounts to obtain money to lend. The government can do it because it uses tax funds to lend and to help defray operating expenses. The public, in short, picks up a large part of the bill," Smith said.

He lists three reasons, however, why the Medical Education Loan Guarantee Program will continue to be needed:

First, the government program will be concentrated on freshmen, sophomores, and juniors, while six of every ten AMA-ERF loans go to seniors, interns, and residents.

Second, because of their artificially low interest rate, demand for government loans may be expected to exhaust the supply of funds, and an appreciable number of students may need to apply elsewhere.

Third, there is a need for a program, such as AMA-ERF's, for students who prefer private financing arrangements and do not wish to feel indebted in any way to the government.

Austin Home Seeks Adoptive Negro Parents

Workers at an Austin shelter for unwed mothers, Home of the Holy Infancy, are interested in locating adoptive parents for Negro babies.

There has been a recent increase in the number of pregnant Negro girls admitted to the home, and there have never been many applications from Negro parents for babies.

Physicians who know possible adoptive applicants may direct them to call or write Home of the Holy Infancy, 510 West 26th, Austin, Telephone: GR 2-8721.

The home also places Latin-American and white children. Adoptive parents are expected to be a couple with a happy marriage who have sound reasons for wanting a child and the ability to give love to the child. They should have average physical and mental health, sufficient income to rear the child, age range which would ordinarily correspond

to that of the natural parents, and adherence to religious principles (Catholic or Protestant). Children are placed throughout the state provided the applicants are able to come to Austin for interview and for the legal completion of adoption.

IRS Issues Rules On Corporations

The Commissioner of Internal Revenue, as of Dec. 16, issued proposed regulations on professional corporations and associations. If adopted, these regulations would ruin chances of physicians, dentists, lawyers, and other professionals to gain tax advantages through incorporation.

Such regulations would specifically cancel out the benefits that would have been provided for by the Texas Professional Corporation bill, introduced in the Texas Legislature last year. This bill, which was not passed, was opposed by Texas Medical Association, acting on the recommendation of the Council on Medical Jurisprudence, and the Reference Committee on Legislation and Public Relations whose recommendations were adopted by the House of Delegates.

Hearings will be held on the proposals before they are adopted or rejected, but at a time as yet unscheduled; however, written comments or requests to be heard orally were required to be filed with the Commissioner by Jan. 16.

The Reference Committee stated in its report that it sympathized "with all of those who . . . [seek] more equitable tax treatment. However, the Committee wonders if seeking such corporate status might not be chasing the rainbow in view of the fact that there are some 200 cases of attempted qualification still pending without affirmative action by the Bureau of Internal Revenue."

The Council in its report stated opposition to the principle of corporate practice of medicine. "While this Council is cognizant of the advantages to be gained from a tax point of view, it is also aware of legal and ethical problems involved," it stated.

It was the belief of the Council that the best interest of the patient is served through the personal physician of the patient's choice —not through employees of corporations.

"The doctor said I needed more exercise . . . to get out and orbit the block three times in the evening."

Border Health Association To Meet in Monterrey

The annual meeting of the United States Mexico Border Public Health Association will be held in Monterrey, Mexico, March 2-5.

For additional information, interested persons may write to Ulpiano Blanco, secretary, United States Mexico Border Public Health Association, 501 U.S. Court House, El Paso.

Medicine, Surgery Conference Set by Scott & White Clinic

A conference in medicine and surgery will be held Feb. 16-18 at Scott & White Clinic in Temple. This is the 12th annual meeting of this conference which is cosponsored by the Division of Continuing Education of the University of Texas Graduate School of Biomedical Sciences at Houston.

Guest speakers will include Dr. Robert J. Coffey, professor of surgery, Georgetown University Medical School, Washington, D. C.; Dr. G. Gordon McHardy, professor of clinical medicine, Louisiana School of Medicine, New Orleans; and Dr. Walter H. Judd, Washington, D. C., former Congressman from Minnesota and medical missionary.

Additional information may be secured by writing to Dr. John F. McKenney, 2401 South 31st St., Temple.

Cost of Carelessness Can Be High

INFANTS OF PARENTS with incompatible Rh blood factors may suffer from erythroblastosis fetalis, a condition detectable by laboratory tests and recognition of clinical symptoms such as anemia, jaundice, enlargement of the spleen or liver, and difficulty with breathing.

A case was brought to trial in which the defendant physician had diagnosed the infant plaintiff's jaundice, recognized within a few days of birth, as physiologic. The evidence developed on the trial of the case established beyond question that the infant plaintiff had kernicterus.

The defendant doctor based his diagnosis on an incorrect laboratory test which reported that the infant's Rh factor was negative when in fact such factor was positive. The mother's hospital records and charts, available to the physician, clearly demonstrated that the mother had a positive Coombs' test, thus indicating her sensitivity to the Rh-positive fetus she was carrying. The trial court gave the plaintiff a substantial judgment and upon appeal of the matter to the higher courts, the decision of the trial court was affirmed.

The court's opinion is illustrative of the current judicial thinking with respect to making use of diagnostic procedures and staying abreast of current developments in such procedures.

> No van den Bergh's test, which would prove the presence of bile in the brain, was given and no follow-up hemoglobin test was ordered, in spite of the fact that the second blood count, made on November 6th (4 days after birth), showed a significant drop in the hemoglobin, admittedly diagnostic evidence of the presence of erythroblastosis fetalis.
>
> . . . The testimony of various doctors established that a child of an Rh-positive homozygous father and a sensitized Rh-negative mother would be Rh positive and, in all probability, would suffer from erythroblastosis fetalis at birth. This medical testimony also showed that where laboratory tests are inconsistent with clinical findings, the laboratory tests should be re-run, particularly, in the circumstances of this case, where a laboratory test, obviously wrong, showed the child as Rh negative. The medical evidence also showed that where a child of an Rh-positive homozygous father and an Rh-negative mother developed jaundice in the first few days of life, a van den Bergh's test was strongly indicated. This same evidence showed that good medical practice would require a follow-up hemoglobin test, in the circumstances of this case where the second hemoglobin test taken showed a falling blood count. The medical evidence also established that if the proper tests used in this area in 1953 had been run, and re-run where indicated, a diagnosis of pathologic, as distinguished from physiologic, jaundice would have been required. The recognized treatment for pathologic jaundice in this area was in 1953, and is now, an exchange transfusion in which the blood of the baby is replaced with healthy blood. While the incidence of mortality in such operations was, in 1953, ten per cent, the alternative is paralyzing brain damage and early death, and the recognized medical practice at that time, as now, was to undertake the exchange transfusion.

Aside from the obvious overtones of the necessity for physicians to keep up with what diagnostic procedures are available, it is likewise important to re-emphasize here the tremendous importance attached to laboratory tests. This in turn should re-emphasize to physicians the importance of knowing the laboratory to which their specimens and tissues are sent, and satisfy themselves that the supervision and competence are of the caliber desired. Cases such as that cited emphasize the tragedy of carelessness. The amount of the judgment emphasizes the economic disaster to the attending physician.

—PHILIP R. OVERTON, LL.B., Austin.

Hospital Accreditation Institute Announced

Originally set for Feb. 7-8, the accreditation institute to be conducted by Texas Hospital Association and sponsored by Texas Medical Association and Private Clinics and Hospitals Association will be held Feb. 15 at the Commodore Perry Hotel in Austin.

The institute is open to hospital administrators, medical staff members, nurses, medical record librarians, and hospital trustees. Those interested in attending should apply to Texas Hospital Association, 1905 N. Lamar Blvd., Austin. Room reservations should be made with the hotel.

Physicians who are to speak at the institute include Drs. Leigh J. Crozier, Houston; A. W. Bronwell, Lubbock; Kenneth Babcock, Chicago; H. F. Rives, Jacksonville; and G. V. Brindley, Jr., Temple.

Personals

New officers of the Houston Radiological Society elected in November are *Dr. Roland B. Carroll,* president; *Dr. Harry Barton,* vice president; *Dr. D. A. Van Velzer,* secretary; and *Dr. Paul Chau,* treasurer.

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Dr. Elizabeth Gentry of Austin is the new president of the Texas Society on Aging.

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New officers of the American Medical Women's Association, Inc., elected for 1964 include two Texans, *Dr. Mary E. Sears* of Houston, and *Dr. Mary Mitchell Henry* of San Antonio.

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New officers of the Eighth District Medical Society are *Dr. Harry K. Davis,* Galveston, president; *Dr. Weldon G. Kolb,* LaMarque, vice president; and *Dr. Stanley Thompson,* Richmond, secretary-treasurer.

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Dr. J. M. Brown of Marlin presented a scientific program on radiation at the December meeting of the Falls County Medical Society.

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Dr. A. C. Surman of Post has announced his retirement effective Jan. 21. He has practiced more than 50 years.

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Dr. Clarence Smith Kemp of Bryan has been appointed by Gov. John Connally to the State Board of Medical Examiners.

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Dr. Robert P. Green of Texas City is one of two physicians in the United States named to the 16-man committee of the United States Chamber of Commerce to look into the problems of the aged.

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Dr. Dolph Curb of Houston is president-elect of the Texas Academy of Internal Medicine. New president is *Dr. Ralph Greenlee* of Midland and the new vice president is *Dr. Ben Wilson* of Tyler. *Dr. Warren W. Moorman* of Fort Worth is secretary.

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Dr. Robert D. Moreton of Fort Worth is president-elect of the Radiological Society of North America.

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New officers of the Texas Association for Mental Health include two physicians: *Dr. Hamilton Ford* of Galveston, president; and *Dr. Robert Stubblefield* of Dallas, vice president.

Topics Announced for Jan. 30-Feb. 1 Meeting Of International Medical Assembly of Southwest Texas

SPEAKERS' TOPICS for the 28th annual meeting of the International Medical Assembly of Southwest Texas have been announced by the Assembly president, Dr. William M. Center of San Antonio. The meeting will take place at the Granada Hotel in San Antonio Jan. 30-Feb. 1.

Dermatologist Dr. A. B. Loveman, chief of section of dermatology and syphilology, University of Louisville School of Medicine, Louisville, Ky., will give three papers: "Cutaneous Disorders of the Female Genitalia," "Common Disorders of the Face and Scalp," and "Mucous Membrane Lesions of Interest to the General Practitioner."

Speakers from the field of internal medicine include Dr. Gordon McHardy, chairman, section of gastroenterology of the AMA, clinical professor of medicine, Louisiana State University School of Medicine, and past president, American Gastroenterological Association, of New Orleans; Dr. Jorge Espino-Vela, chief, Department of Pediatric Cardiology, Instituto Nacional de Cardiologia, Mexico, D.F.; Dr. William Parson, professor of medicine and chairman of the Department of Medicine, University of Virginia School of Medicine, Charlottesville, Va.; and Dr. Douglas G. Cameron, physician-in-chief, The Montreal General Hospital, professor of medicine, McGill University.

Dr. McHardy's topics are "Present Evaluation of Gastric Hypothermia," "Medical Management of Esophageal Hiatal Hernia," and "Hepatitis and Its Sequelae." Dr. Espino-Vela will speak on "The Maya Culture," "Operative Results of Mitral Commissurotomy in Children," and "On Some Varieties of Abnormal Coronary Arteries." Dr. Parson's topics are "Current Concepts of Obesity and Its Management," "Differential Diagnosis and Management of Hypercalcemic States,"

and "Metabolic Bone Disease with Particular Reference to Osteoporosis." Dr. Cameron's topics include "Prophylaxis and Treatment of Tetanus," "Bacteremic Shock—A Medical Emergency," and "Pyelonephritis—A Challenge—Diagnosis and Management."

Dr. John Parks, professor of obstetrics and gynecology and dean, George Washington University School of Medicine, Washington, D.C., will speak on "Womanhood after Fifty," "The Effects of Perinatal Emotions," and "Genetics in Obstetrics and Gynecologic Practice."

Orthopedic surgeon, Dr. Marcus Stewart of Campbell Clinic, Memphis, Tenn., has chosen to speak on "Treatment of Fractures of the Carpal Navicular with Review of Over

"Will you get my wife on the phone?"

500 Cases," "Athletic Injuries with Special Reference to the Knee," and "The Diagnosis and Treatment of Bursitis and Tendinitis of the Extremities."

Pathologist Dr. Edward A. Gall, director, Department of Pathology, Cincinnati General Hospital, Cincinnati, Ohio, has announced the following topics: "Disorders Related to Medical and Surgical Therapy," "System Vascular Disorders, Rare and Common," and "Fatal Hemorrhagic Enterocolitis."

Pediatric topics chosen by Dr. Richmond S. Paine, neurologist, Children's Hospital of the District of Columbia, professor of pediatric neurology, George Washington University School of Medicine, Washington, D.C., are "The Early Recognition and Management of Cerebral Palsies," "Phenylketonuria," and "How Much Diagnostic Evaluation is Required for the Child with Seizures?"

Psychiatrist Dr. Jack R. Ewalt, Bullard professor of psychiatry, Harvard Medical School, Boston, is to speak on "Somatic Equivalents of Depression," "Adolescent Problems; Medical or Social?", and "Trends in the Care of Mentally Ill Persons."

Ophthalmology topics include "The Diagnosis of Congenital Glaucoma," "Detection of Glaucoma by the General Practitioner," and "The Treatment of Congenital Glaucoma." They will be presented by Dr. Joseph S. Haas, associate professor of ophthalmology, University of Illinois College of Medicine, Chicago.

In radiology, topics are to include "Roentgen Manifestations of Acute Abdominal Disease," "Malabsorption Syndromes," and "Radiological Diagnosis of Gallbladder Disease." Speaker is Dr. Robert F. Feltman, associate director, Department of Radiology, Jackson Memorial Hospital, and assistant professor of radiology, University of Miami School of Medicine, Miami, Fla.

In the field of surgery, speakers will be Dr. George T. Pack, attending surgeon, Memorial Cancer Center, and attending surgeon, Pack Medical Group, New York, N.Y.; and Dr. Mark M. Ravitch, surgeon in chief, Baltimore City Hospitals, and associate professor in surgery, Johns Hopkins Hospital, Baltimore.

Dr. Pack will speak on "Present Day Management of Cancer of the Stomach," "The Diagnosis and Classification and Treatment of Sarcomas of the Soft Somatic Tissues," and "The Problem of the Pigmented Mole and Malignant Melanoma." Dr. Ravitch will speak on "Diverticulitis of the Colon," "Tumors of the Head and Neck in Infants and Children," and "Use of the Soviet Stapling Instruments in General and Thoracic Surgery."

In thoracic surgery, topics are "Observations of Mediastinal Granulomata," "Combined Injuries of Upper Air and Food Passages," and "Some Surgical Problems of the Esophagus in Infants and Children." Speaker is Dr. Thomas H. Burford, professor of thoracic and cardiovascular surgery, Washington University School of Medicine, St. Louis.

Urologist Dr. James F. Glenn, professor of urology and chief of Division of Urologic Surgery, Duke University Medical Center, Durham, N.C., will speak on the following topics: "Renovascular Hypertension," "Bladder Dysfunction and Vesicoureteral Reflux in Children," and "Experimental Experience with Mannitol."

The field of aerospace medicine will be discussed by Col. John P. Stapp, deputy chief scientist, Aerospace Medical Division, Brooks Air Force Base, Tex. His topics are "Auto-

mobile Crash Protection" and "Impact Injury and Survival Limits."

Emergency Department Poster Believed Effective

An educational program is believed responsible for a decrease in the number of patients treated at the emergency department of Spohn Hospital in Corpus Christi.

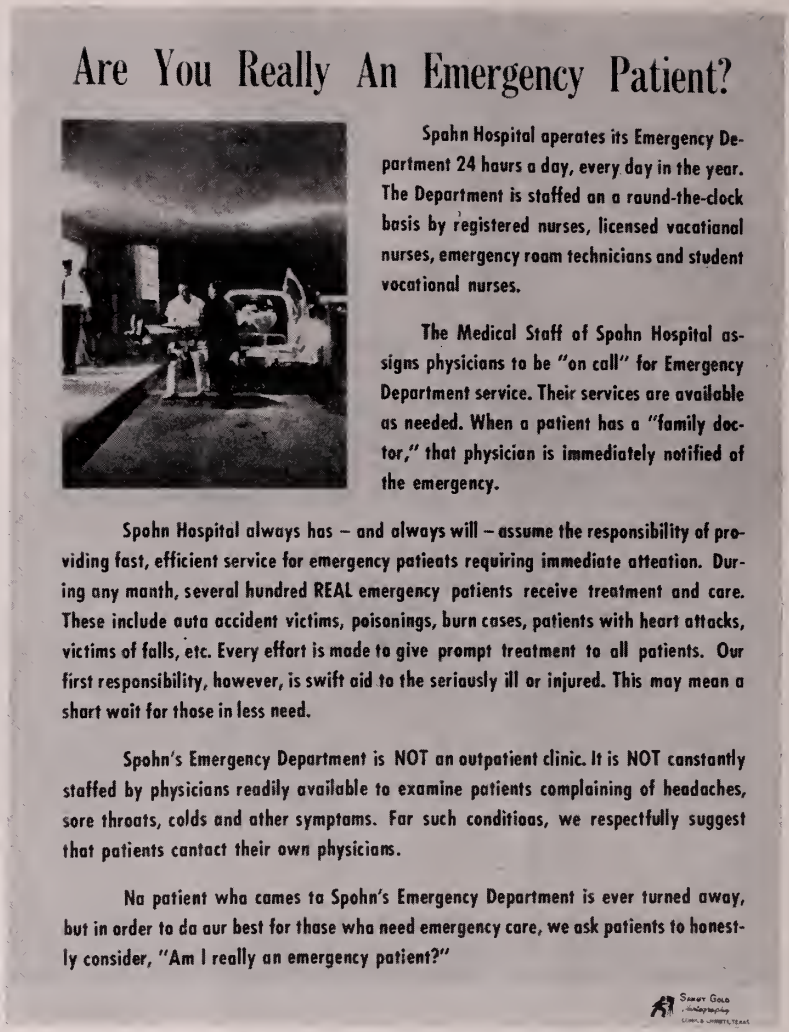
For some time, administrators of the hospital have been aware that many patients coming to the emergency department are not real emergency patients at all but people with such complaints as colds, sore throats, and headaches. Last August, the hospital decided to begin an educational campaign to tactfully discourage use of the emergency department by non-emergency patients in order that true emergency patients might receive the best possible care.

In October the number of medical patients treated in a one-month period had dropped from 237 in July to 190.

The educational efforts of the hospital were directed toward the general public by means of a poster entitled "Are You Really An Emergency Patient?" (see photograph) and displayed in the emergency department.

Members of the medical staff were informed of the educational program and the text of the poster by means of a letter sent to each.

Nurses were made aware of the program in an article carried in the hospital news bulletin, *Spohn-taneous.*


Are You Really An Emergency Patient?
Spohn Hospital operates its Emergency Department 24 hours a day, every day in the year. The Department is staffed on a round-the-clock basis by registered nurses, licensed vocational nurses, emergency room technicians and student vocational nurses.
The Medical Staff of Spohn Hospital assigns physicians to be "on call" for Emergency Department service. Their services are available as needed. When a patient has a "family doctor," that physician is immediately notified of the emergency.
Spohn Hospital always has – and always will – assume the responsibility of providing fast, efficient service for emergency patients requiring immediate attention. During any month, several hundred REAL emergency patients receive treatment and care. These include auto accident victims, poisonings, burn cases, patients with heart attacks, victims of falls, etc. Every effort is made to give prompt treatment to all patients. Our first responsibility, however, is swift aid to the seriously ill or injured. This may mean a short wait for those in less need.
Spohn's Emergency Department is NOT an outpatient clinic. It is NOT constantly staffed by physicians readily available to examine patients complaining of headaches, sore throats, colds and other symptoms. For such conditions, we respectfully suggest that patients contact their own physicians.
No patient who comes to Spohn's Emergency Department is ever turned away, but in order to do our best for those who need emergency care, we ask patients to honestly consider, "Am I really an emergency patient?"

A drop in the number of medical patients treated has followed the hanging of this poster in the emergency department of Spohn Hospital in Corpus Christi.

Postgraduate Courses

Pediatrics, Galveston, Feb. 13-15, 1964.—The Department of Pediatrics, University of Texas Medical Branch in conjunction with the Maternal Child Health Division, Texas State Department of Health, will sponsor the course.

Guest speakers will discuss the following topics: genetics, Dr. Melvin M. Grumbach, New York City; pediatric radiology, Dr. Edward B. D. Neuhauser, Boston; pediatric neurology, Dr. Richmond S. Paine, Washington, D. C.; fluids and metabolism, Dr. William B. Weil, Gainesville, Fla.

Members of the Texas Academy of General Practice may receive 15 hours credit rating. Tuition fee is $15. Information may be obtained by writing Dr. Myron M. Nichols, Department of Pediatrics, University of Texas Medical Branch, Galveston.

Dermal Pathology, Houston, Feb. 17-21, 1964.—The course will be presented by the Division of Continuing Education, University of Texas Graduate School of Biomedical Sciences in collaboration with the Skin and Cancer Hospital of Philadelphia, Temple University, where a similar course has been of-

fered for the last several years. The course is designed primarily for pathologists and dermatologists interested in cutaneous pathology and physicians preparing for specialty boards. Emphasis will be on reviewing the basic concepts of dermal pathology, including embryology, histology, electron microscopy, histochemistry, inflammatory dermatoses, granulomatous dermatoses, reticuloendothelial and alterative dermatoses, and nevi and neoplasms.

The class will be limited to 40 students. There will be a fee of $105. It is recommended that students bring their own microscopes if possible. Participants are encouraged to write to the American Registry of Dermal Pathology, Walter Reed Army Medical Center, 6825 16th Street, N.W., Washington, D.C., and request loan of the skin sets to be covered in this course. It is preferable to study this material prior to taking the course.

Members of the faculty will include Drs. Hamilton Montgomery, Rochester, Minn.; Elson B. Helwig, Washington, D.C.; Herman Pinkus, Detroit, Mich.; James H. Graham, Philadelphia; Waine Johnson, Philadelphia; David Kersting, Milwaukee, Wis.; Alvin Zelickson, Minneapolis, Minn.; Milton Boniuk, Houston; James J. Butler, Houston; J. Leslie Smith, Jr., Houston; and Jeffrey Chang, Ph.D., Houston.

Those interested in further information or advance registration should write to Division of Continuing Education, University of Texas, Graduate School of Biomedical Sciences at Houston, 102 Jesse Jones Library Building, Texas Medical Center, Houston.

Gynecological Pathology, Radiotherapy, and Endocrinology, Houston, March 19-22, 1964.—The tuition for this course sponsored by Baylor University College of Medicine is $110. The group will be limited to ten. Inquiries should be addressed to the Department of Obstetrics and Gynecology, Baylor University College of Medicine, Houston, 77025.

Allergy, Houston, Feb. 3-7, 1964.—The University of Texas Graduate School of Biomedical Sciences at Houston, Division of Continuing Education, will hold this course in the auditorium of the University of Texas M.D. Anderson Hospital and Tumor Institute in the Texas Medical Center. It will be given by Dr. James W. Willoughby of Kansas City, Mo.

The course will consist of lectures and demonstrations and will cover the clinical pattern of allergic reactions and syndromes, the nature and types of etiologic factors, the nature of diagnostic techniques, clinical application of fundamental phenomena of sensitization, and therapy.

Following the course in allergy, the Gulf Coast Allergy Study Group will hold its meeting on Saturday and Sunday, Feb. 8-9, 1964.

For further information, physicians may write to Division of Continuing Education, the University of Texas Graduate School of Biomedical Sciences at Houston, 102 Jesse Jones Library Building, Texas Medical Center, Houston.

Grayson Polio Funds Go to Health Projects

Grayson County Medical Society has voted to split surplus polio funds three ways: one-third to go to countywide health projects, one-third to Sherman projects, and one-third to Denison projects. The surplus amounted to $12,000.

The third for countywide projects has been earmarked for the mental health association, the county crippled children's center, and the newly established tumor register. Sherman's third will go to the crippled children's clinic. Denison is expected to use its money for medical education, primarily through the hospitals.

Texas Urological Society To Meet Feb. 8-9 in Austin

Guest speakers for the annual meeting of the Texas Urological Society will be Dr. James H. De Weerd of Mayo Clinic, Rochester, Minn., and Dr. John A. Hutch of the University of California Medical School, San Francisco.

The meeting will be held Feb. 8-9 at the Villa Capri in Austin.

Officers of the society are Dr. Newton F. McDonald, Houston, president; Dr. Roy Riddel, Lubbock, vice president; and Dr. B. Clary Bates, secretary.

Citizens' Committee Convenes For Mental Health Planning

SOMETHING entirely new in treating mental illness took form in Texas on a cold, foggy Dec. 14th when a citizens' committee for comprehensive state and community mental health planning met in Austin. The meeting, part of a dream and plan of action supported by the late President, was first scheduled for Nov. 23, but was postponed due to the national tragedy which occurred the day before.

One hundred and twenty-seven people attended the meeting at Texas Medical Association headquarters, and 80 members out of the 97 who have said they will serve on general planning committees were present. Many of the people will attend a second meeting scheduled for Feb. 7-8 at the Commodore Perry Hotel in Austin.

Leading up to the December meeting was a series of Congressional actions, culminating in the passing of the mental illness-retardation bill on Oct. 31. A total of $329,000,000 has been authorized for this program, including $150,000,000 in matching grants for the construction of community mental health centers. Such centers may one day reduce the population of large public mental institutions by as much as 50 per cent. As a result of earlier legislation, Texas has been allocated $182,200 which is being matched by public funds being spent within the state for community mental health services and planning.

The Texas Plan, as it is called, originated with a medical advisory committee in the summer of 1962 but was revised along lines recommended by five organizations concerned with mental health. In March, 1963, the Plan was turned over to a steering committee for organizational shaping. Believing that earlier efforts failed because citizen leadership was not involved in planning or implementation, leaders of the Texas Plan resolved to bring in many interested citizens early in the planning stage. This was realized with the Dec. 14th meeting in Austin.

A Charge

Dr. Spencer Bayles, psychiatrist-coordinator of the State Department of Health's Office of Mental Health Planning, outlined the task before the group.

"You're here because the State of Texas needs help to bring about changes you know are necessary—but you can't bring them about alone," he said.

"There must be changes in community structure to make all this possible. The State expects from you the solution, not just words, but action."

The steering committee has broken down this task into manageable jobs. Having identified the major problems, the committee decided on the abilities and qualifications of people to help solve them.

"Each of you is distinguished in his own field, but that is not why you were asked to participate," Dr. Bayles pointed out. "Each of you is here because of your knowledge of the general problem and your specific knowledge of mental health problems in your own area."

He knew of no way to avoid a large investment of time, Dr. Bayles said. It would be necessary to become familiar with the problem and with each other at this first meeting.

At a second meeting, planners will have more information and be better able to start defining certain outlines of their chapter in the statewide mental health plan, he said.

At a third meeting, participants should have something on paper—their agreements and disagreements.

"Each task force is relevant to and dependent upon the other task forces," he said. Reports of other task forces will be circulated among each member for appraisal.

At their fourth meeting, Dr. Bayles suggested that the task forces rewrite their plans with regard to the opinions of other forces.

When the chapters are revised, all the drafts will again be submitted for review and criticism.

Dr. Bayles emphasized that this work must be ready in January, 1965, when the Legislature convenes.

Task Forces

The 13 task forces met after lunch that day for the first time to consider a list of suggested central questions from the steering committee. The committees, questions before them, and members of the committees are as follows:

Legislative Committee.—What are strengths and limitations of our present legal structure relating to mental health services, and what legislative work will be required to implement the proposed statewide plan?

Mrs. Bess S. Clayton and Judge Robert H. Hughes of Dallas; Dr. John M. Smith, Jr., San Antonio; Judge William M. Elliott of Houston; and J. C. Davis, Jr., James A. Hankerson, Jr., Philip R. Overton, and Millard Ruud, all of Austin.

Public Education Committee.—By whom and through what channels can the public be educated?

Dr. E. Winston Cochran of Beaumont; Mrs. Hamilton Ford, LaMarque; William P. Hobby, Jr., Houston; Mrs. Harold E. Murphree, Jr., Dickinson; Alan Scott, Ed.D., Austin; and Mrs. George W. Stevens of Fort Worth.

Prevention Mental Illness Committee.—What should be done, by whom, and where to prevent mental illness and mental retardation, and to promote and sustain mental health on the basis of current knowledge?

Dr. Charles M. Gaitz of Houston; Carson McGuire, Ph.D., John Mayfield, Charles H. Meisgeier, Ed.D., and Bernice Milburn Moore, Ph.D., all of Austin; and Dr. Albert G. Randall of Amarillo.

Services to Troubled People Committee.—What services can be expected to protect and promote the mental health of troubled people and how can adequate services be made available to troubled people at the time of need?

Col. S. Perry Brown, Dr. Samuel A. Hoerster, Jr., Lora Lee Pederson, Victor W. Ravel, and Rev. Robert S. Tate, Jr., all of Austin; Mrs. Ruth Chester, Nederland; James F. Dinsmore, Beaumont; Carl Flaxman, Miss Selby Fly, and Bernard Goldstein, Dallas; Rabbi David Jacobson, San Antonio; Dr. W. R. Metzger, Corpus Christi; Truett A. Roach, Andrews; Dr. Frank P. Schuster, Jr., El Paso; Mrs. Fred Taliaferro, Mt. Pleasant; and Joseph Zarefsky, Houston.

Diagnosis and Treatment—Child Committee.—What services should be available for mentally ill, mentally retarded, and emotionally disturbed children; how much, where, and how provided?

Dr. Arr Nell Boelsche and Dr. Henry L. Burks of Galveston; Ira Iscoe, Ph.D., Austin; Mrs. Nancy Marcus, Dallas; Dr. Jaime Quintanilla, Amarillo; and Mrs. Ovid Youngblood of Waco.

Diagnosis and Treatment—Adult Committee.—What services should be available for mentally ill, mentally retarded, and emotionally disturbed adults; how much, where, and how provided?

Ted Bowen, Dr. William A. Cantrell, Dr. Alex D. Pokorny, and Dr. Hampton Robinson, all of Houston; Dr. Charles H. Brown, Wichita Falls; Dr. George A. Constant, Victoria; Harold A. Goolishian, Ph.D., Galveston; Mrs. John B. Luscombe, Jr., El Paso; Senator Walter Richter and Mrs. Chester Snyder of Austin; and Dr. Robert C. Rowell, Terrell.

Rehabilitation Committee.—What rehabilitation services should be available to the mentally ill and mentally restored and how can these services become increasingly a part of diagnostic and treatment services?

Claire G. Fairchild, Ted Mitchell, and W. L. Platt of Austin; Dr. Preston E. Harrison, Big Spring; and Clifford S. Knape, Ph.D., Waco.

Finance Committee (including insurance).—What is the current cost of all mental health services? What should we be spending? From what sources should the discrepancy be made up? What new patterns of methods of payment for services can be devised so that a patient's financial status or its changes no longer disrupt treatment programs?

Judge C. L. Abernethy, Plainview; Lee T. Bivins, Amarillo; Robert Hawthorne and John McKee of Dallas; George Markins, Houston; and Stephen J. Matthews, Austin.

Administration and Coordination Committee.—What is the best and most practicable modification of the structure and coordination of state agencies in order to improve and expand comprehensive mental health services?

(Members of this committee also make up the steering committee.)

Dr. Spencer Bayles, psychiatrist-coordinator, Office of Mental Health Planning; George Beto, Ph.D., director, Department of Corrections, Huntsville; J. W. Edgar, Ph.D., commissioner of education, Texas Education Agency; Dr. Hamilton F. Ford, chairman, Department of Psychiatry, University of Texas Medical Branch; Dr. S. H. Frazier, chairman, Department of Psychiatry, Baylor University College of Medicine;

Homer Garrison, Jr., director, Department of Public Safety; Dr. Abe Hauser, chairman, Steering Committee for Texas, AMA Congress on Mental Illness and Health; Frank Self, executive director, Texas Association for Mental Health; Dr. Robert L. Stubblefield, chairman, Department of Psychiatry, Southwestern Medical School;

Robert L. Sutherland, Ph.D., director, The Hogg Foundation for Mental Health; Dr. Perry C. Talkington, chairman, Mental Health Committee of Texas Medical Association; James A. Turman, Ph.D., executive director, Texas Youth Council; and John H. Winters, commissioner, State Department of Public Welfare. Ex-officio members include Bill Cobb, director, Governor's Budget Office, and Vernon McGee, director, Legislative Budget Board.

Epidemiology Committee.—What constitutes an adequate epidemiological program in the mental illnesses for Texas and how can this be developed?

Dr. W. V. Bradshaw, Fort Worth; Hiram Friedsam, Ph.D., Denton; Dr. Robert L. Leon and Harry Martin, Ph.D., Dallas; R. L. Skrabanek, Ph.D., College Station; and Gideon Sjoberg, Ph.D., Austin.

Research Committee.—What should be the role of research in a comprehensive mental health program; where conducted, by whom, how financed, how coordinated, communicated, and utilized?

Dr. Neil R. Burch, Bert Kaplan, Ph.D., and Dr. Robert Tips of Houston; Richard Hill, Ph.D., Wayne Holtzman, Ph.D., John T. King, Ph.D., and Dr. Manuel J. Otero of Austin; and Maurice Korman, Ph.D., Dallas.

Training and Recruitment Committee (Manpower).—How can adequate numbers of professional persons be recruited, trained, and upgraded to meet

immediate and long range needs? Are there ways of multiplying effectiveness of present manpower?

Dr. Robert Berson and Sister Mary Immaculate of San Antonio; Laurie T. Callicut, Ph.D., and Mrs. Helen B. Kapiloff of Houston; Dr. Robert S. Glen, Dallas; James Oliver, Austin; and Faye Pannell, Denton.

Facilities and Construction Committee.—What additional facilities are needed; of what kind, where located, and how financed?

J. A. Anderson, Dr. Horace E. Cromer, Jr., Kenneth Nuhn, and Louis Page, all of Austin; Dr. E. Ivan Bruce, Jr., Galveston; Dr. H. F. Connally, Jr., Waco; John A. Davis, Fort Worth; and Manning Grinnan, Dallas.

Other Speakers

The citizens committee heard four other speakers besides Dr. Bayles during the day-long meeting in Austin. Welcoming them was the Honorable Crawford Martin, Secretary of State, who said, "Texas seems to have become a proving ground in mental health. Mental illness threatens the lives of more people than any other illness."

He continued to say mental illness injures not just the individual, but presents a drag on all those with whom the mentally ill person is associated.

Most people who receive early treatment return to normal lives, but "the tragedy and waste of mental health in Texas is a factor to be reckoned with," he said.

"I admire your willingness to grapple with so complex a problem. The work each of you do will be an asset to everyone else."

But, he added, "Your recommendations may follow the same fate as many excellent studies unless you inform the people." Fortunately, he said, people of Texas are aware now, more than ever before, that changes are necessary to help wipe out mental illness.

Martin said he hopes to see in the near future a plan of action on how the objectives the committee decides upon can be achieved and a timetable for the objectives.

State officials, he added, will be glad to aid wherever they can.

State Structure

Seventy thousand cases a year constitute only the visible part of the number of mentally ill people in Texas, according to Dr. James E. Peavy, Commissioner of the State Department of Health, another speaker. Dr. Peavy emphasized that this number represents only those people treated by private psychiatrists, general hospitals, medical schools, veterans' hospitals, and state mental hospitals. There are more that have not received treatment.

Not only the number of people involved, but subproblems of mental health planning have to be considered. These include strong research and rehabilitation and restoration centers, he said.

One of the main problems of the citizens' planning committee, he said, will be deciding on how to coordinate and cooperate with the state agencies directly and indirectly involved with mental health in Texas. A recent addition, the Office of Mental Health Planning, can provide the technical data needed for the citizens' work, he said.

Professional Resources

"Perhaps there is not a perfect solution, there may never be one. There are, however, large enough numbers of our citizens who become incapacitated by mental disorders to make what we do a necessity, not a matter of choice," Dr. Cyril J. Ruilmann, director of Mental Health and Hospitals, Board for Texas State Hospitals and Special Schools, said in a speech to the group.

In informing the citizens' committee of the professional resources in Texas, Dr. Ruilmann pointed out that "The keystone to mental health planning is the number of physicians, nurses, and trained personnel." There are others, teachers, who have big roles to play, he added.

"Certainly the supply of physicians is vital in all health fields," Dr. Ruilmann said. There are 10,920 licensed physicians in Texas; 437 are engaged in psychiatry.

"The amount of treatment will increase as facilities increase," he added. There are 295 new physicians in Texas each year, and 31 residents finish each year in psychiatry, exclusive of those in federal hospitals.

"Much of the care is provided, and should be, by other physicians, general practitioners and internists," he said. "How can this be expanded and expedited? More training is required as curriculum in medical schools."

There are about 32,158 registered nurses in Texas. While the national ratio of registered nurses per thousand people is 2.5, Texas has only 1.28.

There is a great need in Texas for more

auxiliary personnel and nurse-technicians.

He pointed out that there is need for more psychologists, especially clinical psychologists, and social workers in Texas.

"Research scientists are needed for any long-range plan," he said. "The great burden still rests with the laboratory and researchers. These are people, few in number, upon whom much depends. We must keep them in mind in our plans."

Community Resources

Robert L. Sutherland, Ph.D., temporary chairman of the general planning committee, emphasized the need for immediate action. We should start with the problem of how to get into a program what we already know ought to be in it, he said. "None of us is thinking loosely about rushing into a program of action, but if we do the traditional thing and wait five years for research data, we will not act.

"I don't mean to minimize data and research," he pointed out but added that evaluative research can be geared into action.

Dr. Sutherland said that from computer data, there is a systematic list of resources in each citizen's part of the state.

Resources for communities include organizations and nearby foundations. Texas has the largest number of foundations outside New York City.

Local organizations and churches could be a major resource, he added; these are too often overlooked now, and churches are seldom approached for help and funds.

"In spite of these resources, if you would visit a community, you could find from interviews that there are many people who do not know where to turn if a member of the family or friends have emotional problems.

"We have a long way to go," he said. "Many middle-sized cities do not have practicing therapists.

"The lack of provisions and the lack of knowledge is very important."

Executive Committee

The Executive Committee for the Texas Plan is composed of Dr. James E. Peavy, Dr. Cyril J. Ruilmann, and Robert L. Sutherland, Ph.D.

Radioisotope Workers' Eligibility Changed

Eligibility requirements for examinations of certification to the American Registry of Radioisotope Technologists have been changed, according to Mrs. Jewel Benoit, chairman of the Education Committee, Texas Society of X-ray Technicians.

These requirements will be in force for the next examination which will be held the first weekend in November of 1964 in conjunction with the November, 1964, examination in x-ray technology.

Mrs. Benoit announced that those wishing to take this examination should study these new requirements in order to be eligible.

Applicants may choose from two categories of requirements.

The first set of requirements specifies that candidates shall have had at least one year of full time experience in clinical radioisotope work, including didactic experience equivalent to the radioisotope curriculum recommended jointly by the ASXT and ASCP.

In addition, applicants must meet one of the following conditions: (1) graduate of AMA approved program in x-ray technology, (2) certified x-ray technologist by the ARRT, (3) registration as a professional nurse, (4) certified medical technologist by ASCP, (5) bachelor of science graduate in biology, chemistry, or physics, including at least 60 clock hours in basic course of human anatomy and physiology.

The second set of requirements specifies that the applicant must have: (1) successfully completed a course of at least two years in radioisotopes approved by ARRT, (2) graduated from four-year high school course, or the equivalent, plus at least five years of full-time experience in a clinical radioisotope laboratory or department which is ARRT approved, (3) been certified as an x-ray technologist by the ARRT, plus have had at least two years of full-time experience in a radioisotope laboratory or department which is ARRT approved.

Applications materials and full instructions may be obtained from the Executive Director, American Registry of Radiologic Technologists, 2600 Wayzata Boulevard, Minneapolis, Minn. 55405.

Mrs. Benoit urged those wishing to apply to write for their materials and instructions now, since applications must be postmarked

to the Registry Office in Minneapolis no later than midnight on Sept. 1, 1964.

Texans To Receive Radiology Fellowships

The degree of fellow of the American College of Radiology will be conferred on two Texas physicians at the forthcoming meeting of the College in Tucson, Ariz., on Feb. 5-8.

They are Dr. Dean B. Jones, San Antonio, and Dr. John P. McGraw, Houston.

Feb. 8 Meeting Set By 12th District Society

The 12th District Medical Society will meet at the Kyle Hotel in Temple on Feb. 8. Registration will begin at 9:30 a.m.

Dr. J. M. Brown of Marlin will preside at the meeting. The luncheon address will be given by Dr. Robert Mayo Tenery, President of Texas Medical Association, and is entitled "Medical Ethics in Daily Practice."

Scientific papers will be given by Dr. John M. Knox, professor and chairman, Department of Dermatology, Baylor University College of Medicine, "Causes, Treatment, and Prevention of Skin Cancer," and "Current Treatment of Common Dermatological Problems"; and Dr. Robert G. Freeman, assistant professor of pathology and dermatology, Baylor University College of Medicine, "Benign Skin Tumors—Pathophysiology and Treatment" and "Papulosquamous Diseases."

Dr. Stafford Tagged 9,000th Member

Last November, the Membership Office enrolled Texas Medical Association's 9,000th member.* He is a young general practitioner who is beginning the private practice of medicine in Lewisville, a thriving town 24 miles northwest of Dallas.

Dr. John R. Stafford, the latest milestone member, is a native Texan, born Oct. 29, 1934, in Houston. His father, William E. Stafford, an accountant, died as a result of injuries sustained in an automobile accident when young John was 18 months old. His mother, Mrs. Lettie Chambers Stafford, was employed in the Houston offices of Gulf Oil Corporation until her retirement in 1960. His maternal grandfather was a physician, Dr. Robert Alton Chambers, who practiced for a time in Lockhart during the 1890's.

DR. STAFFORD

Dr. Stafford attended public schools in Houston and was graduated in January, 1952, from San Jacinto High School. He enrolled in Rice University and in 1956 received a bachelor of arts degree in biology.

He left Houston for Galveston where he enrolled in the University of Texas Medical Branch. There he met the girl he married, the former Helen Grace Smith of Lubbock, who was in Galveston to attend the University of Texas School of Nursing. They now have two children, John Richard, Jr., "Rick," 4, and Helene, 2.

Following his graduation in 1960, Dr. Stafford served a one-year rotating general internship at Hermann Hospital in Houston. He was one of three interns who shared the Outstanding Intern Award for the year 1960-1961.

Upon completion of his internship, Dr. Stafford spent two years in the Navy. He was based in Florida during the last part of his tour of duty, and from there wrote the TMA Placement Service for information on Texas opportunities for general practice.

From the TMA listings, Dr. Stafford last April selected several which appealed to him and came to Texas for a first hand look.

The final selection of Lewisville as a place to live and practice, was really a "family project," Dr. Stafford says, but the decision to move there is one which he can support for a number of professional reasons. He is associated with the Doctor's Clinic in Lewisville which is a "branch office" of the Medical-Surgical Clinic Association of Denton. There are nine members in the Clinic, representing general practice and several specialties. One of the members, Dr. Stafford says, was three years ahead of him in school in Galveston.

Dr. Stafford and his family are Meth-

*Since that time enrollment increased to 9,078 as of Jan. 6.

odists. He enjoys photography and a variety of "do-it-yourself" projects such as hi-fi kit building, as well as sports such as tennis, golf, and bowling.

Housing Arrangements Announced For TMA Houston Annual Session

HOUSING ARRANGEMENTS for the 97th Annual Session of Texas Medical Association to be held in Houston April 23-26 are being arranged by the staff of the Shamrock Hilton in Houston.

Mr. E. N. Drake, executive assistant manager of the Shamrock Hilton, is coordinating arrangements for housing, and all reservations requests should be directed to him. Requests should indicate first, second, and third preferences. Insofar as possible, preferences will be honored and confirmation of reservations will be sent directly to the individual making the request.

A card listing hotels and motels and their location in respect to the headquarters hotels—the Shamrock Hilton and the Towers—has been inserted in this issue of the *Journal* opposite page 59.

A list of hotels and motels, addresses, and rates follows:

	Single	Double	Twin	Suite
Shamrock Hilton Hotel Main at Holcombe	$8-$20	$12-$20	$12-$20	$25 up ($14-$19, one room suite)
The Towers 2130 West Holcombe	$9-$15	$12-$16	$13-$18	$29.50-$65
Sheraton-Lincoln Hotel 777 Polk Avenue	$9.50-$15.50	$14.75-$16.50	$14.75-$19.50	$32.50 up
Hiway House 9300 South Main	$8-$12	$10.50-$16	$10.50-$16	
Tidelands Motor Inn 6500 South Main	$9-$14	$13-$20	$13-$20	$20-$29
Las Vegas Motor Hotel 9604 South Main	$7.75-$9.75	$9.75-$11.75	$10.75-$14.75	
The Warwick Main and Hermann	$8-$10	$11-$15	$13-$15	$25-$50
The Hotel Plaza 5020 Montrose	$6 up	$8.50 up	$8.50 up	$20-$35
Twenty-Nine Palms Motor Hotel 9430 South Main	$6.75-$7.75	$10.75		
Grant Hotel 8200 South Main	$7 up	$7.50-$8	$9-$12	
Sun Valley Motor Hotel 1310 Old Spanish Trail	$7.50	$8.50	$9.50	
Holiday Inn 10319 South Main	$7.25	$8.50-$9.50	$10.50-$12.50	$15.50 up
Sheriton Oaks Motel 7021 South Main	$8-$10	$10-$12	$12-$14	
The Surrey House 8330 South Main	$9	$11.50-$16.50	$13-$18	

Architect Describes New Concept For San Antonio Medical School

THE NEW University of Texas South Texas Medical School structure in San Antonio will be one of the few schools so far developed under one concept and designed as a complete unit, according to Max Brooks, architect with the Austin firm of Brooks and Barr, designers of the school.

Preliminary plans have been approved for the multi-million dollar brick and glass structure. (See picture of architects' model.)

The school is expected to be the nucleus of an extensive medical center with the Bexar County Teaching Hospital planned to adjoin the building. Across from the main entrance on Floyd Curl Drive will be the Southwest Texas Methodist Hospital.

The core of the medical school building contains teaching laboratories, lecture and conference rooms, cubicles for individual study, a book store, communications center, and student lounge.

The teaching laboratories have been designed for 16 students each and arranged so that they can serve about as well as multi-discipline or single discipline laboratories. The laboratories for anatomic dissections are also designed for 16 students each.

The center for student activities, consisting of the book store, post office, communications center, and student lounge is located immediately adjacent to the entrance of the library for convenience and so that students can become accustomed to making daily use of the library. From this center a main entrance leads across a paved courtyard which will be shaded from the afternoon sun and open to prevailing breezes. Student parking lies beyond.

On the upper and lower of the three main floors of the school, cubicles for individual study by first- and second-year students are located immediately adjacent to the teaching-lab-lecture-conference room complex.

The offices and laboratories for the faculty in the basic science departments are wrapped around the core of teaching labs, whereas the faculty facilities for the clinic departments are located in the wing of the building which leads to the teaching hospital.

Individual study cubicles for students in the third and fourth years have been located at those points in the teaching hospital where the corridors from the upper and lower of the three main floors of the school join the hospital. Four classrooms in the school large enough for a whole class have been included to facilitate the scheduling of whole class

Architect's Concept of Medical School

exercises.

Immediately adjacent to the front entrance and to the core of student facilities space is provided for student counseling, interviews, and functions of the registrar. The areas for the business office and the dean are located adjacent to the auditorium.

The 600-seat auditorium will be across a court from the main entrance of the school. It will be designed so that patients from the hospital may be brought in beds, or on stretchers or wheel chairs to view the auditorium stage.

The basement of the school will contain areas for anatomic preparation, animal quarters and operating rooms, storage of supplies and equipment, and an expansion area for the main power plant. It is anticipated that an off-site animal farm will be eventually developed for medium size and large animals which must be kept for a long time. Animals in the school building will be those under active study.

Because so much of the foot traffic will pass one point in the medical school and because only three floors will be heavily used, it is planned for most of the foot traffic to move from floor to floor by escalators.

Dallas Physicians Progress Against Lung Cancer

Texas physicians at Baylor University Medical Center in Dallas have reported encouraging results against certain lung cancers in an experimental series in which a combination of preliminary radiation and surgery was used.

The cancers are those found in the superior sulcus or upper tip of the lung, where surgery alone or radiation alone had not been effective in providing satisfactory survival rates, said Dr. John T. Mallams, director of radiation therapy and cancer research at the medical center. He spoke to the convention of the Radiological Society of North America meeting in Chicago last November.

Prior to the beginning of their series in 1956, the best reported survival for patients treated for these cancers was an average of 10 to 14 months after surgery with the longest survival 27 months. He reported on a total of 42 patients given the combined treatment. A third of 24 patients followed over two years were still alive and free from symptoms. One patient is still alive and healthy after seven years and another has survived the treatment for six years.

There are two departures in the method worked out by Dr. Mallams, Dr. Donald L. Paulson, Dr. Richard E. Collier, and Dr. Robert R. Shaw, all of Dallas.

The radiation therapy is used before surgery as a method of sterilizing the edges of the cancer, thus hoping to prevent the spread of cells by surgery. The 3,000 roentgens administered over 10-12 days tends to shrink but not destroy the cancer.

There is no biopsy for preliminary diagnosis because a biopsy on a lung segment constitutes a serious surgical procedure which might work against good treatment, he explained. Only five wrong diagnoses were made as a result of omitting the biopsy. Three were chronic tuberculosis and two were other cancers.

Some four weeks after radiation, the patients undergo surgery for resection of the upper tip of the lung. Usually, the surgeon is able to find a definite small cancer which can be removed with good hope of avoiding leaving a remnant, Dr. Mallams said. Thirty-three of the 42 patients, including the five wrong diagnoses, were operated on. Of the 28, eight are still alive after two years or more, 16 have died of cancer or other causes and four have survived less than two years to date.

Based upon their experience in the series, Dr. Mallams and his associates recommended that the combined preoperative radiation and surgery be the treatment of choice for upper lung cancers.

Texas Obstetricians, Gynecologists To Gather in Corpus Christi

The Texas Association of Obstetricians and Gynecologists will meet Feb. 28-29 in Corpus Christi at the Driscoll Hotel. Principal speaker will be Dr. Sprague Gardiner of Indianapolis, Ind., whose topics will be "Emotional Aspects of Obstetrics and Gynecology," and "Endometriosis: Bowel Lesions."

Other speakers will include Drs. Peggy Joyce Whalley, E. B. Mendel, and Paul McDonald, all of Dallas; Drs. A. M. Faris and Allen Kaplan, both of Houston; Dr. Charles Powell of Galveston, and Dr. Charles Gibbs of San Antonio.

Steroids

A REVIEW ENTITLED "The Effects of Structural Alteration on the Anti-inflammatory Properties of Hydrocortisone" provides a comprehensive and reliable summary of the literature pertaining to all facets of the field of structure-activity studies of hydrocortisone congeners.[7] Additionally, this review includes a summary of the important methods of biologic evaluation, clinical effects, clinical utility, side effects and clinical actions, correlation of human and animal data, some biologic parameters of anti-inflammatory steroids, the site of action, sites of loss, and physico-chemical parameters.

Sarett and others[7] conclude their review with the following point: "The anti-inflammatory activity of hydrocortisone is considered to be a function of both its concentration at the site of action and of its intrinsic activity." It therefore follows that chemical modifications applied to hydrocortisone can affect the biologic activity by altering the absorption-distribution pattern and/or the intrinsic chemical reactivity *per se.* It should be noted that concentration at the site of action depends on metabolic stability, as well as on absorption-distribution factors. Chemical modifications that lead to an increase in metabolic stability have been studied and applied with success. These authors also state that in the absence of evidence implicating a coenzymatic role for hydrocortisone it is convenient to visualize a reversible interaction with a hypothetical anti-inflammatory receptor site. Such a hypothesis is taken as the simplest basis for interpreting the effects of structural change on anti-inflammatory potency. Consequently, those structural modifications which do not appear to modify metabolic stability but which affect potency are considered to operate via a change in affinity for such a hypothetical receptor site. It also is proposed that the effect of chemical modifications on affinity is interpretable in many cases by an estimation of the binding energy contributed by the short-range attractive forces which govern binding between substrate and enzyme, an entropy effect which decreases the binding of analogues with greater flexibility, and hydrophobic bonds.

Ring A Olefinic Androstanes

In connection with the introductory statements it is pertinent to mention the views and work of Bowers and others.[1] These steroid chemists of Syntex, S. A. (Mexico) state that although little is known about the mechanism of action of steroid hormones at the cellular level, one may rationalize that the structure of an active hormone must meet certain requirements; that is, its structure should be compatible with the requisites for efficient distribution to its site of action; it must have reasonable metabolic stability; having reached the site of action the steroid should affect a certain metabolic process therein. With this background and the tenet that some form of interaction of steroid hormones involving proteins is a necessary requisite for bioaction, these workers proceeded to study the synthesis and androgenic-myotrophic activities of some Δ-1, Δ-2, Δ-3, and Δ-4 C-3-deoxy-androstanes. These steroids lack a C-3 oxygen atom but possess a center of unsaturation in Ring A. Thus, the importance of sp^2 hybridization at C-2 and C-3 was evaluated. The Δ-1, and Δ-2 and Δ-3 olefins of 5α-androstane-17-β-ol exhibit reasonable anabolic activity, the anabolic-androgenic ratio being more favorable in the latter two steroids. The corresponding Δ-4 compound exhibited only very low activity. Accordingly, it may be concluded that a high electron density at C_2 and/or C_3 in 17-β-hydroxyandrostane is a factor strongly promoting high myotrophic activity. *The desired structural feature may generally be defined to include all 17-β-hydroxy-androstanes which have a high electron density in or near the C-2 and C-3 positions.* This definition includes compounds such as C-3-alcohols which can undergo in vivo transformation to such a structural feature. The preceding generalizations

are substantiated with other studies by the Syntex group; that is, studies on 2-methylene-androstanes and 2-methyl-Δ-1, Δ-2 and Δ-3-androstenes; 2-formyl-Δ-2-androstenes; 2-chloro- and 2-halomethyl-Δ-2-androstenes; 2-methylene-3-oxygenated androstanes.

It is also important to emphasize that Syntex's 2-formyl-Δ-2-androstenes and the 2-hydroxymethyl related compounds represent a new class of potent anabolic agents. The androgenic and anabolic activities of these 2-formyl- and 2-hydroxy-methylandrostenes reveals that substitution at C-2 by hydroxymethyl or formyl groups leads to a substantial reduction of androgenic activity with retention of significant anabolic properties. The ratio of anabolic to androgenic activity is particularly favorable with the 2-hydroxy-methyl compound.

Thus, the preceding résumé should exemplify one of the avenues of research that has led to the development of more selective anabolic steroids with a low degree of undesirable androgenicity. Secondly, from the structure-activity viewpoint it is fundamental to recognize that the work reviewed substantiates the studies of Clinton[4] and others that taught us that the C-3 oxygen atom is not essential for activity.

Novel Anti-Inflammatory Steroids

Many structure-activity studies on steroids leading to the systematic removal of oxygen atoms from hydrocortisone have been motivated by a desire to determine which ones are essential for anti-inflammatory activity. Some of these deoxy compounds are metabolic products of hydrocortisone, that is, 11-deoxyhydrocortisone; 17-deoxyhydrocortisone; 11β, 17α, 20β, 21-tetrahydroxy-4-pregnene-3-one; 3β, 11β, 17α, 21-tetrahydroxy-5β-pregnane-20-one. These metabolic changes effect a decrease in the anti-inflammatory activity of cortisone.[7] The effect of reducing or removing the 3-ketone of hydrocortisone is obscure because the metabolic products are reduced at the 4, 5-alkene linkage and also at the ketone carbonyl to form 3-hydroxy compounds which are known to be inactive.

Nevertheless, it can be concluded that no potent anti-inflammatory agent lacking a C-3-oxygen had been described in the literature prior to a recent report on the steroidal 4-pregneno-(3,2-c)-pyrazoles in the *Journal of the American Chemical Society*.[5] This report by researchers of Merck Sharp and Dohme and the Merck Institute for Therapeutic Research describes steroidal pyrazole derivatives with a high degree of anti-inflammatory activity.

In a previous review[3] in the *Journal*, reference was made to Clinton's work that led to the advent of (3,2c)-pyrazole congeners of androstanes as anabolic agents, hence it is of interest to review some analogues. Clinton's research on the pyrazole-androstanes was directed toward the development of steroids with high anabolic activity and minimal androgenicity. As a consequence, stanozolol (Winstrol) now is well recognized as a relatively selective anabolic agent and is listed in the "New and Nonofficial Drugs, 1963."

The case of the 4-pregneno-(3,2c)-pyrazoles represents a novel class of potent anti-inflammatory steroids. 11β, 17α, 21-trihydroxy-9α-fluoro-16α-methyl-20-oxo-4-pregneno(3,2-c) pyrazole is reported to be approximately ten to twelve times as active as hydrocortisone in the systemic granuloma assay and only three to four times as active in causing adrenal atrophy. Noteworthy is the notation that this compound did not lead to sodium retention in the adrenalectomized rat. The 2′-phenylpyrazole analogue and the two 2′-p-fluorophenylpyrazole analogues were respectively 60, 100, and 500 times more active than hydrocortisone in the granuloma assay. Consequently, the conclusion is made that the 2′-p-fluorophenylpyrazole moiety is the most powerful activity enhancing group to be reported in the field of anti-inflammatory agents. This revelation is a major contribution to the field of structure-activity studies in medicinal chemistry.

Similar studies have been conducted on the 6-dehydro-6-methylhydrocortisone series.[4] Among the members of this new series the (3,2c)-2′-phenylpyrazole derivative of 9α-fluoro-6, 16α-dimethyl-Δ-6-hydrocortisone is reported to be by far the most potent corticoid. This compound is 2,000 times more active than hydrocortisone in the rat granuloma assay.

Hypocholesterolemic Steroidal Pyrazoles

In the latest issue of the *Journal of Medicinal Chemistry*, Robinson and others[6] of the Schering Corporation have summarized their observations on the synthesis and properties of 1, 3, 5 (10)-estratrieno-(17, 16-c)-pyrazoles. These compounds are heterocyclic ster-

oids with a pyrazole ring fused to Ring-D of the steroid nucleus. In accordance with data that certain estrogenic compounds possess hypocholesterolemic activity, Robinson's group synthesized these compounds as hypocholesterolemic agents with the hopes that they would exhibit minimal feminizing activity.

The 3-methoxy-1, 3, 5(10)-estratrieno-(17, 16-C)-pyrazole compound exhibited hypocholesterolemic activity approximately equal to that shown by estradiol, while the estrogenic potency, measured in the mouse uterotrophic assay was less than 0.0001 times that of estradiol.

17-Alkylated Progesterone Analogues

The natural metabolite of the corpus luteum, progesterone, is fundamental in the physiology of the female reproductive system and also participates as an intermediate in the biosynthesis of the adrenalcortical steroids, the androgens and estrogens. It has been rationalized that for the reason of biological economy, natural hydroxylated progesterone analogues are less active than their precursor. It seems that polarity and metabolic factors are more critical than stereochemical ones since compounds with drastically different stereochemistry like retroprogesterone are known to be active, but introduction of a single hydroxyl in position-11 of progesterone greatly reduces its progestogenic activity. Other metabolic mechanisms of such deactivation include hydroxylations at position 16α, 17α, reduction of the 20-ketone and inversion of the C-17 acetyl side chain. Protection of the C-17 side chain is an essential prerequisite for oral activity or absence of side effects due to metabolites. The 17α-substituents meet at least partly this requirement. Accordingly, Deghenghi and others of Ayerst Research Laboratories, Montreal, have synthesized and evaluated a series of 17α-alkylated progesterones. These workers report that the oral activity (rabbit, Cauberg-McPhail test) increases in the order methyl < ethyl < n-propyl. The most potent derivatives are 17α-methyl-6-chloro-6-dehydro-progesterone and 17α-ethyl-6-methyl-6-dehydroprogesterone. Within this series both C-6 and C-17 substituents have to be present for high oral activity; their main function is probably due to a block of metabolic inactivation.[2]

—Jaime N. Delgado, Ph.D.,
Frank P. Cosgrove, Ph.D.,
University of Texas College of Pharmacy.

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First District Society Sets Feb. 1 Meet in El Paso

First District Society will meet at R. E. Thomason General Hospital in El Paso on Feb. 1. A postgraduate course will be offered on Feb. 2.

The meeting will open at noon with a luncheon to be followed by four scientific presentations: "Extraperitoneal Hernia Repair," Dr. R. L. Deter; "Stapes Operation," Dr. C. M. Stanfill; "Use and Abuse of Newer Anesthetic Agents," Dr. L. W. Neill; and "Ectopic Pregnancy," Dr. G. M. Landis, all of El Paso.

Visiting physicians and their wives will be guests of the El Paso County Medical Society for a social hour and dinner at El Paso Manor where they will be addressed by Dr. Robert Mayo Tenery, President, Texas Medical Association.

Pamphlets

A pamphlet, "Self-Help Clothing for Handicapped Children," is designed to serve as a guide for parents and professional personnel in the selection and adaptation of clothing for handicapped children. It emphasizes that the handicapped child can learn to have some part in his personal matters if he is given clothes that are easy to manipulate, plus guidance, time, encouragement, and pa-

tience. The different clothing needs for children in wheel chairs, on crutches, and children with other disabilities is clearly illustrated.

The booklet costs 50 cents and may be obtained from the National Society for Crippled Children and Adults, 2023 West Ogden Ave., Chicago 12, Ill.

Book Notes

Books reviewed in this column are available from TMA Memorial Library.

Gastro-Intestinal Physiology. D. F. Magee, Ph.D. 225 pp. Springfield, Ill., Charles C Thomas, 1962. $11.50.

IT IS HOPED that this book will be of value to medical students and to medical practitioners. There are fashions in physiology just as there are in dress and speech. The gastrointestinal canal has not been a fashion leader for a number of years and for that reason many not engaged in active research in the field have found it difficult to acquire understanding of recent developments. Many of these developments have considerable practical importance. It is hoped that this text will enable readers to obtain a basic understanding of the subject as it is today and also to gain some idea of the directions in which research is currently proceeding.

"No, I don't think 'subdural hematoma.' I think it's a glass eye."

Curable and Incurable Neurotics. Edmund Bergler, M.D. 453 pp. New York, Liveright Publishing Corp., 1961. $6.95.

DR. BERGLER presents a candid investigation of what psychoanalytic psychiatry can and cannot do. Are the limitations inherent in the material, the technique, the patient, or the therapist? The concept of malignant versus neurotic, psychic masochism is introduced and the thesis offered that most "incurable" neurotics are undetected or unavoidably detected too late.

Vaginal Hysterectomy: Indications, Technique, and Complications. Laman A. Gray, M.D., 2nd ed. 228 pp. Springfield, Ill., Charles C Thomas, 1963. $9.50.

THE BOOK IS unique and ought to be on the reading list of every resident in obstetrics and gynecology.

I liked the system of direct reference by number to the bibliography when an author is quoted or his methods described. The description of how the patient's legs should be supported in the lithotomy position appealed to me since I have a pair of leg supports that accomplishes what the author describes. Assistants can help and see more efficiently and comfortably; and even more important, an advantage accrues to the patient in that the straighter position of the legs produces less danger of venostasis.

I liked the detailed description of the preoperative preparation and the listing of instruments and drapes. Important points in operative technique are stated so simply and illustrated so well as to permit no misinterpretation.

The postoperative care as described is very practical. It is reassuring to read of the trials encountered by those with much experience in various complications, particularly the commonly found difficulties in reestablishing normal voiding after repair, and

to read in print about the very measures to which one is accustomed to using to overcome these barriers. One of my teachers was fond of saying that if no difficulty in voiding was present after cystocele repair that the operation was probably inadequate.

—John J. Young, M.D., El Paso.

Bilharziasis. Ciba Foundation Symposium. 416 pp. Boston, Little, Brown, and Co., 1962. $11.50.

IN THIS SYMPOSIUM, 26 world authorities thoroughly discuss modern aspects of research on bilharziasis and its control. In addition to summarizing progress to date, their findings delineate many hopeful lines of attack for the future. The discussion begins with the epidemiology of bilharziasis, the ecology of its intermediate hosts, and the parasites and their intermediate hosts. They continue with the physiology of the schistosomes, clinical and pathological aspects of bilharziasis, and current immunological studies and chemotherapy.

Specificity of Serological Reactions. Karl Landsteiner, M.D. 330 pp. New York, Dover Publications, Inc., 1962. $2.

DR. LANDSTEINER, who won the Nobel Prize in 1930 for the discovery of blood groups, devoted his life to fundamental research and played a guiding role in the development of several important branches of immunology. This book, first published in 1936 and revised in 1945, is the authoritative account of the discoveries he and his colleagues made in antigens and serological reactions with simple compounds.

Included in this edition are the final chapter on molecular structure and intermolecular forces by Linus Pauling, Ph.D., and a new introduction to this edition by Merrill W. Chase, Ph.D., of Rockefeller Institute.

A History of Wine as Therapy. Salvatore P. Lucia, M.D., 234 pp. Philadelphia, J. B. Lippincott Co., 1963.

THE BOOK traces the use of wine in medicine by delving into the works of the great physicians—Hippocrates, Pliny, Galen, Maimonides on through Anstie, Yorke-Davies, Sir William Osler, into modern experimental medicine.

Dr. Lucia long has been interested, also, in wine as a dietary beverage and as a medicine. He has lectured and written on the physiologic effects of wine and its constituents. "Wine and Food as Medicine" was published in 1954. Dr. Lucia served as chairman of the symposium, "Alcohol and Civilization," held in 1961 at the University of California.

Handbook of the Practice of Anesthesia. John R. S. Shields, M.D. 203 pp. St. Louis, C. V. Mosby Co., 1963. $6.85.

THIS IS A worthwhile compendium of modern anesthesiologic practices, stressing the "practical" or operating room performance. Many illustrations are redundant and in Fig. 30 "arterial" is mislabeled for "anterior." The sequelae of conduction anesthesia have not been caused by and large "by infection" (p. 102).

Choice of anesthetic agents and techniques is acceptable but remains a matter of personal judgment and experience. The numerous electrocardiographic tracings are of good quality and serve as a valuable teaching reference.

—R. H. Intress, M.D., Austin.

New Books in TMA Library

Gibson, H. L.: Photography of Patients, 2nd ed., Springfield, Ill., Charles C Thomas, 1960.

Miale, J. B.: Laboratory Medicine, Hematology, 2nd ed., St. Louis, C. V. Mosby Co., 1962.

Wijsmuller, G.: "Naturally Acquired Tuberculin Sensitivity in New Guinea" in Selected Papers, vol. 6, The Hague, Holland, Royal Netherlands Tuberculosis Association, 1963.

Miller, S. E., Ed.: Textbook of Clinical Pathology, 6th ed., Baltimore, Williams & Wilkins Co., 1960.

DeBakey, M. E.: Year Book of General Surgery, Chicago, Year Book Medical Publishers, 1963.

Koss, L. G.: Diagnostic Cytology and Its Histopathologic Bases, Philadelphia, J. B. Lippincott Co., 1961.

Proceedings Conference of Professional and Scientific Societies, Chicago, Commission on Drug Safety, 1963.

Engley, F. B., Jr.: Pocket Reference Guide to Medical Microbiology, Boston, Little, Brown and Co., 1963.

Clark, R. L., and Cumley, R. W., Eds.: Book of Health, 2nd ed., Princeton, N. J., D. Von Nostrand Co., Inc., 1962.

Gordon, B. L., Ed.: Current Medical Terminology 1964, Chicago, American Medical Association, 1964.

Clark, L. L., Ed.: Proceedings of International Congress on Technology and Blindness, 2nd ed., vol. 1-4, New York, American Foundation for the Blind, 1963.

Recent Deaths

DR. L. S. DEASON

Dr. Loyd S. Deason, 66, died in Henderson Memorial Hospital Aug. 6. He was a native of Rusk County and had practiced medicine in Henderson 32 years.

Dr. Deason was born Aug. 2, 1897, at Brachfield. He was the son of the late Dr. and Mrs. Tim Deason and his father was a widely-known Rusk County physician.

He attended school in Minden, Tex., and received his B.A. degree from Baylor University in Waco and his M.D. degree from Baylor University College of Medicine in Dallas. He interned at Baylor University Hospital in Dallas.

In 1931 he began the practice of internal medicine in Henderson and devoted particular attention to diseases of the chest.

He practiced for a time before World War II at the State Sanatorium for Tuberculosis in Norton, Kan.

During the war he served in the South Pacific in the Navy and was a captain in the U. S. Navy reserve.

Dr. Deason was a member of the Rusk County Medical Society, of which he was once president, the Texas Medical Association, and the American Medical Association. He was a former member of the House of Delegates at TMA.

He married the former Nanie Sarah Taylor of Mt. Enterprise, Tex., in a Jacksonville ceremony on July 23, 1927.

Dr. Deason was an active member of the Texas Heart Association. He was a member of the state board of directors and a member of the following heart association committees: personnel policy, certification, professional education, and reorganization. He was past president of the Rusk County Heart Association and the East Texas Heart Association.

He was former chief of staff of Henderson Memorial Hospital and was a member of the American Academy of Tuberculosis Physicians, the Trudeau Society, American Academy of General Practice, the American College of Chest Physicians, and the Texas Society of Athletic Team Physicians.

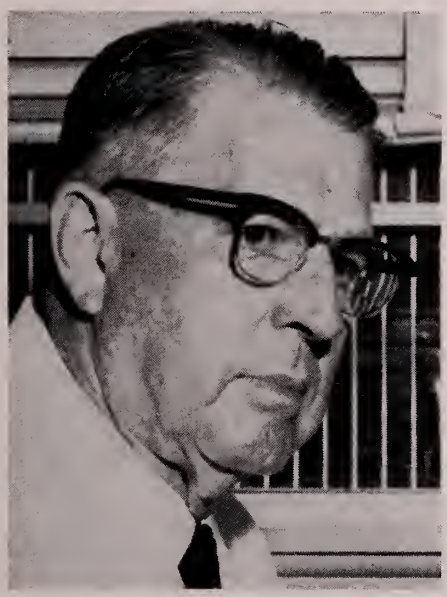
He was a Baptist.

Dr. Deason was interested in fishing and in miniature trains. He was a football enthusiast.

He is survived by his wife, one son, T. D. Deason, two brothers, Charlie Deason and Andrew Deason, all of Henderson; and a third brother, Franklin Deason of Brachfield.

DR. W. M. KNOWLES

Dr. W. Mood Knowles, a Dallas eye, ear, nose, and throat specialist, died Nov. 17 at



DR. L. S. DEASON

DR. W. M. KNOWLES

his home from a heart attack after a brief illness. A native of Kaufman, he had lived in Dallas most of his adult life and had practiced there for 40 years.

He was a former secretary of the board of managers of Parkland Hospital and had been a member of the staff of both St. Paul and Parkland hospitals.

Dr. Knowles grew up in Kemp. He was one of the six children of William B. and Sarah (Williams) Knowles. He attended Southwestern University in Georgetown and received his medical degree from Baylor University College of Medicine. He interned at St. Paul Hospital in Dallas and later did postgraduate work at Chicago Polyclinic. He was certified by the American Board of Otolaryngology.

He married Grace Covell of Portland, Ore., on June 3, 1915, in Dallas. She died on Aug. 25, 1959. On June 11, 1961, he married the former Mrs. Harry Penniman, Jr., of Dallas.

He was a member of the Dallas County Medical Society, Texas Medical Association, American Medical Association, the American Academy of Ophthalmology and Otolaryngology, Texas Otolaryngological Association, and Dallas Southern Clinical Society.

He belonged to Kappa Alpha fraternity, Highland Park Methodist Church, Lakewood Country Club, Dallas Kiwanis Club, Texas Historical Society, and the International Graphoanalysis Society.

His hobbies included playing the organ, studying foreign languages, and handwriting analysis. He was a Mason and a member of Hella Temple of the Scottish Rite.

Survivors include his wife; a son, Richard C. Knowles of Corpus Christi; and two grandchildren.

DR. B. E. LAURIE

Dr. Ben E. Laurie of Houston died on Nov. 21. He was 63 years old and had been in general practice in Houston for 19 years.

Born near Lockhart on June 8, 1900, Dr. Laurie moved with his family to Austin when he was quite young. His parents were Mr. and Mrs. Ben A. Laurie. He left high school in Austin to enter the Navy during World War I. Following the war, he returned to high school to get his diploma and to go to college at the University of Texas, Baylor University, and Baylor University College of Medicine from which he received his M.D. degree.

DR. B. E. LAURIE

He interned at Jefferson Davis Hospital in Houston.

Dr. Laurie first practiced in Bertram. After that he was in Rockdale for 13 years and was then employed as chief industrial surgeon for the Dow Chemical Co. in Freeport for two years.

He was a member of the Harris County Medical Society, Texas Medical Association, and the American Medical Association.

A lover of flowers, he was a member of the National Iris Society and had a prize garden of iris at his home.

Survivors are his wife, Mrs. Faye Laurie of Houston; and his mother, Mrs. Ben A. Laurie of Austin.

DR. P. H. McNEMER

Dr. Philip Hamilton McNemer died Nov. 10, at Hotel Dieu Hospital in Beaumont. He was 61.

He was born on May 3, 1902, in Austin, the son of Philip Hamilton and Ella (Baldridge) McNemer. He attended Bethel College in Russellville, Ky., and Southern Methodist University. In 1927 he received his M.D. degree from Baylor University College of Medicine.

He interned at St. Francis Hospital in Peoria, Ill. He entered practice in Cairo, Ill., in 1928 and left to enter the Navy in 1942 for which he served as a commander in the medical corps. After the war he moved to Beaumont to practice.

DR. P. H. McNEMER

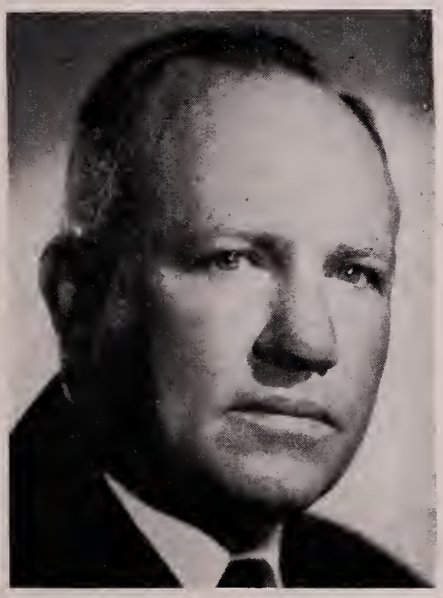
He married the former Era Nell Brown of Rochester, Ky., on May 19, 1946, in Henderson, Ky.

He was a member of the Jefferson County Medical Society, the Texas Medical Association, the American Medical Association, and the American Academy of General Practice.

He is survived by his wife; his son, Philip Hastings McNemer of Boston; and a sister, Mrs. J. Bart Aldridge of Wewoka, Okla.

DR. H. E. DUNCAN

Dr. Horace E. Duncan died in Dallas on Dec. 2.



DR. H. E. DUNCAN

He was born in Larue, Tex., on Dec. 18, 1900. He attended Grand Prairie high school, Burleson College in Greenville, Southern Methodist University, and received a bachelor's degree from Baylor University. In 1929 he was graduated from Baylor University College of Medicine.

Following an internship at Christ Hospital in Jersey City, N.J., and residencies at Margaret Hague Maternity Hospital, Jersey City, and Johns Hopkins, Baltimore, he began practice in Malakoff, Tex., in 1930. He moved to Dallas in 1932. He was appointed health officer Jan. 1, 1933, a position he held until Jan. 1, 1947, when he resigned to join the Veterans Administration branch medical office. He retired from this office in August, 1962.

During World War II, Dr. Duncan was a sanitation officer with the U.S. Navy.

Dr. Duncan was a school teacher before entering medical school, having taught in Plainview and coached at Breckenridge.

He was a member of the Dallas County Medical Society, Texas Medical Association, American Medical Association, Texas Public Health Association, and the American Public Health Association. He was elected to honorary membership in TMA in 1963.

A member of the Masonic Order, he also was affiliated with the Kiwanis Club and the Cliff Temple Baptist Church in Dallas.

Dr. Duncan's survivors are his wife of Dallas; three daughters, Mrs. Patricia Klein of Dallas and Mrs. Barbara Ann Meyer and Mrs. Jacque Epps of Houston; his parents, Mr. and Mrs. J. D. Duncan; a sister, Mrs. Flora Godwin; brothers, Roy P. Duncan and Dan F. Duncan, all of Dallas; brothers, John Paul Duncan of St. Louis, Silas Duncan of Denver, and Harold A. Duncan of Denton; and nine grandchildren.

DR. L. P. MAYES

Dr. Lee P. Mayes of Pasadena, a cardiologist and internist, died Nov. 15. He was reading in bed at his home when death occurred, the result of an acute coronary attack. The 56-year-old physician had practiced in Pasadena for 15 years.

He was a 1932 graduate of the University of Texas Medical Branch in Galveston and had done postgraduate work at the University of Pennsylvania School of Medicine in

DR. L. P. MAYES

Pennsylvania and Cornell University Medical College in New York City. He interned at Grady Hospital in Atlanta, Ga., and with U. S. Public Health Service in Portland, Me.

Dr. Mayes was born Sept. 9, 1907, in Floresville, Tex., the son of a Wilson County judge, Elmo D. Mayes, and Mary Houston Mayes. He attended high school there and went on to graduate from the University of Texas in Austin.

Dr. Mayes spent ten years—1935 to 1945—in the U. S. Army holding the rank of lieutenant colonel. He practiced medicine in San Antonio, Carlsbad, N.M., and Baytown before moving to Pasadena.

On Dec. 3, 1935, he married the former Mildred M. Chase of Pittsburgh, Pa.

He was a member of the Harris County Medical Society, Texas Medical Association, the American Medical Association, American Diabetic Association, Southern Medical Association, and Zeta Chapter of Phi Chi Medical fraternity.

A Roman Catholic, he belonged to the Knights of Columbus and the Catholic Physicians Guild of Houston.

He is survived by his wife, Mrs. Mildred C. Mayes of Pasadena, and one brother, John H. Mayes of Floresville.

Information for Authors

Material for the *Texas State Journal of Medicine* may be sent to the Managing Editor, 1801 North Lamar Blvd., Austin 78701. It must be offered solely to this journal. Each paper is reviewed by a consultant specialist and an editorial board, and is accepted or rejected on the basis of its individual merit and the availability of other material.

Manuscripts should be typed double-spaced with ample margins on firm paper. Carbons are unacceptable, but the author should keep a copy.

The *title* should include the words most suitable for indexing the article in "Index Medicus," should stress the main point, and should be brief. Subtitles should not be connected grammatically with the main title.

The *authors' names* and degrees should follow the title, and their mailing addresses should be listed at the end of the article. A glossy picture of the senior author is needed.

The *text* should be narrative with complete sentences, *few abbreviations*, and logical subheadings. For *spelling and usage* the editors follow "Dorland's Illustrated Medical Dictionary," 23rd edition, and "Webster's Third New International Dictionary, Unabridged." *Quotations* must give credit to both author and source. A concise *summary* or conclusion is of professional value.

Reference lists, alphabetized by authors, should contain in this order (1) author's last name, initials, (2) title of article or book, (3) name of journal, (4) volume and first page numbers, written 33:156, (5) month of issue, (6) year. For books, the title should be followed by place of publication, name of publisher, and year.

Illustrations should be black and white drawings or positive photographs, with neat, uniform, fairly large lettering. A label pasted to the back of each illustration should indicate its number, top, author's name, and title of article in brief. *Legends* should be in complete sentences, numbered, and typed on a separate sheet. *Color illustrations* are authorized if they are of special value and if the author arranges to pay part of the extra expense incurred.

Tables should be typed on separate sheets. Column headings should show points of similarity; side headings, points of difference.

All material is subject to *editing*, but authors receive proofs to check before publication. Factual and typographical errors should be corrected, but because resetting type is costly, further revisions should be kept to a minimum. Prompt return of proofs to the *Journal* office avoids delay in publication.

Reprints are available at cost plus 2 per cent sales tax. Authors automatically receive order blanks when their articles are published.

Permission should be obtained from the publishers and the authors for any previously published material (extensive textual matter, illustrations, tables) used. Short verbatim quotations in the text may be used without permission, but should be quoted exactly with the source credited in the bibliography. Copies of permission letters should be submitted with the manuscript.

Coming Meetings

American Medical Association, Annual Meeting, San Francisco, Calif., June 21-25, 1964. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Houston, Annual Session, April 23-26, 1964; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1964. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

JANUARY

International Medical Assembly of Southwest Texas, San Antonio, Jan. 30-Feb. 1, 1964. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Texas Radiological Society, Dallas, Jan. 30-31, 1964. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Society of Pathologists, Inc., Dallas, Jan. 25-26, 1964. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

FEBRUARY

American Academy of Allergy, San Francisco, Feb. 7-12, 1964. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American College of Radiology, Tucson, Ariz., Feb. 2-8, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American Society of Clinical Pathologists, New Orleans, Feb. 19-23, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

Texas Association of Obstetricians and Gynecologists, Corpus Christi, Feb. 29, 1964. Dr. H. W. Savage, 815 Fifth Ave., Fort Worth, Sec.

Texas Public Health Association, Austin, Feb. 23-26, 1964. J. N. Murphy, Jr., P.O. Box 5192, Austin 31, Sec.

Texas Urological Society, Austin, Feb. 8-9, 1964. Dr. B. C. Bates, 1108 Nueces, Austin, Sec.

First District Society, El Paso, Feb. 1, 1964. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Twelfth District Society, Temple, Feb. 8, 1964. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

National and Regional

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, Atlantic City, April 11-17, 1964. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Philadelphia, April 20-22, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Association of Plastic Surgeons, Chicago, May 13-16, 1964. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N.Y., Sec.

American Association for Thoracic Surgery, Montreal, Canada, April 27-29, 1964. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists, Bal Harbour, Fla., March 1-6, 1964. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, San Francisco, Calif., June 18-22, 1964. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Obstetricians and Gynecologists, Miami Beach, Fla., May 17-22, 1964. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, Atlantic City, April 6-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Hawaii, June 27-July 2, 1964. Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Sec.

American Gastroenterological Association, Dallas, April 24-25, 1964. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C.

American Gynecological Society, Hot Springs, Va., May 25-27, 1964. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Medical Women's Association, Roanoke, Va., May 31-June 2, 1964. Dr. Jessie Brodie, 1790 Broadway, New York 19, Exec. Sec.

American Neurological Association, Atlantic City, June 15-17, 1964. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Philadelphia, May 10-14, 1964. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Surgical Association, Hot Springs, Va., April 1-3, 1964. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., Chicago, April 3-4, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons, Vienna, Austria, May 11-16, 1964. Dr. W. F. James, 1615 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico. Dr. C. Kalil, 200 East Monterey Way, Phoenix, Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Oklahoma City, March 27-28, 1964. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum, Hot Springs, Ark., April 5-7, 1964. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., October, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Society of Nuclear Medicine, Houston, March, 1964. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, San Antonio, April 26-29, 1964. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, Corpus Christi, May 15-16, 1964. Dr. Sanford Glanz, 2425 Medical Center, Corpus Christi, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Houston, April 23-26, 1964. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Public Health Physicians, Houston, April 23, 1964. Dr. W. S. Brumage, 2600 Westover Rd., Austin, Sec.

Texas Chapter, American Academy of Pediatrics, Houston, April 24, 1964. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, Houston, April 26, 1964. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Houston, April 25, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, Houston, April 26, 1964. Dr. John W. Chriss, 2436 Morgan, Corpus Christi, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, Houston, April 23, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, Houston, April 26, 1964. Dr. E. I. Bruce, Jr., 1014 Strand, Galveston, Sec.

Texas Ophthalmological Association, Houston, April 23-26, 1964. Dr. Harold Hunt, 150 Eighth St., S.E., Paris, Sec.

Texas Orthopaedic Association, Houston April 23-26, 1964. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, Houston, April 23-26, 1964. Dr. J. P. Moran, 3154 Reid Dr., Corpus Christi, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, Houston, April 23-26, 1964. Dr. D. Hoehn, Doctor's Bldg., Fort Worth, Sec.

Texas Proctologic Society, Houston, April 23, 1964. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, Houston, April 26, 1964. Dr. L. Duflot, 173 San Marino, Galveston, Sec.

Texas Society of Athletic Team Physicians, Houston, April 23, 1964. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology. Dr. O. Suehs, 14 Medical Arts Square, Austin, Sec.

Texas Society of Plastic Surgeons, Houston, April 23, 1964. Dr. W. C. Sellman, Jr., 610 Medical Arts Bldg., Dallas, Sec.

Texas State Board of Examiners in Basic Sciences, April, 1964. Mrs. Maria P. Hathaway, 1012 State Office Bldg., Austin, Exec Sec.

Texas State Board of Medical Examiners, Houston, Jan. 24-26, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Surgical Society, Waco, April 5-7, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Houston, April 23, 1964. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Austin, March 19-21, 1964. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

United States-Mexico Border Public Health Association, Monterrey, Mexico, March 2-5, 1964. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Huntsville, March 19, 1964. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, Tex., Spring, 1964. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Thirteenth District Society. Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 15-18, 1964. Dr. W. O. Tschumy, 1616 Medical Arts Bldg., Dallas, Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 2-4, 1964. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 412 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals. Mr. R. L. Heath, Malone-Hogan Clinic, Big Spring, Sec.

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Youth Encouraged To Study Medicine

For several years the American Medical Association has been keenly aware that fewer qualified students look to medicine as their future careers. Realizing that the rapidly growing population would ultimately tax the number of physicians and, thereby, profoundly affect quality and quantity in practice, teaching, and research, the AMA embarked on a nationwide effort to recruit qualified students for careers in medicine. Soon advice and materials were provided to medical societies which accepted the challenge to help maintain for our country the finest medical care of this century. One of these was the Bexar County Medical Society, which created a medical careers committee in October, 1961. It developed a program geared not only to encourage promising young students to pursue careers in medicine, but also to convince them and their parents that the goal could be more easily achieved, both academically and financially, than is now generally believed.

The first activity involved the annual Science Fair for junior and senior high school students. A member of the committee was accepted to the board of directors of the Alamo District Science Fair. The Bexar County Medical Society annually gave awards for outstanding exhibits related to fields of medicine, contributed cash to the fair's budget, and sponsored an exhibit on careers in medicine. This allowed distribution of medical career pamphlets to, and direct contact with, several thousand students, parents, and teachers. We heartily recommend that local medical societies support and participate in local Science Fairs, for it is a rewarding experience. The AMA annually presents special awards and trips to National Science Fair winners. Here, the finest type of good public relations is achieved with such little effort and cost! Most important, there has been a continuous increase in

participation in the Science Fairs of students interested in the biological sciences; many of them eventually pursue careers in medicine.

Next, for students' reference, the committee gathered various types of information concerning premedical and medical education, including the annual catalogues of undergraduate and medical schools, academic information, and lists of scholarships, loans, and similar financial data. A special section was set up in the library of the Bexar County Medical Society for this purpose. Through talks and news media, students were invited to use this material as well as the library's general medical references.

Throughout the year, talks were given and panels on careers in medicine presented to junior and senior high schools as well as local college groups. These were made by special requests, after several newspaper articles were published which described the committee's function and volunteered its services. Speakers were always available; material was from the AMA and from sporadic articles in various publications about medical education and medical careers. The talks were made to special honor society science students, general classes during Career Week, P-TA groups, and premed groups at local colleges. During some talks, films from AMA were shown and these were enthusiastically received. The most popular was "I Am a Doctor," but others are available.

Occasional telephone queries from students and parents concerning premedical or medical education were referred to members of our committee. Responses took little time and were always appreciated.

Premed Clubs have existed at the three major colleges in San Antonio for several years. Our function was to volunteer speakers and programs and offer information about the international premedical honor society, Alpha Epsilon Delta.

One of the highlights of the past year's program was a panel discussion on "Careers in Medicine." It was presented to a full house of students, parents, teachers, and counselors. The panel included an undergraduate school professor and premed advisor, a premed student, a recent medical school graduate, a representative from a Texas medical school, and three practicing physicians. Every aspect of a premedical and medical education was discussed, and views of medical practice and medical careers were presented. This was followed by questions from an enthusiastic audience. Our committee plans to make this an annual event.

Since such activities continue on an annual —in fact, daily—basis, we are convinced that they earn deep and warm appreciation. Not only is the good image of organized medicine developed, but qualified students are encouraged to look to medicine as a future career.

Although every effort is made to avoid political implications, one cannot deny that such activities reap inestimable rewards of friendly support and strong allies. To remain strong and independent, the medical profession must be vigilant in keeping its ranks filled, in quantity and quality, with sufficient doctors to serve our nation. Even busy physicians can take this responsibility themselves, and they can do a better job than politicians.

—PETER B. KAMIN, M.D., Chairman, Committee on Medical Careers, Bexar County Medical Society, San Antonio.

Symposium on Renal Diseases

THE SYMPOSIUM ON renal diseases published in this issue was presented at the second annual meeting of the Charles T. Stone Society of Internal Medicine held in Galveston on June 8, 1963.

Formed in 1961 in honor of Dr. Charles Turner Stone, Sr., professor of medicine at the University of Texas Medical Branch from 1926 to 1958, the society held its first scientific meeting in 1962. Charter members of the organization were Department of Medicine faculty members and residents during the period when Dr. Stone was chairman of the department. Membership has been since extended to all physicians who have become members of the Department of Medicine since Dr. Stone's retirement, and to residents in medicine as they complete their residency training.

Goals of the society are simple but important: the progress of medicine in Texas.

—JOHN W. MIDDLETON, M.D., Galveston.

Doctors Urged To Write Congressmen

AS THE TIME draws near for the House Ways and Means Committee to act upon the King-Anderson bill, Texas physicians are urged by Dr. Robert Mayo Tenery, president of the Texas Medical Association, to write their Congressmen and tell them of the success of the positive programs in Texas for medical care of the aged.

Texas leads the nation in the percentage of aged who have one or more health insurance policies. A study by Belden Associates in Dallas showed that almost all of the aged who need and want help now are receiving it. Through the Kerr-Mills program, Texas now has an effective, economical program for 229,000 needy aged, representing more than 30 per cent of the entire 65-and-over population. Voters will have the opportunity to broaden the Kerr-Mills program in November to assist the 3 per cent who need help.

The compulsory program of federal medical care would use at least $69,000,000 in new taxes from Texas employees and their employers the first year to finance medical care for many people who are able to pay for it themselves.

Each physician, his family, and his friends are asked to write their Congressman to convey their views to the two Texas Congressmen on the House Ways and Means Committee, Rep. Clark W. Thompson, Galveston, and Rep. Bruce Alger, Dallas.

PROFESSOR'S MEMO:

Teach Science, Art, and "Something New"

MEDICAL EDUCATORS face a myriad of problems—getting qualified faculties and students, maintaining avant-gardism in a rapidly expanding field, financing medical education, providing better doctors and, by so doing, better patient care.

These are only a few of the "how to" questions they wrestle daily. Many questions worry the practicing physician. How dependent are our medical schools on federal subsidies? Schools turn out good scientists, but what about the art of medicine? Are our medical schools in Texas turning out only researchers and teachers?

The Texas Medical Association's Committee on Liaison with Medical Schools was set up to bridge the gap between the teaching physician and the practicing physician, to see how the medical organization could help Texas medical schools, and to present to educators some of the problems we see in practice.

First, what is a medical school? It has two basic responsibilities:

1. To teach young men and women the science of medicine and introduce them to the art of medicine. Medical educators in Texas believe that a deep concern for the well-being of our fellow man is part of the art of medicine. The science of medicine can be taught. In the final analysis, the art must be learned by practice. This can and does start in the clinical years in medical school.

2. To find new things to teach. It is neither science nor art of medicine. It is not research or practice. One cannot exist without the other.

This brings us to the often-maligned subject of research. Too often, research (so-called) is a continuation of an experience at the medical student level. It is lab-side teaching as compared to bedside teaching.

The deans and faculties of all medical schools in this country seek an answer to the question of a proper balance between research, teaching, and the practical application of the research findings. This is evidenced by a series of articles in *JAMA* entitled "Symposium on Medical Education," starting in the June 3, 1961, issue and continued in the July 13, 1963, issue in a series entitled "Variable Goals for the Medical School."

Dr. Robert H. Alway of Stanford University School of Medicine has this to say:

> "Our entering students are advised that their faculty has deliberately fostered research orientation in this school. To the most recent entering class, I said that:

Alway, R. H.: Care and Nurture of a Scholarly Faculty, JAMA 185:112 (July 13) 1963.

This is not to prepare you to do research. It is not to make it more likely that research will be done. It is to provide research orientation for each of you because we believe that this will better prepare you for continued medical scientific growth and development and adaptation, and that it should make less likely your medical scientific obsolescence. The research orientation which we would have you obtain is not to make of each of you an investigator, but rather to sustain your zest for learning, for reacting to the challenge of the unknown, and to foster questioning rather than a ready acceptance of dogma. You must apply the scientific method to the practice of medicine lest you degenerate into haphazard observation and uncritical data collection and evaluation, slipshod diagnoses, and an uncritical acceptance of your performance as well as that of your associates."

This is an excellent explanation of the need for research, or lab-side teaching, at the medical student level.

The question then arises, are medical schools justified in expanding their faculties and staffs in order to receive more grants? Texas schools expand their staffs and facilities to fulfill their second goal to find new things to teach.

Is a medical school ever *not* justified in expanding its staff and facilities for the ultimate goal we are all striving for—*better patient care?* If, in so doing, they do receive more grants for salaries, who is the winner? The patient, of course.

As to the federal grants for research, our present three schools are receiving about equal amounts for their various research projects.

Our medical schools must be so well-supported that the grants be only ancillary in nature. Private contributions, foundations, local government, state government, and lastly, the federal government must be involved in such a way that if any one of these fail, the school can continue toward its primary objective of *better patient care.*

Medical schools are indispensable to national survival and the entire population has a most vital interest in the quality and quantity of the doctors they produce.

—Russell L. Deter, M.D., El Paso.

Mental Illness Now Covered By Most Major Medical Group Plans

Ninety-nine per cent of those insured on a group basis under new major medical policies are protected to some extent against the costs of nervous and mental disorders, the Health Insurance Institute has reported.

"This represents a record high in the proportion of persons so protected since the first group health insurance survey was completed in 1960," said the Institute, "and there is a continuing trend toward insurance companies providing this type protection."

Contributing to this high percentage of protection is the fact that employee groups of 100 or more almost always are provided with some form of nervous or mental disorder coverage.

Major medical policies are held by more than 40,000,000 people, of whom an estimated 75 per cent are covered for nervous and mental illness while confined to the hospital.

personal

Opinions and Views

Battered Child Syndrome

WILFUL TRAUMA to children is little appreciated by physicians. The history is often misleading and a diagnosis of local trauma or systemic disease is too frequently entertained. Parents often go to a different doctor with each episode, so unless roentgenographic bone survey is ordered routinely, the underlying cause for the trauma will be overlooked.

The battered child syndrome occurs most often in children who are too young to tell what has happened. It is not uncommon to find subdural hematoma or ruptured organs. The most common fractures involve the long bones, skull, and ribs. The pathognomonic x-ray finding is multiple fractures in different stages of healing.

The importance of diagnosing this entity is related to the fact that the battered child syndrome is not a self-limited disease, and frequently requires removal of the victim from the home. In 1961, the American Academy of Pediatrics, meeting in Chicago, agreed that this syndrome was a major problem and should be included as a reportable disease.

From a psychiatric viewpoint, the physical battering is an extreme symptom of a common entity, the "scapegoat" child. The "scapegoat" child is only a symptom point in an intra-family aggressive pattern. As the name implies, the child in this particular situation has become a secondary target for the expression of the aggressive conflicts that exist within a family. The evolution of a particular member of a family to bear the brunt of these hostile conflicts seems to be a necessary escape valve for the family pattern to persevere. The survival of the family as a group is dependent upon this "scapegoat" child. The child is made to blame, in some way, for everything bad that goes on in a family. This sick homeostasis of family living is maintained through this particular safety valve. It is most surprising that, if there are other children in the family, they seem to make a good adjustment and they also find some sort of economy in having one child that can be blamed for everything. When an older child has inflicted the trauma, he is often a tool in the hand of the parent who directs the attack.

Classically, one parent (usually the mother but occasionally both parents) interacts with the child in an uncontrolled, murderous anger and with episodic, wild, psychotic-like rage. The other parent, usually the submissive father who plays a passive role in the marriage, becomes the ally of the aggressive, hostile mother in her attack upon the child. The selection of one particular child often is related to that child's having a defect or being unwanted.

The battered child is often willing to accept this particular role in the family be-

From the Department of Pediatrics, Baylor University College of Medicine, Houston.

Editor's Note: At its Jan. 18 meeting, the Texas Medical Association's Council on Medical Jurisprudence approved in principle proposed state legislation which would make mandatory the reporting by physicians of cases of physical abuse of children. Further, it would provide that anyone making a report under this proposed bill or participating in a judicial proceeding resulting therefrom would be immune from any liability, civil or criminal, that might otherwise be incurred or imposed. In order to present the problem to the public at a local level, the Council recommended that assistance be sought from the Woman's Auxiliary, the Texas Pediatric Society, the Texas Chapter of the American Academy of Pediatrics, and the Texas Academy of General Practice.

cause it gives him a spotlighted position from which he derives some neurotic gratification, and to which he seems amazingly able to adjust unless there has been a serious, crippling assault. It is only after we see such a child that we are able to identify the sick parent and offer therapy. The patient is the symptom of the family problem, and the child who is faced with emotional growth during a period in which he is subjected to this kind of experience must develop deep psychological problems as he grows older. Psychiatrists see many adults who are still reacting, in their behavior and adjustments, from themselves having been "scapegoat" children. The mother of a battered child has in some degree had the same type of childhood experience.

Some doctors are reluctant to testify in court, and this fact can be appreciated from past experiences. However, medical testimony is most beneficial to the jury. The problem that must be solved is one of how much trauma comes from normal discipline. Every parent has a right to discipline his child; the big disagreement centers around how much trauma the law will allow. In the battered child syndrome the parent has gone further than discipline and probably needs to answer to society for what he has done. In most communities, the proper procedure for the physician is to call the police juvenile department, where men of experience will determine whether legal action is necessary. Many times there is insufficient evidence to prosecute because the sole witness may be the father or mother who is precluded by rules of evidence from testifying in these cases. Therefore, information from the physician may be the only facts admissible in court.

One of the many problems in the medical world regarding the battered child concerns the ambivalent attitude of doctors—the pediatrician is angry with the parents and identifies with the child, and the psychiatrist feels sorry for the parents and gives the excuse that they are disturbed. As a result, it is difficult to take the child out of the home. Many psychiatrists feel that any parent who would wilfully injure a child must be schizophrenic, and therefore a potentially treatable individual who should not be prosecuted. It seems paradoxical, however, from the scanty literature on the subject, that there is an extremely low incidence of schizophrenia among these parents. Thus, it appears that some psychiatrists are too prone to defend the parents in this situation.

When the average doctor is told that his case represents one of a battered child, the standard answer is, "I would never have thought that this mother was capable of such an action." Many juvenile departments actually have found that neighbors, other members of the family, school teachers, or ministers are aware of the problem and will substantiate the findings. Removing the child from the home does not always solve the problem. Ideally, the entire family should be treated together and separation should be undertaken only if the target of the attack is showing a degeneration in his behavior pattern.

The recognition and classification of the syndrome of the battered child is in its infancy and deserves special attention by all who deal with the care of children. Federal legislation is now pending which will give immunity to the reporting physician in cases of unsuspected trauma in children.

—A. A. MINTZ, M.D., Houston.

Long Trail of Paper

To keep up with developments published in medical journals alone, a physician would have to read the equivalent of one book every hour. This is the calculation of Dr. Milford O. Rouse, Speaker of the AMA House of Delegates.

"Information now published on new medical developments is contained in some 8,000 medical journals and bulletins appearing annually," Dr. Rouse informed an audience of physicians at the Texas Medical Association Conference on Medical Service and Legislation, Jan. 18, in Austin, "About half of these are printed in the United States alone."

"Stretched end to end, their 11,000,000 pages would easily extend from AMA headquarters in Chicago all the way to Austin," he said.

The Physicians Benevolent Fund

Dr. May Owen, Past President of the Texas Medical Association, donated $2,500 in 1962 to establish a fund to help destitute physicians and their widows. Management of the fund was assigned to the Board of Trustees, and a committee was appointed to explore the best way to use this gift. It became apparent that the need was greater than anticipated, that the $2,500 would soon be exhausted if used to assist those who are presently in need of help, and that this donation should be a nucleus to be enlarged and invested so that the interest could be used to help those for which it was intended.

In recognition of Dr. Owen's generosity and great service to the Association, the Board of Trustees recommended that the fund be named after her; however, at her request, it has been called the Physicians Benevolent Fund. It will be used to help needy physicians, young or old, or their widows, who do not have enough income to maintain a subsistence standard of living and who are not able to work.

Knowing the generosity of Texas physicians, the committee is hopeful that voluntary contributions to this worthy cause will enable it to reach its goal. To date, the response has been most gratifying. A number of county medical societies have 100 per cent participation, but much more response is needed from the larger societies.

We cannot close our eyes to the fact that the need is here and now. There are physicians who can no longer practice their art and whose security for the future has been destroyed by misfortune or inflation, and there are doctors' widows who are living on a pittance. Our profession is founded upon service and concern for all mankind, and we must not overlook this obligation to our less fortunate colleagues.

Robert Mayo Terry

President
Texas Medical Association

Renal and Electrolyte Alterations In Respiratory Insufficiency

SYMPOSIUM

IN DISCUSSING the renal and electrolyte alterations of respiratory insufficiency, it is helpful to first review the current relevant theories of renal electrolyte metabolism; then to retrace, in the light of these theories, the sequence of events leading up to the findings in respiratory insufficiency; and lastly, to project the resultant understanding into a reasonable therapeutic regimen for the patient with respiratory insufficiency.

Since the primary alteration concerned is carbon dioxide retention, the effects of such retention provide a good starting point for study. Rector and Seldin[10] and others[16] have shown that an increased carbon dioxide pressure (pCO_2) causes an increased reabsorption of bicarbonate by the kidneys. In fact, an increased pCO_2 causes an increased bicarbonate tubular maximum. This was shown experimentally in dogs by elevating the plasma bicarbonate to a high level and maintaining it there with intravenous infusion of bicarbonate solution, while pCO_2 was stepwise increased by changing the carbon dioxide content of the animal's inspired air. When the resulting plasma and urine findings were plotted as bicarbonate reabsorbed per 100 cc. of glomerular filtrate against plasma pCO_2, a curvilinear increase in bicarbonate reabsorption was shown to occur with increasing pCO_2 (Fig. 1). No plateau was ever reached. However, these were all acute experiments, and no time was allowed for maximum effect from each carbon dioxide increase. Others have shown that with chronic exposure to a high fixed carbon dioxide level, the maximum effect is not reached for 24 to 48 hours.[14] Low levels of pCO_2 could not be studied because of severe hemolysis of the animal's blood, but extrapolation of the curve showed that there was little or no bicarbonate reabsorption at zero pCO_2, therefore carbon dioxide seems to be necessary for any bicarbonate reabsorption at all. In similar experiments, but with the pCO_2 held constant while plasma bicarbonate was stepwise increased (Fig. 2), Rector and Seldin showed clear-cut bicarbonate tubular maximums for different levels of pCO_2, with increased tubular maximums occurring for each increase in pCO_2.

How does this tremendous increase in bicarbonate reabsorption come about? All bicarbonate reabsorption seems to depend upon hydrogen ion secretion by the tubule cell, but where does this increase in hydrogen ion come from? Rector and Seldin reasoned that the hydrogen ion must initially become available in the tubular cell by the hydration of carbon dioxide to form carbonic acid (Fig. 3). Carbon dioxide apparently is the only source of hydrogen ion since without it there is little or no evidence of hydrogen ion secretion as determined by bicarbonate reabsorption. The hydrogen ion so produced by carbon dioxide hydration is then secreted into the tubule lumen to combine with the bicarbonate in the glomerular filtrate, producing carbonic acid in the tubule, which then decomposes to water and carbon dioxide. The latter then diffuses back into the cell and the cellular bicarbonate diffuses into the blood so that, in effect, bicarbonate is reabsorbed.

There are two possible mechanisms for hydrating the carbon dioxide; one by enzyme reaction, and the other, much slower one, by uncatalyzed reaction. The questions of which mechanism is most important, and to what extent and where it occurs in the nephron, spurred the researchers to repeat the aforementioned experiments after inhibiting carbonic anhydrase, the enzyme for hydrating carbon dioxide. The result was a constant reduction in bicarbonate reabsorption for all levels of pCO_2, the uncatalyzed hydration of carbon dioxide still increasing in curvilinear fashion with increasing pCO_2 (Fig. 1). The uncatalyzed portion therefore seems to be pCO_2-dependent, carbonic anhydrase-independent, and unsaturated (that is, it increases in rate for each increase in pCO_2); where-

Dr. Herring's work referred to herein was supported by National Institutes of Health Post Doctoral Fellowship Grant No. HPD-15,806.

Evidence indicates that continued respiratory insufficiency, whatever the cause, results in potassium and chloride depletion from renal and electrolyte changes. Replenishment of these electrolytes should be considered whether renal disease is or is not present.

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AUGUST R. REMMERS, M.D.;
HARRY E. SARLES, M.D., and
RAYMOND L. GREGORY, M.D.

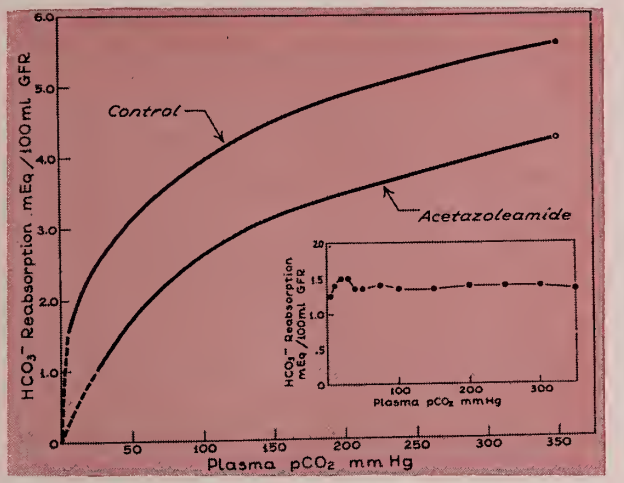

Fig. 1. Comparison of the relation between HCO_3-reabsorption and plasma pCO_2 with and without acetazolamide.*

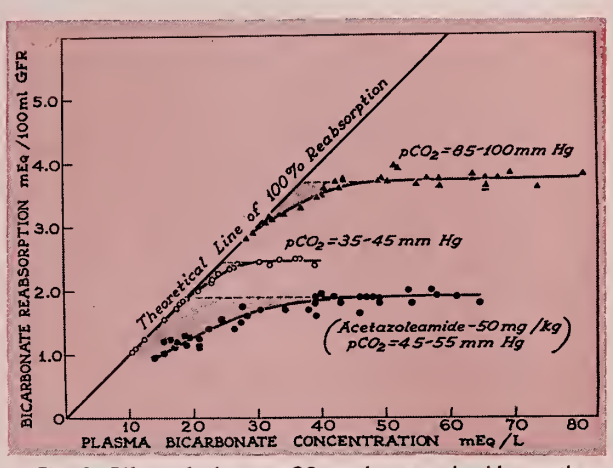

Fig. 2. Effect of plasma pCO_2 and acetazolamide on the relation between HCO_3-reabsorption and plasma HCO_3-concentration.*

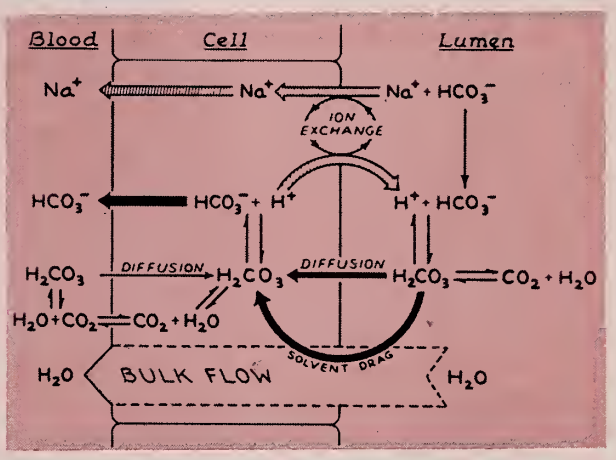

Fig. 3. Role of back-diffusion of H_2CO_3 in the supply of intracellular H+. By recycling into the cell, H_2CO_3 can furnish H+ to the exchange process, and to that extent reduce the requirements for CO_2 hydration. The pCO_2 will nevertheless determine the steady state concentration of H_2CO_3 at which the system operates.*

as the catalyzed portion is pCO_2-independent, depends upon the presence of carbonic anhydrase, and is functioning at the maximum rate at all times.

Rector and Seldin further demonstrated that uncatalyzed hydration of carbon dioxide using known rates of reaction at body temperature and normal pCO_2 cannot produce enough hydrogen ion to account for all the bicarbonate reabsorbed after carbonic anhydrase inhibition; therefore, it was theorized that there must be some back diffusion of carbonic acid from the tubule lumen to provide hydrogen ion for recycling. Even this does not occur at a rapid enough rate, so it was further theorized that in the part of the tubule where a large amount of fluid was being reabsorbed (that is, the proximal tubule, where 80 per cent of the glomerular filtrate is reabsorbed), carbonic acid would be "washed" back into the cell; therefore, the only place in the tubule where the recycling and uncatalyzed hydration could occur effectively would be in the proximal tubule (Fig. 3). Obviously, some carbonic acid would be lost to decomposition, carbon dioxide, and water in the tubular lumen. However, this small loss could easily be compensated for by the uncatalyzed hydration of carbon dioxide in the tubule cell. It is obvious that this recycling process would break down if the lumenal bicarbonate were to decrease, as it would if the lumenal pH decreased. Note that the only way to increase hydrogen ion secretion here is to increase the rate of uncatalyzed hydration of carbon dioxide; in other words, this is the only way to add to the recycling process. Since the rate

*J. Clin. Invest. 39:1706 (Nov.) 1960.

of production of hydrogen ion by uncatalyzed hydration is small and rate-limited, it takes time for the maximum effect of any increased carbon dioxide to be seen (24 to 48 hours).

What Is Now Known

In summary, then, we know these facts:

1. Increasing pCO_2 causes increased bicarbonate reabsorption and increased ability to reabsorb bicarbonate.
2. Bicarbonate reabsorption depends upon the secretion of a hydrogen ion into the tubule lumen.
3. This hydrogen ion comes from the hydration of CO_2, and only from this source, since there is no bicarbonate reabsorption without CO_2.
4. Carbon dioxide is hydrated both enzymatically and non-enzymatically.
5. The enzymatic process occurs mostly in the distal tubule and the non-enzymatic in the proximal tubule. This has been supported by microanalytic procedures on the dissected single nephron, which showed carbonic anhydrase to be present in much higher concentration in the distal tubule,[9] and by stop-flow and micro-puncture techniques which show that the pH of the distal tubule is too low to allow recycling.
6. In the proximal tubule, most of the hydrogen ion becomes available from recycled carbonic acid and cannot be secreted against a low lumenal pH.
7. The proximal tubule reabsorbs bicarbonate at a widely varying rate, depending upon the level of pCO_2 (this response to increased carbon dioxide is sluggish for reasons mentioned previously), whereas the distal tubule reabsorbs bicarbonate at a fixed rate regardless of pCO_2.

Other investigators have shown that bicarbonate and chloride seem to compete for reabsorption as the anion to accompany sodium ions across the tubule wall, but since bicarbonate reabsorption involves an active process, hydrogen ion secretion, it takes preference over the chloride ion; therefore, when bicarbonate reabsorption increases, chloride ion reabsorption decreases, and vice versa. Schwartz has shown in dogs that with chronic carbon dioxide exposure, the resultant increase in plasma bicarbonate was equal to the observed fall in plasma chloride ion[8] (Fig. 7). Chloride ion seems to be passively reabsorbed down an electromotive force gradient that is created by active sodium ion reabsorption. This gradient has been measured.[3-5, 15-17, 19, 22]

Stop-flow and micro-puncture techniques have uncovered many interesting facts about the kidney, such as the following:

1. There is little pH decrease in the proximal tubule, and considerable pH decrease in the distal tubule—this correlates well with the theories of Rector and Seldin since the proximal tubule recycling process for producing hydrogen ion could not work without bicarbonate in the lumen, and there can be little bicarbonate in an acid medium. Only in that part of the tubule where hydrogen ion is enzymatically produced can enough hydrogen ion be secreted to decrease pH.
2. Potassium is almost completely reabsorbed in the proximal tubule, and excreted, apparently in competition with hydrogen ion, in the distal tubule[2, 4, 11, 18, 20] (Fig. 4). Although it has not been proved experimentally, the rate of potassium excretion probably depends upon plasma concentration of potassium, so that increased plasma level causes a greater load on the proximal tubule and a greater spill of potassium.
3. There is active sodium reabsorption in the proximal tubule where it is exchanged for a hydrogen ion, and in the distal tubule where it is exchanged for a hydrogen or a potassium ion. The exact mechanisms for these phenomena are not clearly understood. Sodium ion reabsorption involves energy, which is aerobically derived since sodium ion reabsorption has been shown to increase or decrease in proportion to the per cent of oxygen saturation of the blood. However, these changes with oxygen per cent saturation are very small.[2, 4, 11, 17]

Calcium and magnesium ions seem to be reabsorbed in competition with each other in the proximal tubule and the first part of the distal tubule. The exact mechanism for this is unknown, but it is an active mechanism that is partly influenced by Parathormone.[1, 6, 13, 21] Phosphate ion is almost completely reabsorbed in the proximal tubule and secreted in the distal tubule in response to the plasma level of Parathormone. There appear to be no direct effects of increased pCO_2 on any of these mechanisms, except indirectly by its effect on plasma pH.

Extrarenal Phenomena

There are certain extrarenal phenomena

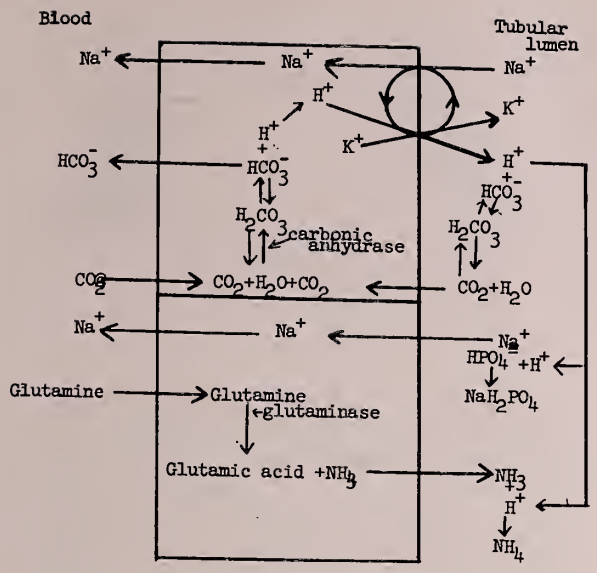

Fig. 4.

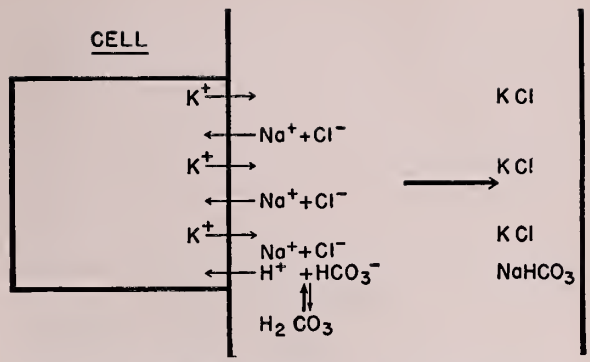

Fig. 5. The exchange of potassium for sodium and hydrogen ions between the intracellular and extracellular fluid.†

that are also pertinent to this discussion. These are the electrolyte shifts between the intra- and extracellular space as a result of changes in the extracellular space concentrations (Fig. 5). It has been conclusively demonstrated by analysis of fat-free, dry, solid preparations of tissue, and by micro-electrode estimation of intracellular pH that there is a shift of sodium and potassium ions with a change in extracellular pH, such that in extracellular alkalosis there is a shift of hydrogen and sodium ions out of the cell and a shift of potassium ions into the cells in a ratio of three potassium for every one hydrogen and two sodium ions. In extracellular acidosis, the hydrogen and sodium ions shift into, and the potassium ions shift out of, the cell. The precise mechanism, other than differences in concentrations, whereby this exchange occurs is not clearly understood.[4, 11, 17, 20]

With this admittedly meager understanding of normal electrolyte metabolism, let us now trace the development of respiratory acidosis and its recovery phase (Fig. 6). The first event is the retention of carbon dioxide, with the resultant increased pCO_2 which occurs very soon in all body activities, since carbon dioxide diffuses freely across all cell membranes. With the increased plasma pCO_2, there is a reduction of the bicarbonate/carbonic acid ratio, and therefore a pH decrease. This state of hydrogen ion excess, acidosis, will shift potassium ions out of the cells, leading to a rise in plasma potassium and its loss from the body by increased proximal tubule spill and increased distal tubule excretion. Serum sodium will decrease, but not significantly, and serum bicarbonate and chloride will remain normal for a while, changing only as the increased pCO_2 stimulates increased bicarbonate reabsorption. The latter results in a displacement of chloride ions from their reabsorption route across the tubule wall and their loss in the urine. Urinary pH will remain normal or decrease initially, depending upon how well the renal mechanisms compensate for the acidosis by bicarbonate reabsorption. Urinary chloride and potassium will increase. Urinary sodium will stay the same. The state of uncompensated acidosis, with high plasma pCO_2, rising plasma bicarbonate, and rising total plasma carbon dioxide persists for the first 24-36 hours of acute respiratory insufficiency (Fig. 8). There is probably little change in the plasma calcium and phosphate except for an increased ionized calci-

RESPIRATORY ACIDOSIS

	Onset		Recovery	
Plasma	initial	compensated	initial	compensated
PCO_2	↑	↑	↓	N
pH	↓	N	↑	N
HCO_3	N	↑	↑	N
Cl-	N	↓	↓	N
K+	↑	N	↓	N
Na	N	N	N	N
Urine				
pH	↓-N	N	↑	N
HCO_3-	↓	N	↑	N
Cl-	↑	N	↓	N
K+	↑	N	↓	N
Na+	N	N	N	N

Fig. 6.

†From Welt, L. G.: Clinical Disorders of Hydration and Acid Base Equilibrium, ed. 2, Boston, Little, Brown & Co., 1959.

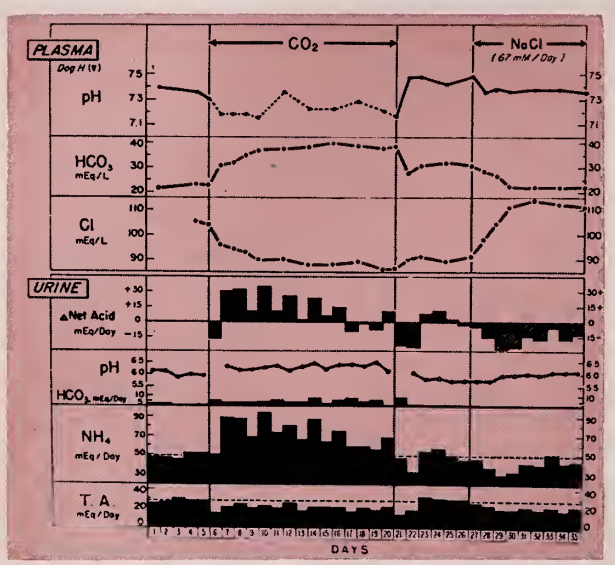

Fig. 7.‡

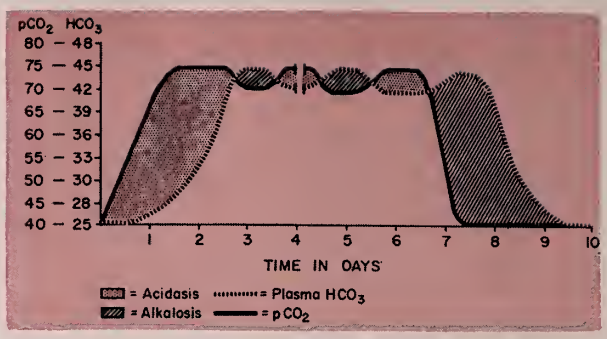

Fig. 8.

um fraction. Throughout the stage of compensated or partially compensated respiratory insufficiency, there is a continued loss of chloride and potassium ions in the urine (Fig. 7).

In the past few years, several detailed experimental and clinical studies have confirmed previous reports of paradoxical alkalosis in the recovery phase from chronic respiratory insufficiency.[8, 11, 12] This paradoxical state probably occurs for a brief period in every recovery phase but is not recognized. The mechanism of development of this paradoxical alkalosis, and some pointers for its prevention and/or treatment, can now be outlined in the light of the theories just discussed.

When respiratory insufficiency is corrected, and the pCO_2 falls to normal, and it does so very rapidly, the lag in the compensatory elimination of bicarbonate allows a definite alteration of the bicarbonate/carbonic acid ration, so that the pH increases (Fig. 8). This state of alkalosis may persist under two independent conditions:

1. The sudden increase in pH shifts potassium into the cells, leading to a hypokalemia and slight increase in sodium. (Note that the ratio of potassium to sodium in the shift is 3:2, therefore a decrease in serum potassium of 3 mEq. per liter would be accompanied by a sodium change of only 2 mEq. per liter (Fig. 5). With this shift out of the extracellular space, coupled with a total body deficit of potassium from the previous acidotic state, there is usually a noticeable change in serum potassium, and if inadequate potassium is provided in the diet, this deficit will persist. Since there is a deficit of potassium in the distal tubule, a hydrogen ion will be secreted for each sodium ion reabsorbed, with the resultant bicarbonate reabsorption and loss of, or failure to reabsorb, more chloride. Note that the recovery phase from respiratory acidosis has exactly the same electrolyte alterations as hypokalemic, hypochloremic alkalosis. One would expect, and it has been shown clinically, that a state of alkalosis will persist after correction of respiratory insufficiency until adequate potassium is added to the diet to make up the potassium deficit.

2. The great deficit in chloride brought about by an increased reabsorption of bicarbonate with its resultant chloride diuresis in the previous state of acidosis must now be corrected. This correction cannot take place if there is not enough chloride in the diet, even though urinary excretion of chloride may drop to extremely low levels. The fact that this deficit of chloride will lead to a persistence of the state of alkalosis and all its electrolyte alterations has been well documented by previous workers. Schwartz believed the chloride deficit was the major factor prolonging the alkalotic state, but Refsum and others have shown that although it is important, the alkalosis can be corrected without chloride correction if enough potassium is given.[11, 12, 15, 16]

The effect of the sudden development of this alkalotic state of calcium has not been well documented, but reported clinical observations of muscle twitching, convulsions, and sudden death in the recovery phase of respiratory insufficiency could be caused by the sudden decrease in calcium resulting from

‡From Polak, A., et al.: Effects of Chronic Hypercapnia on Electrolyte and Acid-Base Equilibrium; I. Adaptation. II. Recovery with Special Reference to the Influence of Chloride Intake, J. Clin. Lab. Invest. 40:1223, 1239 (July) 1961.

alkalosis. Since this phase is equivalent to hyperventilation in the normal subject, one might expect the reported acute changes in phosphate with hyperventilation to occur during recovery from respiratory insufficiency.

The patient with chronic lung disease and respiratory acidosis drifts into and out of the initial acidotic, compensatory, and initial recovery phase frequently in the course of his disease as his respiratory insufficiency worsens, stabilizes, and improves in cycles. Fig. 8 shows this schematically. The graph is not based on actual research data, and represents what the authors think must happen to explain the occasional report of paradoxical alkalosis in the untreated patient with chronic respiratory insufficiency. Note that the scale for plasma pCO_2 and plasma bicarbonate are so calibrated that pH is 7.4 when the lines showing pCO_2 and bicarbonate are superimposed. A state of acidosis exists when the pCO_2 line is above the bicarbonate line, and a state of alkalosis exists when the pCO_2 line is below the bicarbonate line.

To the authors' knowledge, there have been no adequate studies of the effects of the recovery phase from chronic CO_2 acidosis on renal blood flow and glomerular filtration rate. In our studies and in the study of others on acute changes, there have been no reproducible effects on these parameters.

Since there is normally a lag in diffusion of bicarbonate and hydrogen ions across the blood-brain barrier, while carbon dioxide diffuses very rapidly, one would expect the state of alkalosis to persist in the cerebrospinal fluid for some time even under ideal conditions of potassium and chloride replacement.[7]

Conclusion

The state of chronic respiratory acidosis leads to a total body deficit of potassium and a large deficit of chloride, both of which accentuate and cause a persistence of the state of alkalosis that obtains in the recovery phase. These deficits must be corrected, or at least these corrections must be initiated along with restoration of adequate ventilation, else the patient will remain in a state of alkalosis for an extended period of time. Ideally, the patient with marked respiratory acidosis should be ventilated slowly to prevent rapid, severe drops in pCO_2 with its resultant severe alkalosis.

Dr. Herring is a National Institutes of Health fellow in renal and electrolyte diseases at the University of Texas Medical Branch, Galveston. His co-authors* are instructors in internal medicine. This paper was presented to the Charles T. Stone Society of Internal Medicine in June, 1963.

Dr. Gregory is professor and chairman of the Department.

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Acute Diffuse Proliferative Glomerulonephritis
Pathogenesis

SYMPOSIUM

THE CURRENTLY accepted classification of glomerulonephritis is presented in Fig. 1. The development and acceptance of this classification represents the work of many investigators covering more than a century of medical history. Richard Bright first described glomerulonephritis in 1836. As clinicians observed the natural history of patients with glomerulonephritis, difficulty was encountered in placing patients in Bright's classification. Ellis, Evans, and Wilson first clearly divided patients with glomerulonephritis into two groups. Their work was monumental, covered a 20-year experience in The London Hospital, and was first published in 1942.[3] The acceptance of their work, however, has been slow because of the difficulty in retrospective analysis of cases of glomerulonephritis when viewed at postmortem examination. The percutaneous renal biopsy of the kidney has greatly facilitated the clinicopathologic correlation of glomerulonephritis. Serial renal biopsy studies have substantiated the work of Ellis, Evans, and Wilson.

Glomerulonephritis can be of three types: diffuse proliferative (Ellis 1), diffuse membranous (Ellis 2), and acute focal. This discussion will be limited to the pathogenesis of diffuse proliferative glomerulonephritis.

Renal Biopsy

The renal service at the University of Texas Medical Center has had considerable experience in the use of the percutaneous renal biopsy in patients with proliferative glomerulonephritis. We are now using the Menghini renal biopsy needle for most biopsies and rarely have to resort to the use of the more classical renal biopsy needle, the Franklin modification of the Vim-Silverman needle. The service has had no mortality associated with renal biopsy. In the last 160 renal biopsies, there has been virtual absence of morbidity from the Menghini renal biopsy. Only two patients have had gross hematuria persisting longer than one day (three and five days, respectively), and only two patients had enough pain to require codeine following the procedure. No patient required a blood transfusion or operative intervention. In no instance has pyelonephritis been clinically exacerbated nor has there been any infection in the needle tract. This experience which suggests lower morbidity following the use of the Menghini needle confirms a report by Kerr.[5] We attribute this lower morbidity rate to the following characteristics of the Menghini renal biopsy:

1. The small caliber of the Menghini needle; it has a #17 gauge compared to the #15 gauge of the Vim-Silverman needle.
2. The speed with which the biopsy is performed; the patient is required to hold his breath only one second.
3. The sharper cutting edge of the Menghini needle.

Etiology

The photomicrographs (Figs. 2-5) demonstrate the histologic features of diffuse proliferative glomerulonephritis at various stages as seen in this series of patients. These histologic features should be kept in mind during the following discussion of the etiology of the disease.

The Organism.—The initial bout of Ellis type 1 glomerulonephritis is the result of an antigen-antibody reaction in the glomerulus secondary to an infection elsewhere in the body, usually of streptococcal origin. Eighty-four per cent of cases have a preceding in-

In a discussion of the present knowledge of the pathogenesis of diffuse proliferative glomerulonephritis, the authors point out the value of serial percutaneous renal biopsies. They also draw other scientific conclusions regarding the etiology of the disease.

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MAURICE E. HERRING, M.D.

fection due to group A beta hemolytic streptococcus, serologic type 12. Occasionally types 1, 4, and 49 may be nephritogenic.[8] Any bacterial and some viral infections can initiate an acute exacerbation of acute diffuse proliferative glomerulonephritis in patients who have chronic glomerulonephritis.

Serologic type 12 organisms have several unique properties:

1. They may have no alpha or beta hemolysis on culture plates so they may be overlooked on a routine bacteriologic study.

2. They may fail to produce streptolysin S and O antigens so that patients infected may fail to have a rise in appropriate antibody titers.

3. When these organisms are phagocytized by a leukocyte, within 30 seconds the leukocyte begins to disintegrate. This "leukotoxic" factor parallels the production of diphosphopyridine nucleotidase by type 12 organisms.[1]

4. Type 12 organisms elaborate a mucopolysaccharide antigen similar to the mucopolysaccharide in the glomerular basement membrane.[7] When the organism is phagocytized by leukocytes, the lysis of the bacteria and the leukocyte may facilitate the systemic release of a mucopolysaccharide an-

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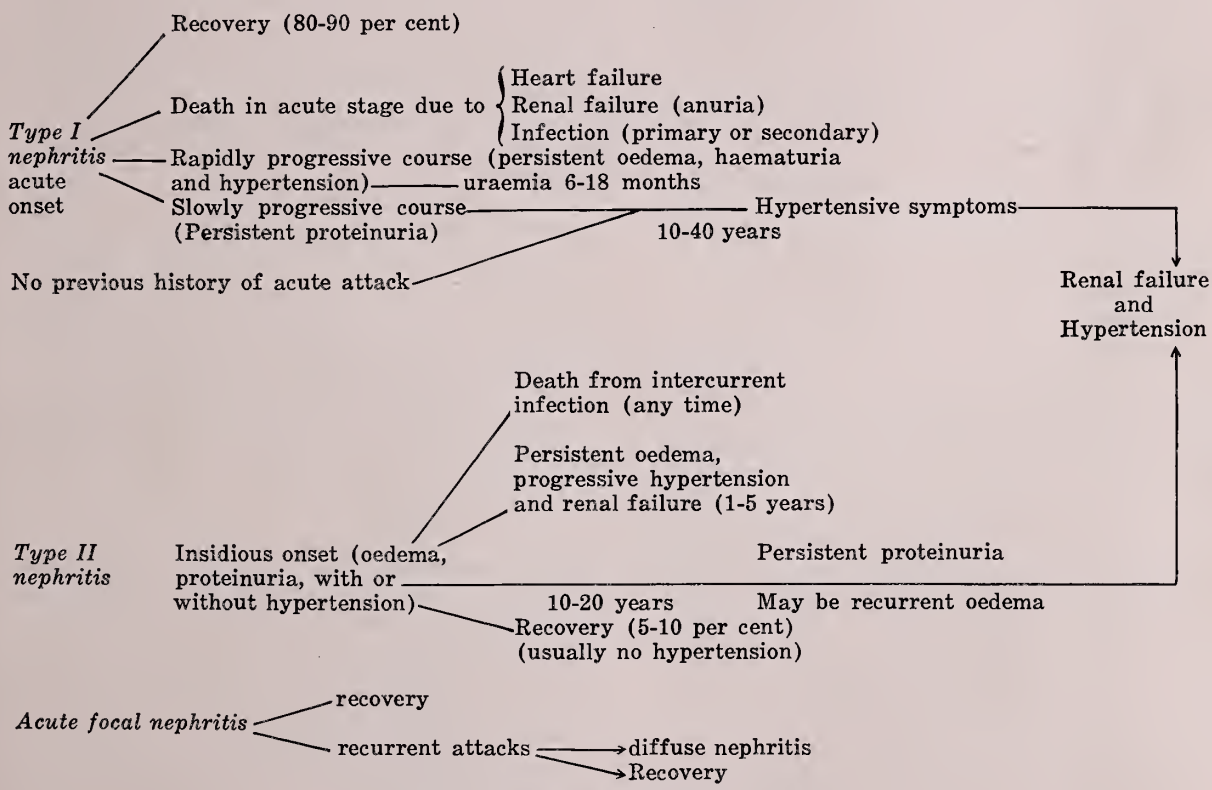


Fig. 1.* Glomerulonephritis. Clinical Courses.

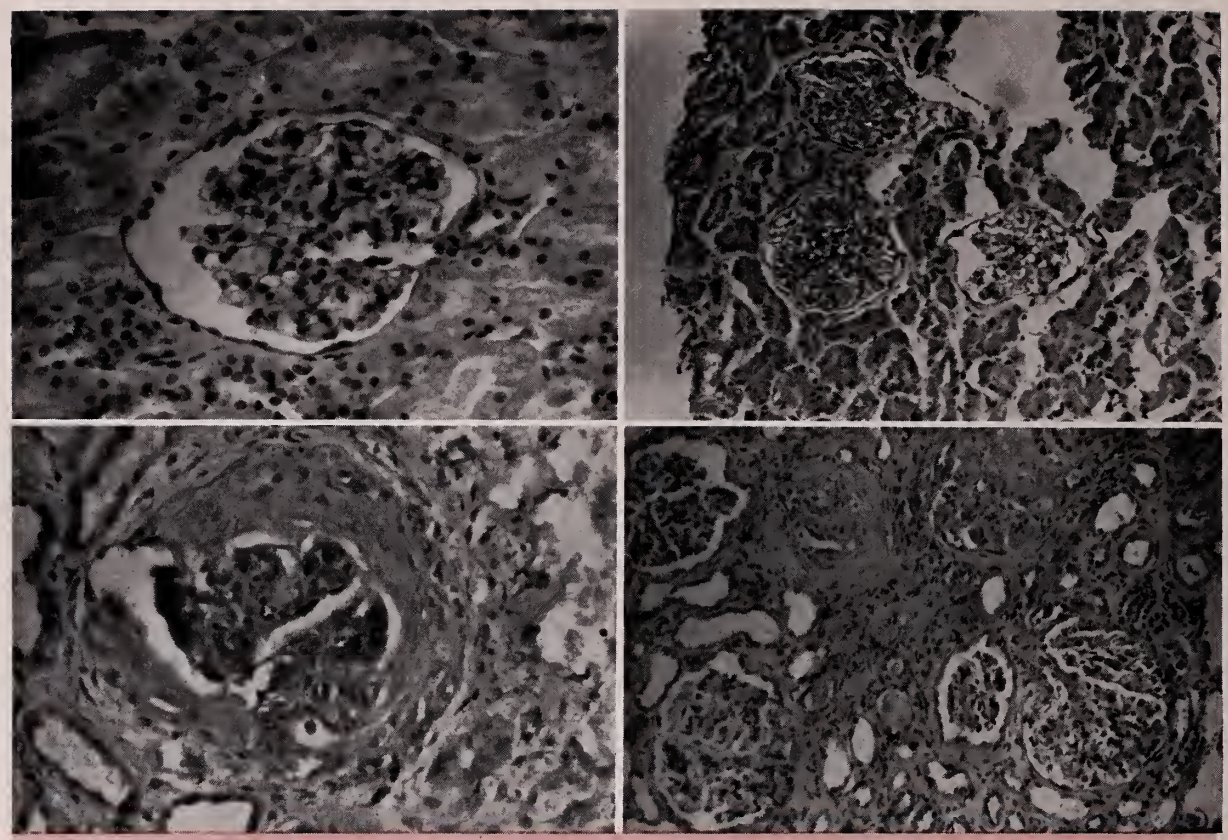


Fig. 2. This is a normal glomerulus using the periadic acid-Schiff stain. Note the normal thickness af the basement membrane, the narmal number af epithelial and endothelial cells, and the normal size af the glamerulus.

Fig. 3. This section demanstrates acute diffuse proliferative glamerulanephritis. Nate the large size af the glameruli, and the striking increase in cellularity of the glamerulus.

Fig. 4. This picture is an example af subacute proliferative glamerulanephritis. There is considerable crescent farmatian and cantinuing cellular praliferatian.

Fig. 5. This tissue section is an example of chranic praliferative glomerulonephritis. There are adhesians between the glomerulus and Bawman's capsule, cansiderable cellular praliferation, naticeable glamerular stalk thickening, and arterialar thickening. An absolete glomerulus is present alang with tubular atrophy and interstitial fibrosis in the regian af the tubules which ance supplied the hyalinized glomeruli.

tigen so similar to the glomerular basement membrane that the antibodies produced indiscriminantly injure the glomerulus.

The Host.—The average latent period for acute glomerulonephritis is 10 days, whereas the average latent period for rheumatic fever is 18 days. The attack rates for glomerulonephritis with subsequent infections with nephritogenic streptococci decrease sharply, whereas the attack rates increase in rheumatic fever with subsequent group A streptococcal infections. This phenomenon is presumably due to the development of type-specific antibodies following an infection with nephritogenic streptococci; the antibodies lead to protection against recurrent attacks. In various epidemics the attack rate has varied from 2 to 33 per cent.[8]

Experimental studies in animals have shown repeatedly that animals given an extremely deficient protein diet are resistant to the development of nephrotoxic nephritis.[7] This finding has led to the confusing clinical question as to whether or not the protein intake should be restricted in patients with acute proliferative glomerulonephritis.

Experimental glomerulonephritis in animals has been accomplished by three different means. Representative animal experiments of each type are described:

1. Post-streptococcal nephritis has been demonstrated in rabbits which were given a type 12 streptococcal infection of the paranasal sinuses. Several weeks later the intravenous injection of the polysaccharide fraction of nephritogenic streptococci was followed by the development of acute proliferative glomerulonephritis.[1, 7]

2. Heterologous protein nephritis has also been demonstrated. Lesions resembling acute,

subacute, and chronic diffuse proliferative glomerulonephritis were produced in animals by injections of horse serum, albumin, and gamma globulin from a different animal species. The critical factor apparently is the amount of antibody formed against the foreign protein injected. If more antibody is produced than antigen given, the picture of acute diffuse proliferative glomerulonephritis is produced. If less antibody is formed than antigen given, the animal develops chronic glomerulonephritis. By fluorescent antibody techniques, the antigen and antibody complexes plus gamma globulin of the host were demonstrable in the glomerular capillary wall during the development of experimental glomerulonephritis.[7]

3. Nephrotoxic glomerulonephritis was produced in another experiment. Antigens from homogenized whole kidney, glomeruli, and basement membranes were injected into a different animal species. Serum was then harvested and injected into the original species. The renal lesion is presumably produced by the union of anti-kidney antibody with homologous antigen in the kidney of the host. Flourescent tagging studies of this phenomenon have demonstrated the localization of the antibody serum on the glomerular basement membrane.

By varying the dose and source of the antigen (whole kidney, glomeruli, basement membrane), the lesions of both proliferative and membranous glomerulonephritis can be produced. Iodine 131-tagged nephrotoxic serum was localized in the glomeruli within two and one-half minutes after the injection. The blood is completely cleared of I^{131}-tagged antibodies within 18 minutes.[7]

Serum complement activity must also be discussed. Complement is a factor which consists of a mixture of several serum proteins. It combines with most specific antigen-antibody complexes, and this furnishes the basis for the complement fixation reaction. Complement is present in normal serum and is not increased by immunization; therefore, it is not a part of the specific immune mechanism *per se*.

Each complement component may be inactivated by appropriate serum treatment. Table 1 demonstrates some of the properties of the four complement components.[2]

In man and animals, 24 to 48 hours before the onset of hematuria, the serum complement activity decreases to a level of 20 per cent normal activity. This persists until a few days before the urine sediment becomes normal. In animals, only those animals with a reduced serum complement level will manifest glomerulonephritis. In man, if the serum complement level is normal, the diagnosis of acute proliferative glomerulonephritis should be seriously questioned. If the serum complement level fails to rise during the four weeks after the onset of the acute attack, the prognosis is grave.[6] In the few patients who go on to subacute and chronic proliferative glomerulonephritis, the serum complement level gradually rises to near normal levels.[4]

In diffuse membranous glomerulonephritis the serum complement level is decreased in 80 per cent of cases. The complement level parallels the clinical course in that it rises and falls as there are clinical remissions and exacerbations.[6]

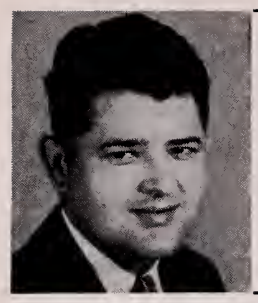
Low levels of serum complement activity are also seen in systemic lupus erythematosus with and without renal involvement. Steroid therapy if given in sufficient dosage levels to these patients will return the serum complement levels to normal. In periarteritis nodosa, serum sickness, trichinosis, and following heavy alcohol ingestion the serum complement level is regularly reduced.[4, 6]

Conclusions

The present pathogenetic concept of diffuse proliferative glomerulonephritis sug-

TABLE 1.—*Properties of the Four Complement Components.*

Complement Fraction	Synonym	Electrophoretic Mobility	Water Soluble	Destroyed by Heating 30 Min. at 56° C.	Inactivated by Cobra Venom or Yeast	Inactivated by Ammonia or Primary Amines
C'_1	mid-piece	Serum globulin between gamma and beta globulins	—	+		
C'_2	end-piece	$Alpha_2$	+	+		
C'_3		??	—	—	+	—
C'_4		$Alpha_2$	+	—	—	+



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gests that the disease is due to structural damage of the glomerulus arising from the union of organ-specific antibodies with elements of the nephron. This antibody is formed in response to the release of nephritogenic streptococcal mucopolysaccharide antigen into the circulation. The antibody elaborated indiscriminately injures the host's glomeruli. Complement is absorbed during the process in large quantities in the acute phase and in decreasing amounts as the cellular disorganization becomes less diffuse and less intense.

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Foreign MD's To Become "Pen Pals" of Americans

Americans who are interested in medicine, nursing, and dentistry will be able to correspond with people with the same interests in other parts of the world as part of the largest international pen pal project ever conducted.

Called the "International Penfriend Program," the project's goal is to promote a better understanding among peoples of the world through person to person correspondence. A million or more persons of all nations are expected to be matched by electronic computer in the next two years.

The project will be conducted by a national pen manufacturer at the New York World's Fair, which opens April 22.

Names to be fed into the computer for matching at the Fair currently are being gathered throughout the world. Registrants can select the language in which they wish to correspond, the area of the world to which they desire to write, and can choose from 389 specific subjects of interest about which they would like to write. They will then receive from the computer the name and address of their overseas penfriend.

The Idiopathic Nephrotic Syndrome: Pathophysiology and Treatment

LUTHER B. TRAVIS, M.D.; WARREN F. DODGE, M.D.; and ROBERT A. TURNER, M.S.

SYMPOSIUM

THE NEPHROTIC syndrome is the metabolic consequence of massive urinary loss of protein, and may be the result of a wide variety of diseases of the renal glomerulus. The syndrome is defined by hyperlipemia, massive proteinuria, and definite hypoproteinemia producing generalized edema.

In the adult, it is most frequently seen in its "secondary" form and may occur during the course of such systemic diseases[13, 15, 19, 22, 25] as diabetes mellitus, disseminated lupus erythematosus, constrictive pericarditis, primary or secondary amyloidosis, and syphilis. Chronic glomerulonephritis and renal vein thrombosis are the most common renal diseases which may be associated with the development of the nephrotic syndrome. An ever increasing number of chemical or toxic agents (trimethadione, paramethadione, heavy metals, penicillin, probenecid, aminonucleoside, poison oak, bee stings) also are capable of inducing a nephrotic syndome.[2, 6, 23, 28, 33]

The syndrome may also occur as a "primary" renal disease of unknown etiology, and this form includes most childhood cases. Although the nephrotic syndrome is more commonly of a secondary nature in the adult, approximately 25 per cent of the adult cases are of the "primary" or unknown etiology type.[1]

The latter type of nephrotic syndrome has been referred to by a number of terms: idiopathic nephrotic syndrome, childhood nephrosis, lipoid or lipid nephrosis, membranous glomerulonephritis, and Ellis type 2 glomerulonephritis.[4] The first term (idiopathic nephrotic syndrome) is probably more acceptable and is the one preferred by the authors since it does not classify this as a specific disease entity nor does it presuppose a common etiology.

It is the purpose of this report to review some of the aspects of the pathogenesis and pathophysiology of the idiopathic nephrotic syndrome and to present an outline of the current management.

Etiologic Considerations

As the name suggests, a definite etiologic agent has not been found in cases of the idiopathic nephrotic syndrome. Since the clinical disease is often first noted following an upper respiratory infection (60 per cent of the cases), extensive investigations have been directed toward the possibility of a common infectious process. Preceding streptococcal illness, well established as the etiologic agent responsible for acute glomerulonephritis, accounts for only a few cases of idiopathic nephrosis. Attempts to incriminate a viral etiology have been unsuccessful. Indeed, because of the high incidence of upper respiratory infections in the most commonly affected age group (two to six years), these are probably coincidental.

The bulk of present evidence suggests that the idiopathic nephrotic syndrome occurs as a consequence of a hyperimmune response

Hyperimmune response of the renal glomerulus to an agent or agents yet unidentified probably causes the idiopathic nephrotic syndrome of childhood. Clinical disease is produced by a massive leakage of plasma proteins through the altered glomerular capillaries.

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in which the renal glomerulus is the target area. The finding in nephrotic children of decreased serum complement,[7, 17, 18] elevated hemagglutinin[16] and precipitin antibody titers,[20] and the localization of fluorescent-labeled gamma globulin to the glomerular basement membrane[8] support the concept of a hyperimmune or hypersensitivity type reaction.

Various experimental studies have added considerable support to the immunologic theory;[11, 12, 24] for example, guinea pig kidney extracts when injected into a rabbit produce circulating antikidney antibodies. If some of the rabbit's hyperimmune sera is then injected into a guinea pig, cellular changes occur within the glomerulus and the animal promptly develops a typical nephrotic syndrome. Similarly, if guinea pig kidney is incubated with Freund's adjuvant and injected into a guinea pig, a mild nephrotic syndrome usually ensues. Associated with the experimental disease, there is a rise in precipitin antibodies and a fall in serum complement.

Also worthy of mention is the possibility that the idiopathic nephrotic syndrome represents an unusual form of metabolic or familial disorder. Its occurrence in more than one member of a family group is not uncommon and has been noted in almost every large series. Neither is it uncommon to find several interrelated hypersensitivity diseases within a single family unit. The authors recently have observed a two-year-old child with the classical picture of the nephrotic syndrome. This patient's older brother had anaphylactoid purpura with nephritis, one sister had bronchial asthma, and another had severe and classical milk hypersensitivity. The father of this group had a history of Bright's disease as a child.

Pathologic Considerations

The histologic changes which occur within the glomerulus in the idiopathic nephrotic syndrome are highly variable. This is partially dependent on the duration of the disease process but it is likely that this variation may also be related to different etiologic circumstances.

The earliest and most consistent lesion is best demonstrated by electron microscopy and consists of loss of the interdigitations (foot processes) of the epithelial cell cytoplasm.[5, 31] Although this fusion of the foot processes is most often diffuse and homogenous, it is sometimes focal with normal interdigitations appearing in close proximity to fused ones. Fig. 1 illustrates this fusion and smudging of the epithelial cell foot processes in a four-year-old child with the idiopathic nephrotic syndrome of eight months' duration. Present evidence suggests that this lesion is the result, rather than the cause, of the massive filtration of plasma proteins.[30] The alterations of the foot processes

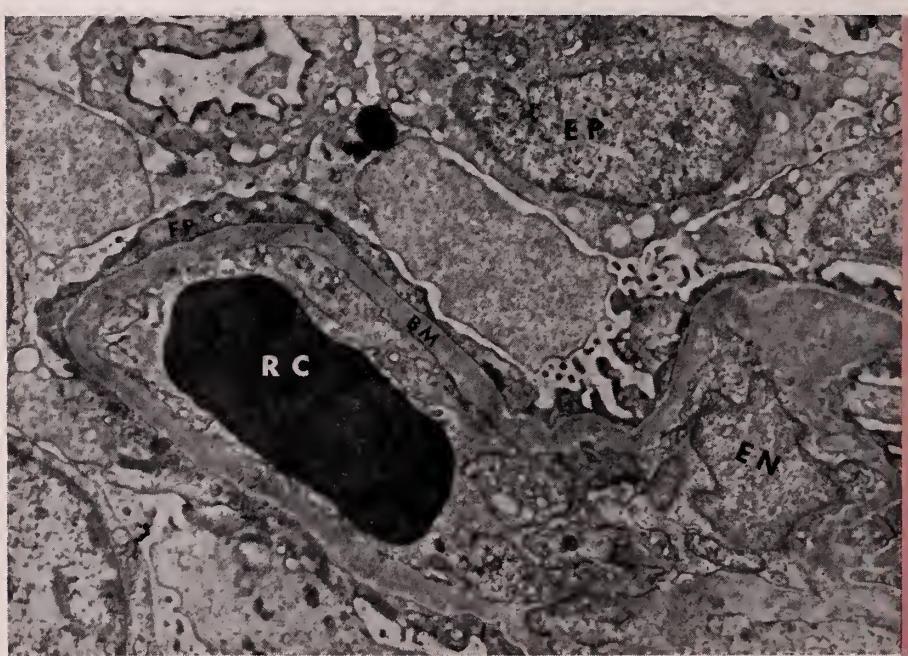

Fig. 1. Electron microscopy specimen of a glomerular capillary in a four-year-old child with the nephrotic syndrome. A large red blood cell (RC) is present within the capillary lumen. The foot processes (FP) of the epithelial cell (EP) are fused and reveal little evidence of the normal interdigitations. The cytoplasm of the endothelial cell (EN) appears swollen and some vacuolization is present. The basement membrane (BM) is of relatively normal thickness. X 4620

Fig. 2. This section from a three-year-old nephrotic child reveals minimal diffuse thickening of the basement membrane associated with nodular irregularity (arrows). The epithelial foot processes are again fused and smudged. X 2970

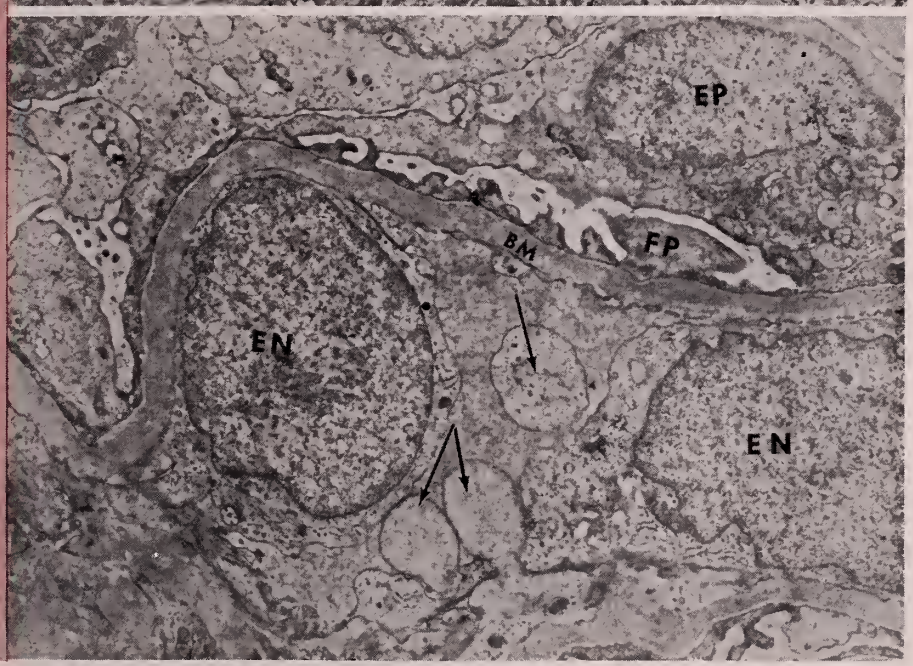

Fig. 3. Electron microscopy specimen of a portion of a glomerular capillary. The two large endothelial cell nuclei and their associated cytoplasm almost completely occlude the capillary lumen. In three areas (arrows) there is a swelling and ballooning of the cytoplasm. The basement membrane is moderately thickened and the foot processes reveal some fusion. X 5940

are reversible, and return to normalcy may be observed following either spontaneous or steroid-induced remissions.[5, 30, 31]

Considerable controversy exists concerning the status of the basement membrane in the idiopathic nephrotic syndrome, but most investigators believe that there is usually mild to moderate thickening of the basement membrane.[5, 30, 31] In children, this is most often manifest by a nodular irregularity occurring on the endothelial surface and frequently associated with mild endothelial cell proliferation. These findings are well demonstrated in Fig. 2, a section from a three-year-old with the nephrotic syndrome of 15 months' duration. In contrast, present evidence suggests that adults with the idiopathic nephrotic syndrome acquire their basement membrane thickening by the addition of a material on the epithelial surface.[5]

Additional lesions which are frequently seen in the idiopathic nephrotic syndrome consist of vacuolization of the epithelial cytoplasm and swelling and ballooning of the cytoplasm of the endothelial cell (Fig. 3).

Since the capillary wall is composed of the endothelial cytoplasm, the true basement membrane (lamina densa), and the epithelial foot processes, and since the usual resolution by light microscopy is not sufficient to distinguish between them, a thickening of any one might be interpreted as "basement membrane thickening." In the appraisal of light microscopy specimens, it is therefore important to minimize this potential error by (1) examination of peripheral capillary loops which are some distance removed from cell nuclei, and (2) application of a mucopolysaccharide stain (periodic acid-Schiff) since the lamina densa stains much more intensely than do the other component parts.

The authors have used a modification of Ellis' classification[4] and have found it exceedingly useful in the evaluation of light microscopy sections obtained from children with the nephrotic syndrome. This classification (Table 1) divides the possible histologic changes into two major groups—those manifesting primarily a proliferative lesion (type 1) and those showing predominantly membranous changes (type 2).

The majority of children with the idiopathic nephrotic syndrome reveal type 2 histologic changes. Fig. 4 is a typical renal biopsy specimen obtained from a three-and-one-half-year-old child with the nephrotic syndrome of two months' duration. The glomeruli reveal minimal, if any, alterations from normal and would therefore be classified as a type 2 A. This is the usual finding in a child with the nephrotic syndrome of less than four months' duration, and generally denotes a favorable response to steroids and a good prognosis.

TABLE 1.—*Classification of Diffuse Glomerulonephritis.*

Ellis Type 1	Glomerulonephritis:
	Group A—Acute proliferative disease
	Group B—Active disease process, in repair
	Group C—Inactive process, repair largely complete
Ellis Type 2	Glomerulonephritis (requires a clinical diagnosis of the nephrotic syndrome):
	Group A—Normal glomeruli by light microscopy
	Group B—"Membranous" glomerular changes (thickening of the capillary basement membrane)
	Group C—"Membrano-lobular" glomerular changes (thickening of the basement membrane and increased lobulation)

When histologic specimens are examined later in the course of patients with an "active" nephrotic syndrome, mild to moderate basement membrane thickening is generally noted. Fig. 5 is a representative specimen obtained from a two-and-one-half-year-old child with the nephrotic syndrome of longer duration (seven months), and demonstrates moderate diffuse thickening of the capillary wall (type 2 B). An example of type 2 C (membrano-lobular glomerulonephritis) is noted in Fig. 6 and was obtained from an 11-year-old child with a seven-year history of the nephrotic syndrome. The children whose

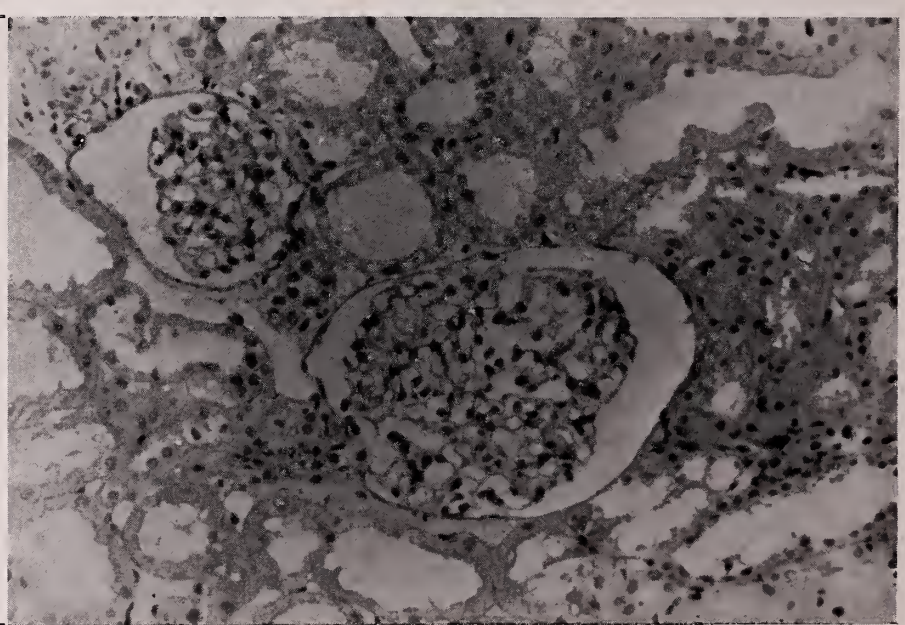

Fig. 4. Light microscopy specimen (periodic acid-Schiff stain) of two glomeruli from a three-and-one-half-year-old nephrotic patient. The capillary loops are open and the "basement membrane" is of normal thickness. This biopsy specimen conforms to an Ellis type 2 A (normal glomerulus).

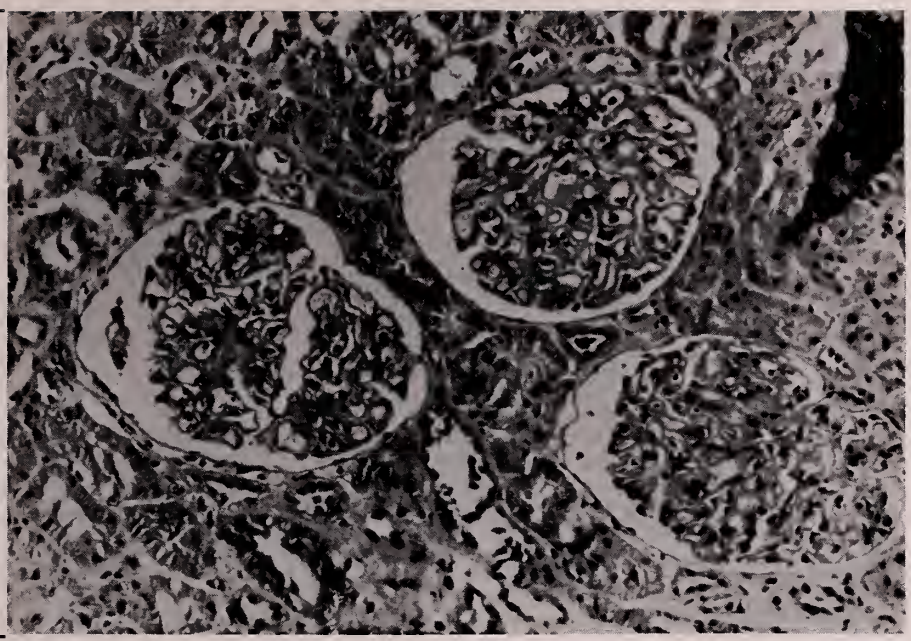

Fig. 5. Renal biopsy specimen from a two-and-one-half-year-old child reveals a diffuse thickening of the capillary basement membrane (type 2 B).

kidneys reveal these latter two changes generally have a poorer prognosis and respond less well to glucocorticoid therapy. Most adult patients with the idiopathic nephrotic syndrome show lesions classified as 2 B or 2 C.

Occasionally, children with the classical clinical picture of the idiopathic nephrotic syndrome demonstrate renal lesions which are typically of a proliferative type (Ellis type 1). The histologic specimen in Fig. 7 is from a seven-year-old child with an illness of three months' duration. The glomeruli reveal extensive proliferation, hyperplasia of the lobular stalk, and broad adhesions. In the authors' experience, these children generally do not respond to steroid therapy and their prognosis is guarded.

Physiologic Considerations

The physiologic alterations consequent to the massive urinary loss of protein are responsible for the majority of the clinical observations. Although the mechanism for the production of these alterations is still open to considerable speculation, Fig. 8 depicts the

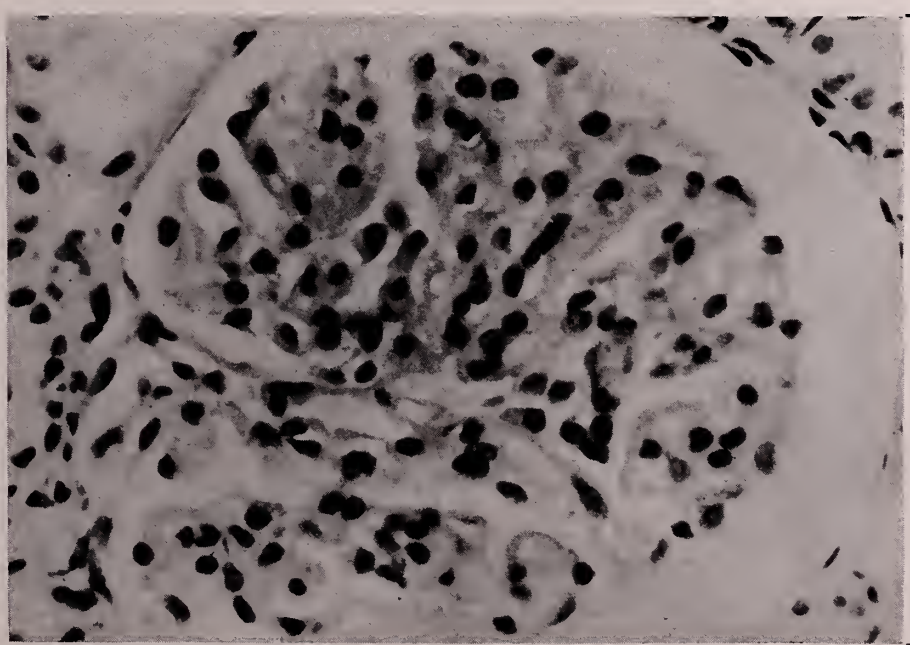

Fig. 6. High magnification (X 713) of a glomerulus shows a mild proliferation of the tuft and evidence of a diffuse thickening of the basement membrane. There is slight evidence of beginning separation of the tuft lobules (early type 2 C).

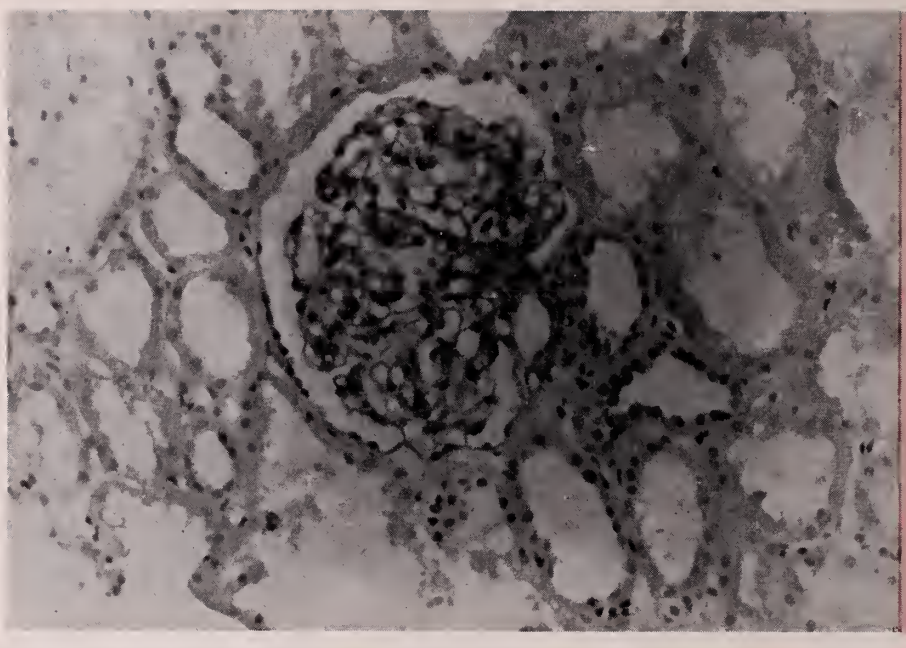

Fig. 7. Representative glomerulus from a seven-year-old child with the recent onset of a typical idiopathic nephrotic syndrome. The glomerulus is hypercellular and shows hyperplastic changes of the lobular stalk. A definite adhesion is noted at six o'clock. The macula densa is also hyperplastic in this non-hypertensive subject. The changes conform to an Ellis type 1 lesion.

most widely accepted concepts for the pathogenesis of edema in the nephrotic syndrome.

Following the initial immunologic insult, there occur alterations in the wall of the glomerular capillary which produce a massive "leakage" of protein into the usually protein-poor filtrate. This is predominantly a loss of the proteins of small molecular weight (albumin, gamma globulin),[10] and the electrophoretic pattern of these urinary proteins (Fig. 9) bears close resemblance to that of normal serum. This loss, plus the in-

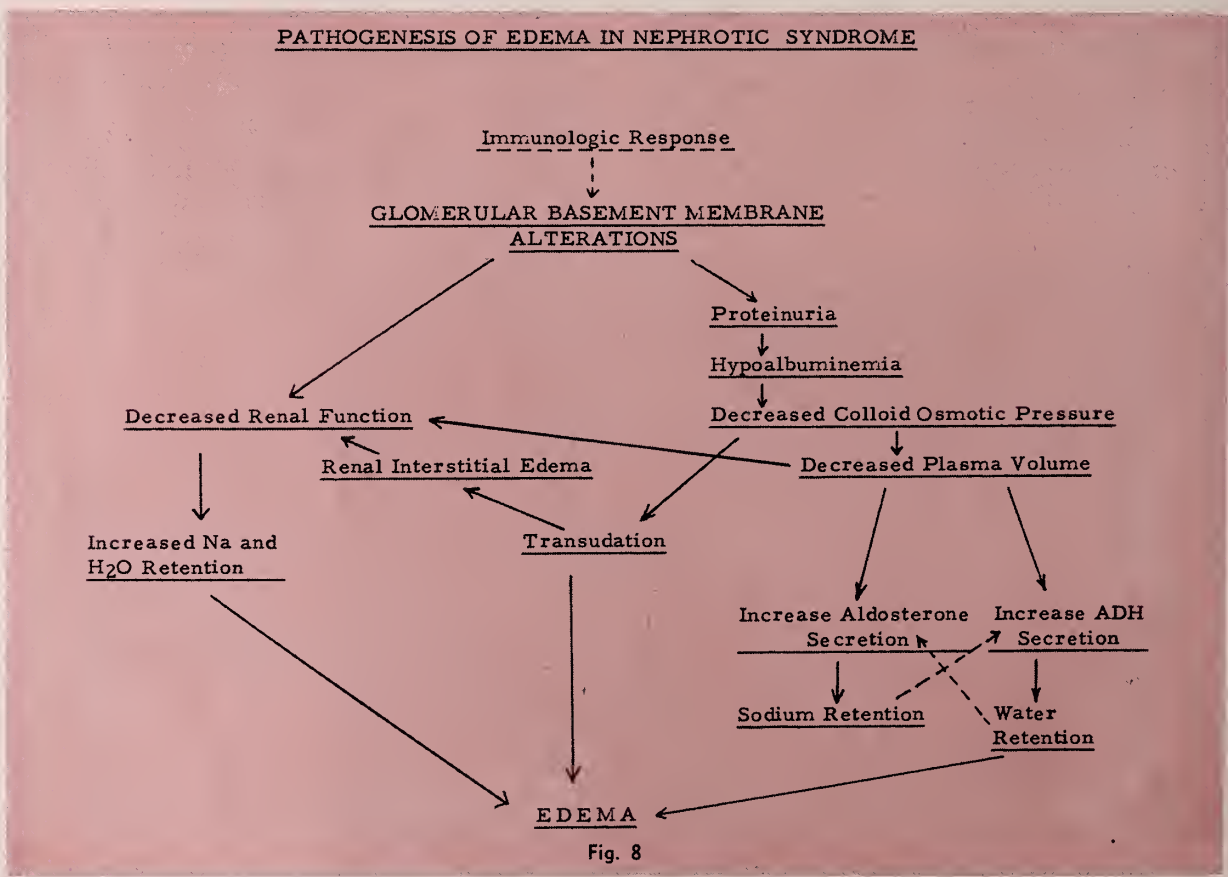

Fig. 8

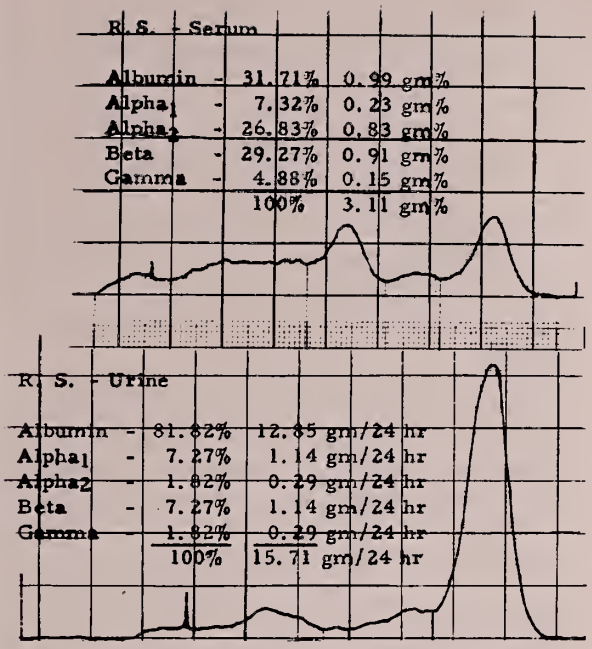
R. S. - Serum
Albumin - 31.71% 0.99 gm%
Alpha1 - 7.32% 0.23 gm%
Alpha2 - 26.83% 0.83 gm%
Beta - 29.27% 0.91 gm%
Gamma - 4.88% 0.15 gm%
100% 3.11 gm%
R. S. - Urine
Albumin - 81.82% 12.85 gm/24 hr
Alpha1 - 7.27% 1.14 gm/24 hr
Alpha2 - 1.82% 0.29 gm/24 hr
Beta - 7.27% 1.14 gm/24 hr
Gamma - 1.82% 0.29 gm/24 hr
100% 15.71 gm/24 hr

Fig. 9. Electrophoretic pattern of serum and urine proteins in a nephrotic child. The distribution of the urinary proteins bears striking resemblance to that of normal serum proteins. The serum pattern is characteristic in that there is excessive depression of albumin with an associated increase in the alpha$_2$-globulin fraction.

creased protein catabolism observed by Gitlin and associates,[9] leads to a lowering of the serum protein and a decrease in plasma colloidal osmotic pressure. As a consequence there is a net transfer of fluid into the interstitial spaces and a decrease in the effective plasma volume.[29]

Both the reduced plasma volume and the increase in renal interstitial pressure tend to reduce renal function and thereby reduce sodium and water excretion. Further retention of salt and water occurs as the body's normal homeostatic mechanisms (increased secretion of aldosterone and antidiuretic hormone) attempt to restore the decrease in plasma volume.[27-32]

The result of these various forces is the production of massive, and frequently intractable, edema.

Therapeutic Considerations

Since the widespread use of glucocorticoid therapy was introduced in the early 1950's, the morbidity and mortality rate associated with the idiopathic nephrotic syndrome has been significantly reduced. The success of any therapeutic regimen depends on the selection of patient material, and it has been our experience as well as that of others that the most important factor determining the outcome of therapy is the duration of the disease before the institution of steroids. The most successful outcome occurs in the younger child (two to six years of age) who has a classical, insidious onset of edema and who is presented for initial therapy within the first four months of the illness. Other characteristics of the child likely to do well on steroid therapy include urine containing massive protein with little or no cellular elements, normal blood pressure, and normal or only slightly reduced renal function as evidenced by a normal blood creatinine or urea nitrogen content.

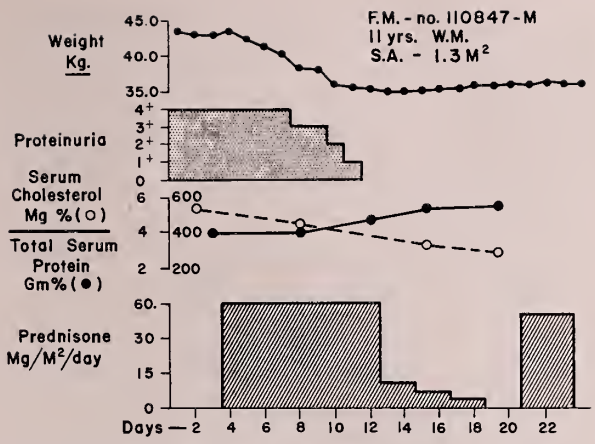

Fig. 10. Initial response to steroid therapy in an 11-year-old boy. Beginning diuresis was noted on the third steroid day. The weight loss (that is, diuresis) preceded any measurable rise in plasma proteins. This fact has been previously noted by other investigators.

Experience with the percutaneous renal biopsy has indicated still another important characteristic of the child who is likely to respond to steroid therapy, and, thus, have a favorable outcome.[3] Those patients manifesting a type 2 glomerulonephritis (particularly type 2 A) by light microscopy have a far more favorable prognosis than do those with a histologic lesion compatible with a type 1 glomerulonephritis.

For initial therapy, the patient is hospitalized and observed for two to three days to insure the absence of any infectious process. Reverse isolation is maintained during the course of intensive glucocorticoid therapy. It has been our practice to use prednisone in dosages of 50 to 60 mg./M^2 per day (or an equivalent dosage of other delta-1-derivatives) given in four equally spaced doses. Semi-quantitative determination of the urine

albumin (using equal parts of a solution of 3 per cent sulfosalicylic acid and urine) is made daily and steroids are continued until the urine has remained free of protein for two successive days, or to a maximum of 21 days if proteinuria persists.

The child with the classical idiopathic nephrotic syndrome who meets the criteria noted will generally show clearing of the proteinuria and significant diuresis in from 7 to 14 days following initiation of therapy. Fig. 10 is a diagrammatic representation of an 11-year-old boy who was treated in this manner. Beginning diuresis was noted on the third day of steroid treatment, and an ultimate loss of 8 kg. occurred by the 12th day.

Although most children who are going to respond to steroid therapy do so within the first three weeks of intensive treatment, a considerable number will ultimately clear their urine after one to three months of intermittent therapy. If the proteinuria has not cleared after three or four months of intermittent corticosteroids, it has been our practice to institute a second course of intensive therapy.

Long-term management is undoubtedly the most important aspect of treatment in the individual with the nephrotic syndrome, and should be instituted immediately following the course of intensive therapy. The authors utilize the following in their program:

1. Freedom from infection is one of the prime essentials of any therapeutic regimen in the nephrotic patient. This is particularly true during the initial months when superimposed infections are most likely to produce exacerbations of the active disease process. The *sine qua non* for achieving this relative freedom from infection is the strict observance of "social isolation." Children under therapy are removed from environments where potential contact with infectious diseases are common (schools, nurseries, theaters, and other places where more than two or three children are likely to congregate). For school age children it is desirable to have home teachers, which are frequently available through the local public school system. This social isolation should be continued for 9 to 12 months after the disease has become inactive.

Although prophylactic antimicrobials are frequently used by others, the authors have observed neither an increase in the number of infections nor an increase in morbidity since routine penicillin prophylaxis has been discontinued. Prompt institution of specific antimicrobial therapy with the onset of bacterial infections is still extremely important. The physician must be keenly aware of the frequent "masking" of infections by the use of steroids, and highly suspicious of nonspecific symptoms and signs (anorexia, lethargy, sudden increase in proteinuria) which may be the first signs of a superimposed infection.

Superimposed pyelonephritis is frequently observed but may go clinically undetected if periodic quantitative urine cultures are not performed. It has been our practice to obtain routine cultures at three-month intervals during the first year following the onset of the disease. When a superimposed urinary tract infection is discovered, it is important that specific therapy be instituted lest a potentially favorable prognosis be significantly altered.

2. Steroid "prophylaxis" should be instituted immediately after intensive therapy. The authors have used prednisone (or its equivalent) in dosages of 40 to 50 mg./M^2/day on three consecutive days of each week for six to nine months following complete clearing of the proteinuria. It has been our experience that exacerbations of the nephrotic syndrome are more likely to occur during the winter months (when the incidence of respiratory illness is higher), so it has been our practice to discontinue steroid therapy during the spring or summer months.

The four-year-old child represented in Fig. 11 illustrates the importance of intermittent maintenance therapy. The initial course of steroids was successful in promoting diuresis, but proteinuria and edema recurred within two months necessitating a second course of therapy which was only partially successful. A third course reduced the degree of proteinuria, but complete clearing was not attained and symptoms promptly returned. Following the partially successful fourth course, intermittent maintenance therapy was begun. Proteinuria gradually diminished and finally disappeared in about three months, and did not return after an additional six months of "prophylactic" maintenance.

3. Daily urine testing for proteinuria should be performed by the parents using a solution of 3 per cent sulfosalicylic acid. The parents should be instructed in the use

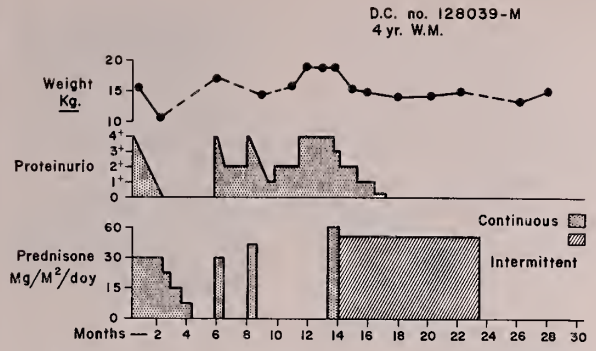

Fig. 11. Long term steroid "prophylaxis" in the nephrotic syndrome (see text). Intermittent steroids were essential for ultimate clearing of proteinuria.

of this reagent and should be made aware of the significance of "trace" reactions. The presently available urine "dip sticks" for protein are not recommended since they frequently miss these trace reactions.

If there is a recurrence of the proteinuria, *daily* treatment should be *promptly* reinstituted and continued until the urine is once again protein-free. When daily therapy begins immediately, the proteinuria generally subsides within a few days, but when there is a delay of even two to three days, steroids frequently must be administered for a longer period.

It is important that urine testing be continued—even after all other prophylactic measures have been discontinued. Recurrences of the full nephrotic syndrome have been observed up to seven years following the initial disease. If the recurrence is discovered early and steroids are begun promptly, the daily therapy and subsequent maintenance is likely to be brief. However, if the proteinuria is not observed until the clinical syndrome has recurred, intensive and long term maintenance therapy is again indicated.

4. Omission of routine immunizations is important during the period of maintenance therapy and for approximately one year following complete remission of the disease. Exacerbations of the full clinical syndrome are frequently observed following immunizations for tetanus, pertussis, diphtheria, smallpox, and typhoid. The authors have not observed recurrences following either the Salk or Sabin polio vaccine and therefore do not exclude these.

Emergency immunizations should, of course, be given, but the child should be observed closely following administration for a recurrence of proteinuria.

5. Diet and activity are not generally altered during the period of therapy. It is probably advisable to limit the intake of sodium during the course of intensive steroids but this does not appear necessary during the period of prophylactic therapy.

Some of the newer steroids (dexamethasone, betamethasone, paramethasone) have greater appetite-stimulating properties than either prednisone or triamcinolone, and it is occasionally desirable to limit the caloric intake if obesity becomes a problem.

6. Emergencies occasionally occur during or shortly following steroid therapy which require alterations of the dosage schedule. These usually are infections, but may result from other situations of stress such as accidents or operations. It has been our practice to alter the steroid therapy during moderately severe to severe infections (and certain viral infections such as varicella) to levels commensurate with what endogenous production would be in the non-steroid treated individual (approximately a two-fold increase in glucocorticoid production). This, in the patient whose endogenous production is suppressed by steroid therapy, would amount to 10 to 15 mg. prednisone/M^2/day. This should be administered in four equally spaced doses and should be continued until the infection is under control. A similar practice is followed during other situations of stress.

Prognostic Considerations

Since the introduction of steroid therapy, the ultimate prognosis of the nephrotic syndrome in the child appears to have been favorably influenced[26, 27] In the pre-antibiotic era, approximately one-third of all nephrotic children died in the acute phase, principally from superimposed or coincidental infection. Of the remaining patients, about one-half experienced spontaneous, and sometimes permanent, remissions while the remainder progressed to a chronic renal failure.[14]

Widespread use of antimicrobials virtually eliminated the acute deaths and, in the pre-steroid days, a favorable prognosis might be expected in approximately 50 per cent of all patients. Most investigators now feel that a favorable prognosis may be anticipated in 75 to 80 per cent of the cases. The ultimate prognosis of steroid-treated nephrotic patients is, however, still uncertain because of

the relatively short period of post-treatment observations.

There is still no formula for accurately assessing prognosis at the onset of the disease, but several factors appear important. When the disease first becomes manifest in late childhood, or when there is evidence of either hypertension, gross hematuria, and/or significant azotemia, the ultimate prognosis appears less favorable. Extensive examinations of renal biopsy specimens indicate that lesions which are primarily proliferative in nature, or which have progressed to the stage of membranous or membrano-lobular glomerulonephritis (light microscopy) have a more guarded prognosis. Further studies with serial renal biopsy and more detailed examination of the glomerular ultrastructure (electron microscopy) are needed to confirm these impressions.

Summary

The idiopathic nephrotic syndrome of childhood appears to be a hyperimmune response of the renal glomerulus to an agent, or agents, yet unidentified. The clinical disease is produced by a massive leakage of plasma proteins through the altered glomerular capillaries. A brief review of the pathogenesis and pathophysiology is presented and and a course of therapy is outlined.

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Diagnosis of Hypertension Caused By Unilateral Renal Artery Disease

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CASE REPORTS

UNILATERAL RENAL artery disease is a well documented cause of systemic hypertension. This is proved by many reports of long term reversion to normotensive levels following nephrectomy or revascularization of the kidney.[5, 7, 14] Following Butler's original case report in 1937 of a cure of hypertension following removal of a single diseased kidney,[4] many nephrectomies were performed with generally poor results.[16] With the advent of the aortography and split renal function studies, patient selection became more accurate, and the results of surgery improved.[2, 6, 7]

There is much controversy regarding the usefulness and precision of tests used in the diagnosis of this disease.[2, 7] The purpose of this report is to present a diagnostic approach to the hypertensive patient in whom the possibility of unilateral renal disease is entertained as the cause of the hypertension.

Incidence, Pathology And Pathophysiology

The true incidence of this disease is unknown, but is much greater than the other causes of surgically correctable hypertension. The disease can occur in any age group and in any sex. For unknown reasons it is not common in the Negro.[14]

Although many types of unilateral renal disease have been reported as causes of hypertension, renal artery lesions are the commonest. This discussion will be limited to diseases of the renal artery.

Atherosclerosis is the most frequent cause of renal artery narrowing. The lesion is most commonly found in the proximal one-third of the artery (Fig. 1). Statistically, it is more common in males over the age of 50.[7] Fibromuscular hyperplasia is the other important cause of renal artery disease. Pathologically there is extensive hyperplasia of the muscular layer of the vessel with or without concomitant thickening of the intima and adventitia. The involvement is usually diffuse, affecting the renal artery from its origin to its primary branches at the hilum of the kidney. The lesion is commonly discontinuous, producing areas of stenosis and poststenotic-dilatation, and giving the radiographic appearance of a string of beads (Fig. 2). It is more common in young adults and in females.[12]

Alterations in renal function are the basis for many of the diagnostic studies. Decreased blood supply to normally functioning nephrons must probably be present to produce hypertension secondary to unilateral renal disease.[11] This results in excessive reabsorption of sodium and water by the involved nephron. It is the only consistently altered renal function in this disease.[11] This abnormality has had wide confirmation in both humans and animals and is the basis of split function studies.[2, 3, 11, 13, 17]

Besides excessive reabsorption of sodium and water, a decreased blood supply to functionally normal nephrons probably has one other effect: the release of renin from the juxtaglomerular cells due to a decreased pulse pressure in the afferent arteriole.[9] Ren-

Unilateral renal artery disease as a cause of hypertension cannot be differentiated clinically from hypertension of other causes, and a battery of tests is required to properly evaluate the patient. A preliminary report of the pyelogram-urea washout test indicates that the test holds promise in diagnosis of the disease.

Fig. 1. Translumbar aortogram in a normotensive male revealing noticeable bilateral renal artery narrowing with poststenotic dilatation due to atherosclerosis (arrows point to constrictions in renal arteries).

Fig. 2. Retrograde femoral aortogram in a hypertensive female revealing the typical radiographic appearance (arrows) of fibromuscular hyperplasia of the right renal artery.

in then acts upon an alpha-2-globulin which eventually leads to the formation of angiotensin II with its well-known vasopressor effect.

Clinical Manifestation And Patient Selection

There are no typical symptoms, characteristic physical findings, or usual signs of progression of the disease process which can aid in the selection of hypertensive patients for study. The hypertension may be fixed or labile, severe or mild. Good response to bed rest, salt restriction, and drug therapy is not unusual. The duration of hypertension is of no real significance in the diagnosis. Of more importance is the age of the patient at onset of the disease. Hypertension with its onset before or after the essential hypertension age group is most likely to be secondary. The only helpful physical finding is the presence of an abdominal bruit. This murmur is characteristically heard over the upper abdomen and may be either harsh or soft in quality. When localized to the epigastric area and high pitched in character, there is a strong likelihood that it is secondary to a narrowed renal artery. The patient is best examined in a relaxed, recumbent position with his knees flexed. Tense abdominal muscles or significant background noise can obliterate the murmur. A hypertensive patient with a characteristic abdominal bruit has been reported to have a 66 per cent chance of having a renal artery lesion.[15] We have found that the abdominal bruit was a less accurate indication of the lesion than this percentage suggests.

Since there are no reliable historical or physical clues or simple, quick screening tests available, it is necessary to utilize certain criteria for selecting patients from a hypertensive population for further diagnostic studies to differentiate unilateral renal artery disease. The following are the criteria used at the University of Texas Medical Center:

1. Malignant hypertension of recent origin.
2. Onset of hypertension before 30 or after 50 years of age.
3. Hypertensive patients with a history of upper abdominal trauma, flank, or costovertebral angle pain.
4. Hypertensive patients with a characteristic upper abdominal bruit.
5. Hypertensive patients with unilateral abnormalities on renogram or intravenous pyelography.
6. Noticeable increase in severity of pre-

viously documented hypertension.

These are basically the criteria set forth by Howard[11] with minor additions and modifications.

Diagnostic Tests

Intravenous pyelography is the most useful screening test. Two parameters are measured: the appearance time of the contrast susbtance and the vertical length of the kidney. The appearance time of the dye is simply a gross measurement of renal blood flow. Films are made at 1, 3, 5, and 15 minutes. A kidney with grossly decreased renal blood flow will often show a delayed appearance time of the dye in comparison with the opposite normal side. Renal length is measured along the vertical axis. This is accomplished best when the nephrogram is present on the three- or five-minute film. The length of the two kidneys may vary normally as much as 1 cm., the right usually being slightly smaller than the left. If the difference in length is more than 1 cm. or if there is a delayed appearance time of the pyelogram, then further study is indicated. Dustan and Page found that 71 per cent of patients with proven unilateral renal artery disease will show one or both of these pyelographic abnormalities.[7] A number of patients with unilateral renal artery disease will therefore be excluded from further study if the pyelogram is the only screening test used.

The radioisotope renogram consists of the intravenous injection of I^{131} iodohippurate sodium (Hippuran) followed by recording the time of appearance and excretion of the isotope by both kidneys using external counters. This has recently been evaluated critically by Stewart and Haynie.[18] Their results revealed that the renogram, like the pyelogram, is not completely effective as a screening test because of a significant number of false negatives in patients with proven hypertension due to renal artery disease. It is accurate in patients with unilateral main renal artery stenosis and is of distinct value in following patients after revascularization procedures.

Aortography is the most widely used study in the diagnosis of this disease. Considerable controversy exists regarding the relative value of the aortogram and the split function studies. Some centers rely heavily on the aortogram as the definitive study[7, 14] whereas others depend on the split function studies.[2, 11, 17] It is our feeling that they are both necessary in the critical evaluation of these patients. The aortogram demonstrates the anatomical defect in the renal artery and the split function studies measure altered function. Except for the report of Morris and others,[14] surgical cures based on series using the aortogram alone are significantly lower than those based on split function studies.[2, 6, 7, 11, 12, 17] The mere presence of a renal artery lesion does not necessarily mean that the lesion is responsible for the patient's hypertension (Fig. 1). This is borne out both by the failure of some patients to respond to surgery and the demonstration of significant renal artery lesions in normotensive individuals.[8]

The percutaneous retrograde femoral approach under local anesthesia is preferred for aortography except when contraindicated by the presence of significant disease of the lower abdominal aorta, or its branches. Flexibility is greater, morbidity is less, and visualization is better with this technique.[10]

The split function studies measure the only consistent abnormality in this disease which correlates well with the results of surgery, that of excessive reabsorption of sodium and water by the involved renal tubules. The technique of this study is described elsewhere.[5, 11] Two exactly timed urine samples are obtained simultaneously from each ureter during a water diuresis with a urine flow rate of at least 2 cc. per min. The samples are analyzed for volume (cc. per min.), sodium (mEq. per liter), creatinine (mg. per 100 cc.), and osmolarity (mOsm. per kg. water). The contralateral normal kidney serves as the control. A significant reduction in volume of at least 35 per cent, and sodium of at least 15 per cent, with an increase in creatinine and osmolarity indicate a typical positive test and a high probability that the patient will have a good response to surgery.[2, 11, 17] The findings should be reproduced on the second collection. The creatinine levels and osmolarity are simply a reflection of the excessive reabsorption of water by the involved nephrons. In pyelonephritis and unilateral renal diseases with small degrees of involvement, the sodium concentration may be equal. In this situation the volume will be decreased and the creatinine and osmolarity elevated, constituting a positive test despite the sodium concentration.[11] In the usual cases in which a large

amount of involvement is present, the test is considered positive if the sodium and volume are decreased regardless of the creatinine or osmolarity levels.[17] In our experience, the osmolarity is unreliable in reflecting the excessive reabsorption of water.

Despite its accuracy, the split function study has serious disadvantages. Leakage of urine around the catheters into the bladder is not uncommon and at times makes interpretation of the results impossible. In addition, the test is time consuming, requires considerable technical skill, and the hazard of a urinary tract infection is always present. In bilateral renal artery disease, the study may be unreliable since the technique of the test presumes that the contralateral kidney is normal.

Because of the difficulty with the split function studies, we are evaluating the recently described pyelogram-urea washout test.[1] It uses two widely confirmed physiological principles, that of excessive reabsorption of water by involved tubules,[11] and exaggeration of this difference by a urea diuresis.[17] After the usual intravenous pyelogram preparation, a large volume (50 cc.) of concentrated diatrizoate sodium (Hypaque Sodium) or other suitable contrast substance is injected intravenously. Films are made at short intervals until an adequate pyelogram appears bilaterally. At this point, 500 cc. of normal saline containing 40 gm. of urea and 30 cc. of Hypaque are infused over a 15-minute period. Films are made at three-minute intervals from the start of the infusion, and the disappearance of the pyelogram is observed. An individual with normal renal function should "wash out" the pyelogram within 15 minutes of the beginning of the urea infusion due to the intense osmotic diuresis. The tubules of an involved kidney re-

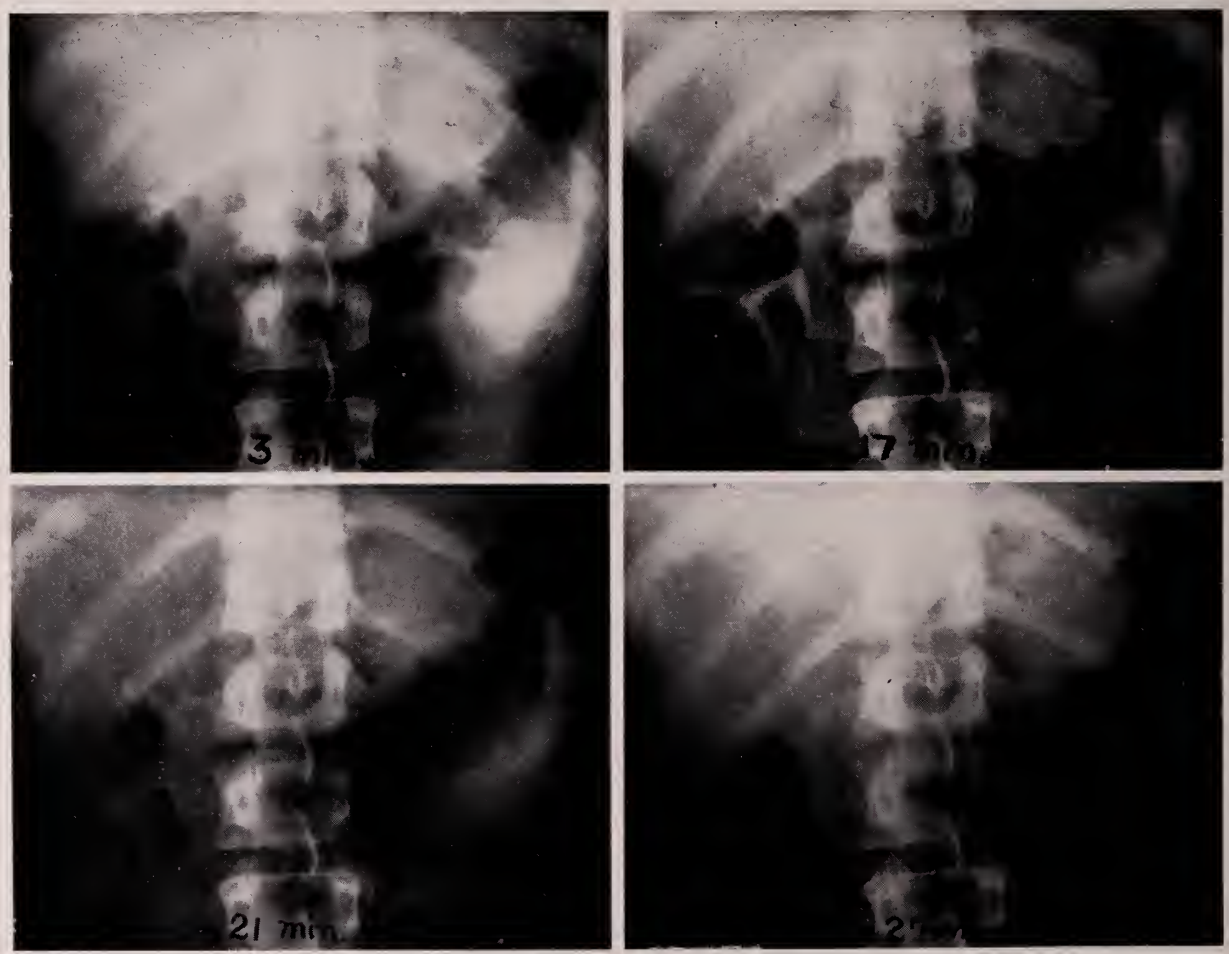

Fig. 3. Negative pyelogram-urea washout test. The kidney collecting systems had the appearance shown on the film marked 17 minutes when the urea infusion was begun. The films marked 21 and 27 minutes were exposed four and ten minutes respectively following the start of the urea infusion. Note the prompt bilateral disappearance of the contrast substance with no evidence of selective retention on either side.

TABLE 1.—*Pyelogram-Urea Washout Test of 39 Hypertensive Patients.*

	Number of Patients	Negative	Positive	False Positive	Results of Surgery
No evidence of unilateral renal artery disease	29	29*			
Unilateral renal artery disease	5		5†		4 cured, 1 improved
Obstructive uropathy	3			3‡	
Bilateral disease	2		2§		

*All had normal split function studies and/or aortograms.
†All had positive split function studies and aortograms.
‡Two had ureteral obstruction, 1 had severe prostatic hypertrophy. All had normal aortograms.
§One had bilateral renal artery disease by aortogram, 1 had ischemic tubular atrophy by needle biopsy.

absorb more water, even during a diuresis, and continue to concentrate the contrast material. Consequently, the pyelogram on the involved side does not become diluted as rapidly but persists and may even become denser.

We have employed this study in 39 hypertensive patients, all of whom had split function studies and arteriography. Five of these had unilateral renal artery disease. The results are shown in Table 1. In every instance the abnormal kidney retained the dye for 30 to 60 minutes while the pyelogram on the normal side was diluted and washed out

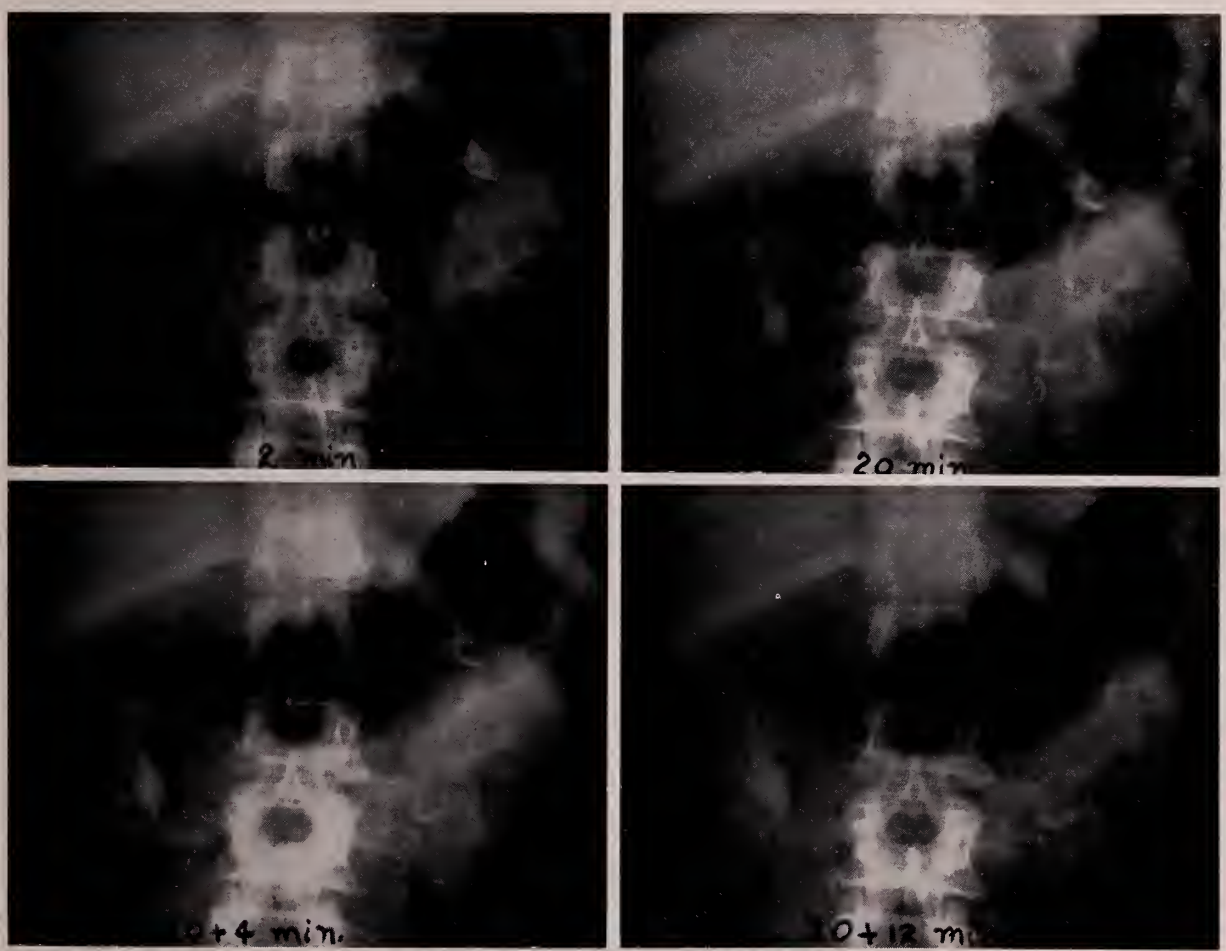

Fig. 4. Positive pyelogram-urea washout test. The two-minute film shows delay in appearance of the pyelogram on the right. The collecting systems had the appearance shown on the film marked 20 minutes when the urea infusion was begun. Note the prompt disappearance of the contrast substance on the left at 4 and 12 minutes following the start of the urea infusion with retention of undiluted contrast substance on the right during that period. The test is positive on the right.

promptly (Figs. 3 and 4). There was excellent correlation with the split function studies and results of surgery. There were false positive tests in three patients with urinary tract obstruction. In these instances, the pyelogram was retained longer on the obstructed than the nonobstructed side. Bilaterally positive tests occurred in two cases. Both of these had evidence of bilateral disease. It is of considerable importance that there were no false negative studies in our series.

The pyelogram-urea washout test, although admittedly crude, promises to be useful in the diagnosis of unilateral renal disease because of the sound physiologic principles upon which it is based. The small number of cases in our series precludes any far-reaching conclusions at this time, but the results indicate that further evaluation of the method should be made. Its reliability in segmental and bilateral disease has yet to be determined.

Summary and Conclusions

Unilateral renal artery disease, although uncommon as a cause of hypertension, is the most common cause of surgically correctable hypertension. It cannot be differentiated clinically from hypertension of other causes, and a battery of tests must be used to properly evaluate the patient. With proper selection, the results of surgery are excellent. A preliminary evaluation of the pyelogram-urea washout test is reported, indicating that the study may hold considerable promise in the diagnosis of this disease.

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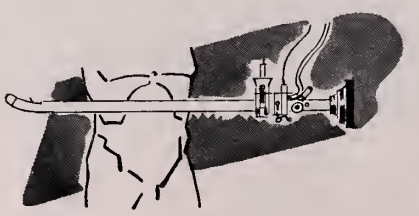


Bacteriuria in Pregnancy
Preliminary Report

A. L. LE BLANC, M.D., and WILLIAM J. McGANITY, M.D.

CASE REPORTS

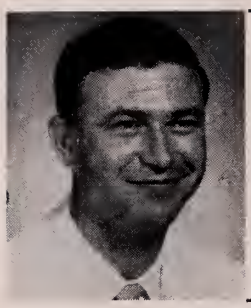
IN 1958, at a symposium on pyelonephritis held at the Henry Ford Hospital, Kass presented evidence that asymptomatic bacteriuria had been found in 6 per cent of 4,000 pregnant patients surveyed. For satisfactory reasons, asymptomatic bacteriuria was defined as a condition in which positive urine cultures with colony counts of greater than 10^5 organisms were found in the absence of symptoms of urinary tract infections. In a smaller, more carefully studied group, he presented evidence that bacteriuria, if it were to occur, would appear early in pregnancy, thus making it amenable to early diagnosis and treatment. Far more provocative was his finding of an incidence of subsequent acute pyelonephritis of 42 per cent and a prematurity rate of 24 per cent in a group of untreated, pregnant patients with asymptomatic bacteriuria. In a similar group of pregnant patients with initial asymptomatic bacteria, who were treated with antimicrobials until the culture was negative and then prophylactically through the remainder of pregnancy, there were no cases of acute pyelonephritis and the prematurity incidence was 10 per cent.[13] Although the groups were relatively small, the implications of these findings were considerable; for not only had a means of diagnosing a condition of significant risk to mother and infant been presented, but also presented was a method of adequately eliminating that risk.

The importance of prematurity is unquestioned, and represents one of the most important challenges to obstetrical investigation. The importance of acute pyelonephritis in pregnancy is also considerable, occurring with an incidence of 1 to 2 per cent[14] and being implicated as a significant cause of gram-negative shock,[1] premature labor, and eventual chronic pyelonephritis.[28] Since both acute pyelonephritis and prematurity are important obstetrical problems, it is no wonder that a great deal of interest has arisen from Kass's work, and that the problem has, so far, been found to be much more complicated than initially suggested. It is, however, surprising that the extent of this problem and its true significance is unanswered.

Much current investigation is being directed toward clarifying the problem of urinary tract infection in general. The predominance of the gram-negative coliforms as the causative organisms in acute pyelonephritis has presented an enigma, as these organisms rarely cause significant infection elsewhere in the body, except in the newborn or debilitated. Evidence that the interstitial tissue of the renal medulla is the initial site of renal infection is accumulating,[7] and inadequate or altered immunological defense in this area may be of considerable importance.[2] Homogenates of renal tissue have been shown capable of decreasing complement activity of serum.[2] Bacterial ureases and renal interstitial acidosis may increase this anti-complementary defect.[3, 6] These factors may be important in explaining predominance of coliforms in acute pyelonephritis, and the increased susceptibility in pregnancy and diabetes to urinary tract infection. Evidence of altered renal function in patients with asymptomatic bacteriuria has appeared in recent investigations.[12]

Pregnancy represents a condition, among others, in which there is increased susceptibility to acute pyelonephritis. This has been

In an attempt to clarify the significance of bacteriuria in pregnancy, the investigators have begun a comprehensive survey scheduled to run several years. Results of the first year survey, which covered 857 patients, suggest several interesting possibilities.



Dr. A. L. Le Blanc is an instructar in the Department af Obstetrics and Gynecolagy at the University af Texas Medical Branch and his ca-authar is professar and chairman af that department.*

related to urinary stasis, demonstrated radiographically by dilatation of the urinary collecting system.[5] This dilatation appears early in pregnancy, and evidence indicates that it is hormonally rather than mechanically induced.[19, 24, 27] Specifically, progesterone has been shown to decrease the tone and contractility of the smooth muscle of the ureter *in vitro*.[15]

Ureteral dilatation would encourage the proliferation of bacteria in the collecting system on the basis of a static fluid obstruction. Ascending infection, by the Brownian movement of particles,[13] leading to an increased susceptibility of all pregnant women to urinary tract infection, and the occurrence of an asymptomatic bacteruric state in pregnancy would then complete the necessary background for a smaller, well-defined group of patients in whom acute pyelonephritis would be expected to predominantly occur—that is, a group comparable to Kass's untreated bacteruric group.

The use of colony count culture methods has been of tremendous value to those who have investigated this problem since Kass's work was published, and several surveys of bacteriuria in pregnancy utilizing colony count cultures are available in the literature. Monzon, and others,[16] surveyed 1,400 pregnant patients and found an incidence of asymptomatic bacteriuria of 7 per cent, with the causative organism being primarily coliforms. Approximately one-third of the patients in the asymptomatic bacteruric group spontaneously became culture negative without therapy. Antimicrobial therapy resulted in reversal to negative cultures in 60 per cent of the patients with asymptomatic bacteriuria. Kaitz and others[11] surveyed 616 pregnant patients in a slightly higher socioeconomic group, and found an incidence of asymptomatic bacteriuria in pregnancy of 4.4 per cent. The incidence of acute pyelonephritis was considerably higher in the bacteruric group, although cases of pyelonephritis did appear in patients with initial negative cultures. The relationship of bacteriuria to prematurity is much less clear. Henderson and others[8] presented a group of patients in whom the prematurity rate was higher in those with bacteriuria. Turck and others[26] were unable to relate prematurity to bacteriuria. In the most recent report in this regard Sanford and others[20] could determine no difference in prematurity between bacteruric and nonbacteruric patients in a group of 418 pregnant patients surveyed. To clarify the relation of bacteriuria to acute pyelonephritis and to prematurity, a large population survey, begun without preconceived ideas and continued to its conclusion in spite of initial findings, would be required. If analyzed frequently, leads to possible physiological defects in the bacteruric patient could be pursued. No such survey exists at present. However, many institutions are now proceeding along these investigative lines, and the answers undoubtedly will be forthcoming.

Clinical Survey Procedure

General Aims.—In April, 1962, a clinical survey was begun at the University of Texas Medical Branch in an attempt to clarify the significance of bacteriuria in our own obstetrical, clinic population. The overall survey was to continue for three to four years so that eventually 4,000 to 5,000 patients would be screened and analyzed. The aims of the first year of the survey were as follows:

1. Accurate definition of the clinic population being surveyed as to race, age, gravidity, and so on.
2. Determinations of the incidence of asymptomatic bacteriuria in this clinic group.
3. Initial assessment of the relationship of bacteriuria to prematurity and acute pyelonephritis to allow proper group division in subsequent years.
4. Evaluation of fetal toxicity of the drugs chosen for long-term therapy.
5. Evaluation and alteration of deficiencies of laboratory procedures and techniques.

All urine specimens cultured and examined

*This paper was presented originally during the TMA Annual Session in Dallas in 1963, and later to the Charles T. Stone Society of Internal Medicine in June, 1963.

TABLE 1.—*Protocol Designation.*

Protocol	Past History And/Or Symptoms	Initial Urinalysis	Initial Culture	No. of Patients	% Of Total
A	—	N.A.	—	608	70.9
B	—	—	+	35	4.1
C	—	+	+	23	2.7
D	+	N.A.	+	28	3.3
E	+	N.A.	—	134	15.6
F	Initial Entry With Pyelonephritis			4	0.4
G	Original A-Subsequent s/s with Positive Culture			16	2.6*

* Of Group A.

during the first year of the survey were to be collected by catheterization to allow:

1. Accurate evaluation of initial asymptomatic bacteriuria at the first clinic visit.
2. Definition of the accuracy of clean-caught specimens by comparison of a second year of clean-caught technique to the first year of catheterization technique.
3. Evaluation of the risk of catheterization in pregnancy.

Each year's survey was to be altered to allow pursuit of leads discovered in preceding years. However, the group was to be left generally intact, so that at the end of the survey enough patients could be evaluated to answer the basic question of the relationship of asymptomatic bacteriuria to acute pyelonephritis in prematurity.

Protocols.—At the time of initial visit to the Prenatal Clinic, each patient was questioned in regard to current urinary symptoms and to past history of urinary tract infection, and a specimen was obtained by catheter for colony count culture and routine urinalysis. On the basis of these parameters, the patients were assigned to protocols (Table 1). Group A included patients who were asymptomatic and who had negative cultures. Group B and C consisted of patients with initial asymptomatic bacteriuria (colony counts of greater than 10^5). Group B contained patients with an initial negative urinalysis—that is, proteinuria of less than 1+, white blood cells less than 10 per high power field, and absence of bacteria in the urinary sediment. Group C consisted of patients with initial positive urinalysis by any of the preceding criteria. Groups D and E included patients with either a significant past history of urinary tract infection or positive urinary symptoms. Group D included patients with initially positive cultures, and Group E included those with initially negative cultures. A separate Group F was established to include patients initially hospitalized for acute urinary tract infection in pregnancy prior to being seen in the Prenatal Clinic. Also a Group G was designated for those patients who initially were assigned to Group A and who subsequently developed acute pyelonephritis with positive cultures. This group was designed to furnish the probable gross incidence of infection following catheterization.

Drug Randomization.—All patients other than those in Group A were assigned to randomized drug treatment by the last digit of their unit history number. The possible treatments consisted of (1) no drug; (2) Mesulfin (Thiosulfil and Mandelamine combination); (3) Mandelamine; and (4) Furadantin (Table 2). Group E, though initially culture negative, was assigned to drug randomization to serve as a drug control group. All drugs were begun in therapeutic dosage and continued at this level until a negative culture had been obtained. At this time the dosage was reduced by half and continued as a prophylactic medication until delivery, unless drug toxicity or patient failure interfered. Drugs were not changed on the basis of resistant organism, unless acute pyelonephritis appeared.

Follow-up and Laboratory.—Repeat catheterizations were performed in all protocols other than Group A at two-week intervals until a negative culture had been obtained and then at six-week intervals until delivery, unless urinary symptoms or a positive culture recurred. All patients delivered from the survey group were catheterized at delivery for urinalysis for colony count culture. Also at delivery, specimens of maternal venous blood, cord blood, and amniotic fluid were obtained for measurement of bilirubin levels on all groups, and sulfa levels

TABLE 2.—*Drug Schedules Used.*

Drug	Therapeutic Dose (mg. q.i.d.)	Prophylactic Dose (mg. q.i.d.)
Mesulfin	1000	500
Mandelamine	500	250
Furadantin	100	50

on those patients on Mesulfin. These collections were randomized only by the pressure of the work load.

All laboratory procedures were performed by the clinical laboratory of this hospital by currently used techniques. Running records were kept on all patients throughout their prenatal course, except those in Group A, and a coding mechanism was established to permit the entry onto IBM cards of all pertinent information on all patients, so that a complete analysis of as many parameters as possible could be performed.

Results

General Information.—On Feb. 1, 1963, initial analysis was begun, primarily to allow planning for the second year of the project. By this time, approximately 1,300 patients had been entered on protocols, and 857 of these were known to have delivered. Accurate delivery information was available on the 817 of this group who had delivered at the Medical Branch hospital. Three sets of twins were included in this group. The information thus far has been of some interest, and will be presented for that reason. However, caution should be exercised in arriving at the final conclusions from any survey still in progress. Many of the groups are too small to do more than suggest interest. The overall incidence of initial bacteriuria on catheter-drawn specimens was 10.5 per cent, and the incidence of asymptomatic bacteriuria in the total group of 857 patients was 6.8 per cent (Table 3). Negative urinalysis was found in 60.3 per cent of the patients with initial bacteriuria by culture.

Dysuria appeared in 8.1 per cent of the patients, however, only 24.6 per cent of the patients with dysuria had a positive culture. Frequency of urination was present in 11.7 per cent of the total group, with 22 per cent of these patients having positive cultures. Positive past histories of urinary tract infection were present in 14.6 per cent of the population, and of this group 18.4 per cent had an initial bacteriuria. In the group of patients with a negative past history of urinary tract infection, who made up 85.4 per cent of the total, 8.6 per cent were found to have initially positive cultures. Of the total group with initially positive cultures, only 30 per cent had bacteria in the urinary sediment, and only 23.3 per cent had more than 10 white blood cells per high power field in the sediment (Table 4).

Incidence of Pyelonephritis.—Fourteen cases of acute pyelonephritis, by rigid criteria, occurred in the total group giving an overall incidence of 1.7 per cent. In the 730 patients with initial negative cultures, 11 cases of acute pyelonephritis appeared, giving an incidence of 1.5 per cent. However, the incidence of acute pyelonephritis in the initial bacteruric non-treated group was 10.5 per cent, as compared to 1.5 per cent in the initial bacteruric treated group (Table 5).

Effect of Bacteriuria on Prematurity and Delivery Culture.—Prematurity is, in all tables, defined purely on the basis of a weight

TABLE 3.—*General Information.*

Total Patients Analyzed	857
Total Delivered at John Sealy Hospital	817
Total No. Infants	820
% With Initial Positive Culture (B, C, D, F) — Total	10.5%
% With Initial Positive Culture (B, C)— Asymptomatic	6.8%
% Asymptomatic Bacteriuria With Negative Urinalysis	60.3%
% Prematurity Total Group	12.0%

TABLE 4.—*Symptoms, Past History, Urinalysis—Culture (857 Patients).*

Symptom Or Past History	% Total Group Exhibiting Finding	% Subgroup With Positive Initial Culture
Dysuria	8.1	24.6
Frequency	11.7	22.0
Positive Past History	14.6	18.4
Negative Past History	85.4	8.6
Initial positive culture	30.0% Bacteria in sediment	
	23.3% Greater than 10 WBC/HPF	

TABLE 5.—*Incidence Acute Pyelonephritis.*

Group	No.	Acute Pyelonephritis No.	% Incidence
Total Group	817	14	1.7
Initial Negative Culture	733	11	1.5
Initial Positive Culture— With Rx	65	1	1.5
Initial Positive Culture— No Rx	19	2	10.5

TABLE 6.—*Outcome Initial Negative Culture Group—Prematurity.*

Drug	No.	% of Total	Premature Births No.	%
No Drug	612	83.5	75	12.3
Drug Rx-Total	121	16.1	9	7.4
Mesulfin	55	7.5	5	9.1
Mandelamine	18	2.5	2	11.1
Furadantin	45	6.1	2	4.4
Other Drug	3	0.4	0	0.0

TABLE 7.—*Outcome Initial Positive Culture Group—Prematurity.*

Drug	No.	% of Total	Premature Births No.	%
No Drug	19	22.6	5	26.3*
Drug Rx-Total	65	77.4	6	9.2
Mesulfin	35	41.7	5	14.3
Mandelamine	7	8.3	0	0.0
Furadantin	23	27.4	1	4.3

*Significant at 1%.

of less than 5 pounds, 8 ounces. The overall incidence of prematurity in the total group was 12 per cent. In the group of patients with initial negative cultures who received no drug, the incidence of prematurity was 12.3 per cent. In the drug controlled group, that is, patients with initial negative cultures who received drugs during pregnancy, the incidence of prematurity was 7.6 per cent (Table 6). In the initial positive culture group, who received no drugs, the incidence of prematurity was 26.3 per cent, as compared to a 9.2 per cent incidence of prematurity in the bacteruric group treated with drugs through pregnancy (Table 7). The untreated positive group is small, but the increased prematurity rate is significant at 1 per cent limits of confidence, both to the bacteruric group and to the total group. The incidence of positive cultures at delivery was related to the initial culture (Table 8). In the group of patients with initial negative cultures, the incidence of positive culture at delivery was 9.6 per cent, and did not vary with or without drug treatment. In the initial bacteruric group receiving no treatment, the incidence of positive delivery cultures was 53.3 per cent, as compared to 24.6 per cent in the initial bacteruric drug treated group. Again, though small, the untreated group is significantly different at 1 per cent reliability.

Effect of Age, Gravidity, and Race on Bacteriuria and Prematurity.—The specific effects of age, gravidity, and race were analyzed to attempt to prevent bias in the findings, based on the effect of these factors on prematurity. As related to age, it was evident that though the incidence of bacteriuria was essentially the same, the prematurity rate was slightly higher in the less than 20 and greater than 30 year age groups (Table 9). Again, though the groups are small, the highest incidence of prematurity in each age group occurs in the patients with initial positive cultures who received no treatment during pregnancy (Table 10). The initially positive culture group receiving treatment had a similar prematurity incidence as the total group or the initially negative culture group.

In analyzing the effect of gravidity (Table 11), it was noted that the primigravida had the highest incidence of initial bacteriuria. Beginning with the second pregnancy, the incidence of initial bacteriuria increased with each pregnancy thereafter. The primigravida also had an increased risk of prematurity, in general, as compared to the other groups.

The most notable effect seen in our clinic population was related to race, and because of the racial distribution, separate analysis was performed for race (Table 12). Negroes make up 62.4 per cent of our clinic population, and the prematurity incidence in the Negro was significantly different from the white group. This was not surprising in that 5 pounds, 8 ounces had been picked as the dividing line on prematurity. No particular

TABLE 8.—*Delivery Culture Outcome by Group and Drug.*

Initial Culture	Drug Group	No.	% Total	Positive Delivery Culture No.	%
	No Drug	572	83.3	55	9.6
Negative	Drug Rx	115	16.7	11	9.6
	Total	687	100.0	66	9.6
	No Drug	15	19.7	8	53.3*
Positive	Drug Rx	61	80.3	15	24.6
	Total	76	100.0	23	30.3
	Mesulfin	33	43.4	7	21.2
	Mandelamine	7	9.2	2	28.6
	Furadantin	21	27.6	6	28.6

*Significant at 1%.

TABLE 9.—*Effect of Age on Bacteriuria and Prematurity.*

Age in Years	No.	% Of Total	Positive Initial Culture No.	%	Prematures No.	%
20	197	24.0	21	10.7	27	13.7
20-29	439	53.5	47	10.7	45	10.3
30	181	22.1	16	8.8	25	13.8

TABLE 10.—*Effect of Age, Bacteriuria and RX on Prematurity.*

Age	Group	Total No.	Prematures No.	Prematures %
20	Negative Culture	175	24	13.7
	Positive Culture—With Rx	18	2	11.1
	Positive Culture—No Rx	3	1	33.3
	Total	197	27	13.7
20-29	Negative Culture	391	38	9.7
	Positive Culture—With Rx	36	4	11.1
	Positive Culture—No Rx	11	3	27.3
	Total	439	45	10.3
30	Negative Culture	165	24	14.5
	Positive Culture—With Rx	11	0	0.0
	Positive Culture—No Rx	5	1	20.0
	Total	181	25	13.8

TABLE 11.—*Effect of Gravidity on Bacteriuria and Prematurity.*

Gravidity	No.	% of Total	Initial Positive Culture No.	Initial Positive Culture %	Prematures No.	Prematures %
1	136	16.6	18	13.2	20	14.7
2	147	17.9	9	6.1	16	10.9
3	112	13.7	10	8.9	10	8.9
Over 4	421	51.3	47	11.2	51	12.1

TABLE 12.—*Effect of Race on Prematurity and Bacteriuria.*

Race	No.	% of Total	Prematures No.	Prematures %	Initial Positive Culture No.	Initial Positive Culture %
White	131	16.0	12	9.2	35	11.5
Latin	174	21.2	15	8.6		
Negro	512	62.4	70	13.7	48	9.6

TABLE 13.—*Effect of Race, Bacteriuria, and Rx on Prematurity.*

Race	Group	Total No.	Prematures No.	Prematures %
Negro	Total	512	70	13.7
	Negative Culture	464	62	13.4
	Positive Culture—With Rx	37	4	10.8
	Positive Culture—No Rx	11	4	36.4*
White & Latin	Total	305	27	8.9
	Negative Culture	269	24	8.9
	Positive Culture—With Rx	27	2	7.4
	Positive Culture—No Rx	8	1	12.5

*Significant at 1%.

difference in initial bacteriuria was noted between the white and non-white groups. Separate analysis of the effect of bacteriuria, with and without treatment, on prematurity was performed in the Negro and white populations (Table 13). In both groups the incidence of prematurity was, again, highest in the initial bacteruric untreated group, although this was certainly more notable in the Negro population where the incidence of prematurity in the initial bacteruric nontreated group was 36.4 per cent. The treated Negro group, with initial bacteriuria, had a prematurity rate less than the total group or the initial negative group. Again, though small, these figures are significant at the 1 per cent level in the Negro population.

Effect on the Fetus of Long-Term Drug Therapy Introduction.—Soon after the appearance of the sulfonamides, investigation revealed rapid movement across the placenta to reach equilibrium in the fetal circulation in two to three hours, with a relative concentration of the drug in the amniotic fluid.[23] Although similar, the various sulfonamide derivatives differed in some respects.[22] It has been amply demonstrated that unconjugated bilirubin levels are related to kernicterus,[4, 9] and that the sulfas compete with unconjugated bilirubin, both for protein carrying and for conjugation. This competition has been demonstrated in the test tube,[17, 18] the laboratory animal,[10] and the newborn premature infant.[21] There seems no argu-

TABLE 14.—*Maternal Venous Bilirubin—143 Specimens.*

Fraction	Drug Group	No.	Range	Mean	Standard Deviation*	Standard Error*
Total Bilirubin	Sulfa	36	0.1 - 2.0	0.76	0.44	0.07
	Other Drug	36	0.1 - 1.8	0.60	0.22	0.04
	No Drug	71	0.1 - 2.2	0.61	0.34	0.04
	Total	143	0.1 - 3.0	0.64	0.34	0.03
Direct Bilirubin	Sulfa	36	0.0 - 1.2	0.26	0.19	0.03
	Other Drug	36	0.0 - 0.7	0.28	0.17	0.03
	No Drug	71	0.0 - 1.2	0.20	0.18	0.02
	Total	143	0.0 - 1.2	0.24	0.18	0.02

*All standard deviations and standard errors are approximations.

TABLE 15.—*Amniotic Fluid Bilirubin—33 Specimens.*

Fraction	Drug Group	No.	Range	Mean	Standard Deviation*	Standard Error*
Total Bilirubin	Sulfa	8	0.1 - 0.5	0.28	0.16	0.06
	Other Drug	7	0.1 - 1.0	0.40	0.30	0.11
	No Drug	18	0.1 - 1.4	0.37	0.27	0.06
	Total	33	0.1 - 1.4	0.35	0.25	0.04
Direct Bilirubin	Sulfa	8	0.1 - 0.2	0.13	0.04	0.01
	Other Drug	7	0.0 - 0.4	0.13	0.12	0.05
	No Drug	18	0.0 - 0.6	0.17	0.16	0.04
	Total	33	0.0 - 0.6	0.15	0.13	0.02

*All standard deviations and standard errors are approximations.

TABLE 16.—*Umbilical Cord Bilirubin—149 Specimens.*

Fraction	Drug Group	No.	Range	Mean	Standard Deviation*	Standard Error*
Total Bilirubin	Sulfa	38	0.5 - 3.2	1.40	0.76	0.13
	Other Drug	40	0.2 - 3.7	1.55	0.82	0.13
	No Drug	71	0.2 - 3.6	1.43	0.71	0.08
	Total	149	0.2 - 3.7	1.47	0.75	0.06
Direct Bilirubin	Sulfa	38	0.0 - 1.1	0.46	0.27	0.04
	Other Drug	40	0.0 - 1.1	0.46	0.28	0.04
	No Drug	71	0.0 - 1.1	0.36	0.20	0.02
	Total	149	0.0 - 1.1	0.41	0.25	0.02

*All standard deviations and standard errors are approximations.

TABLE 17.—*Incidence Exchange Transfusion.*

Drug Group	No Exchange		Exchange - Rh		Exchange - ABO	
	No.	%	No.	%	No.	%
Sulfa	91	100.0	0	0	0	0
Other Drug	98	100.0	0	0	0	0
No Drug	625	99.0	3	0.5	3	0.5

ment to the rule that sulfa should not be given to the newborn, as an increased incidence of kernicterus can be expected, but there has also been the implication that these drugs should be withheld from the pregnant patient, at least on a long-term basis, and specifically for the patient in whom the possibility of an Rh or ABO problem exists. Because of this, it appeared necessary to relate the fetal toxicity of the drugs chosen in this survey. Mesulfin, the sulfa chosen for this survey, contains a soluble sulfa which presents in the urine in free form primarily. Only 2 per cent is found in the form of the glucuronate salt and 7 per cent acetylated sulfamethizole being found in the urine in prior studies.[25] No specific fetal toxicity has previously been related to Furadantin or Mandelamine.

Drug effects on fetal toxicity from the drugs chosen for this survey were evaluated by studying three parameters. Concomitant samples of amniotic fluid, cord blood, and maternal blood were obtained at delivery for evaluation of the total and direct bilirubin. Also, the number of exchange transfusions, and the fetal outcome were analyzed.

The ranges and mean bilirubin in each compartment were analyzed separately for the drug and the non-drug groups and no specific difference in either range or mean value was found in any compartment (Tables 14, 15, 16). Only six exchange transfusions were required in the entire group, and all

TABLE 18.—*Fetal Outcome by Drug.*

Fetal Outcome	Sulfa		Other Drug		No Drug		Total	
	No.	%	No.	%	No.	%	No.	%
Abortion	1	1.1	1	1.0	6	1.0	8	1.0
Immature Died	1	1.1	1	1.0	3	0.5	5	0.6
Stillborn	2	2.2			11	1.7	13	1.6
1000 gm. Died 24 hrs.	2	2.2			3	0.5	5	0.6
1000 gm. Died 24-72 hrs.					1	0.2	1	0.1
1000 gm. Died 72 hrs.					2	0.3	2	0.2
1000 gm. Living 72 hrs.	85	93.4	96	98.0	605	95.9	786	95.9
Total	91		98		631		820	
Fetal Death Excluding Abortion	5	5.5	1	1.0	20	3.2	26	3.2

Overall Pregnancy Loss	4.1%
Loss 400 gm.	3.2%
Loss 1000 gm.	2.6%
Stillbirth Rate	1.6%

occurred in patients receiving no drug (Table 17). The blood groups and types in each drug group were determined to avoid bias in the sulfa or no treatment group, and were found to be similar.

There was no significant difference in fetal outcome in any of the drug groups, although the fetal death rate was slightly higher in the sulfa group (Table 18). In analyzing the causes of death in the drug group, no toxic deaths could be found (Table 19).

Conclusion

No definite conclusion as to the true effect of untreated bacteriuria on prematurity can yet be delineated in this group of patients, as the sample size is yet too small. There is suggestive evidence that the prematurity rate, at least in the Negro, is higher in those with initial bacteriuria. There is also suggestive evidence of an increased rate of acute pyelonephritis subsequent to untreated bacteriuria. These tendencies do not appear altered by any bias of racial group, gravidity, or age. Treated bacteriuria does not appear to carry an increased risk of either acute pyelonephritis or prematurity, and enough patients were analyzed in this regard.

There appears to be little correlation between urinary symptoms, past history of urinary tract infection, or the findings on routine urinalysis to positive culture.

Catheterization does appear to carry a significant risk, lying somewhere between 2.5 and 10 per cent. It will not be possible to analyze the true significance of catheterization until the second year survey comparison with clean-caught specimens is completed. It is of interest that almost half of the patients with initial bacteriuria, who were not treated during pregnancy, had negative cultures at delivery.

No significant difference in bilirubin levels was determined in the sulfa group. A number of sulfa determinations were made from maternal blood, cord blood, and amniotic fluid, and although they are too small to permit any analysis (Table 20), the mean value for sulfa is significantly higher in the amniotic fluid compartment. Thus far, no increased risk of exchange transfusion or fetal loss has been evident in the drug treated group. It appears that although sulfa may exert a detrimental effect when given to the

TABLE 19.—*Causes of Fetal Death in Drug Group.*

Drug	Death Classification	Wt. (oz.)	Cause of Death
Sulfa	Immature	34	Immaturity - Respiratory
	Stillborn	90	Abruptio Placenta - Anoxia
	Stillborn	87	Anoxia - Antepartum Death
	Neonatal	71	Subarachnoid Hemorrhage, Atelectasis
	Neonatal	56	Tracheo - Esophageal Fistula
Mandelamine	Immature	35	Atelectasis

TABLE 20.—*Sulfa Determinations—74 Specimens.*

Source	No.	Range	Mean
Maternal Venous	31	0.1 - 2.4	0.41
Amniotic Fluid	7	0.3 - 2.2	1.00
Umbilical Cord Blood	36	0.1 - 2.5	0.45

newborn prior to the development of a conjugating system in the newborn liver, a protective device exists during pregnancy and prevents the adverse effects of sulfa on bilirubin metabolism. This protective device appears to be the maternal liver.

Summary

An initial analysis of 857 patients studied for the effects of bacteriuria in pregnancy is presented. The overall incidence of bacteriuria in the total group was 10.5 per cent. The incidence of asymptomatic bacteriuria was 6.8 per cent.

Prematurity rates were higher in the initial bacteruric nontreated groups (26 per cent), and this effect was not altered by separate evaluation of the population on the basis of age, gravidity, or race.

In the initial bacteruric, drug treated group, the incidence of prematurity was no different than the initially negative culture group.

The incidence of acute pyelonephritis was higher in the nontreated initial bacteruric group, and this tendency was apparently reversed by drug treatment.

No adverse effects on bilirubin ranges or mean values in the cord blood or amniotic fluid were determined in the sulfa treated group as compared to the untreated group. No adverse effects of long-term drug therapy were noted in the incidence of exchange transfusions or fetal survival.

All groups are felt to be too small to allow final conclusions, and suggest only interesting possibilities at present.

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Opportunistic Fungal Infections Of the Urinary Tract

SYMPOSIUM

IT IS APPARENT that the incidence of infections due to the more opportunistic fungi is increasing at a rapid rate. This phenomenon is probably due to a change in the host resistance of the general population rather than to an increase in occurrence of these organisms in our environment. Chronic diseases, malignancies, leukemia, diabetes mellitus, antimetabolite and adrenocorticosteroid therapy, prolonged antibiotic administration, and extensive surgical procedures, all more common in this era of modern medical practice, have been shown as predisposing factors in patients with various fungal diseases.

Recent observations at this institution of two patients with severe *Candida* pyelonephritis prompted a review of fungal infections of the urinary tract.[22]

Candidiasis

More than 100 cases of disseminated candidiasis have been described in the literature; 67 of these were documented as having severe renal damage due to proliferation and invasion of *Candida* organisms.[7-10, 16, 17, 19, 25] All had extensive involvement of the renal parenchyma with *Candida* abscesses. One patient had enough fungi in the renal pelvis to produce ureteral obstruction,[7] and one had necrotizing papillitis.[17] It was significant that, from the clinical viewpoint, these patients had all the symptoms of bacterial pyelonephritis, including pyuria, and that *Candida* organisms were invariably found in large numbers during adequate examination of the urine. The majority of patients died in renal failure, the most common cause of death in patients with this disease.[19] Two patients with severe renal involvement were cured following the administration of amphotericin B.[19, 22]

There have been four cases of primary renal candidiasis reported since 1930[1, 24, 29] and possibly three cases before then.[20] All were unilateral in distribution and in three of the four well-documented instances there was no parenchymal involvement.[1, 24] In these, the fungi were limited to the kidney pelves and calyces. One patient had complete obstruction due to fungus balls in the renal pelvis,[29] and another passed a soft whitish membrane in the urine.[1] Clinically, all had symptoms of severe unilateral pyelonephritis and had urine cultures positive for *Candida*. Two patients were cured following nephrectomy.[1] None were treated with amphotericin B. Another patient, recently observed at this institution, with primary unilateral *Candida* pyelonephritis and numerous fungus balls in the urine, was cured with amphotericin B.[22]

Candida cystitis has been observed in more than 30 patients.[3, 13, 16, 21, 26, 28] All had symptoms of chronic cystitis which were not relieved by and were often aggravated by antibiotic therapy. At cystoscopy, most of these patients had a shaggy white membrane adherent to the bladder mucosa. Two patients had gas-containing fungus balls, 8 to 10 cm. in diameter, lying free within the bladder lumen.[3] Although many forms of therapy have been tried with generally poor results, local instillation of amphotericin B was apparently curative in two recently reported cases.[13]

Aspergillosis

Five cases of disseminated aspergillosis with renal parenchymal involvement[2, 11, 14,

The rapidly increasing incidence of urinary infections caused by the more opportunistic fungi probably is due to a change in the resistance of the general population, the author says. He reviews some of these infections and points out the importance of prompt therapy.

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[23, 31] and five others with unspecified genitourinary tract invasion [16] have been described. These characteristically had numerous small abscesses composed of hyphae scattered throughout the kidney parenchyma. One specimen demonstrated invasion of small arterial walls by Aspergillus, producing a picture compatible with ischemic infarction.[31]

Primary renal aspergillosis has been reported in one patient with diabetes mellitus and adrenal insufficiency.[5] The patient underwent a nephrectomy and was found to have an obstructing cast of gray cheesy material which completely filled the renal pelves, calyces, and ureter. There was no evidence of parenchymal involvement.

Penicillinosis

There has been one reported case of unilateral pyelonephritis due to *Penicillium citrinum*.[12] This patient passed pink fungus balls in the urine. After eight years of symptoms, she succumbed to the disease. *Penicillium glaucum* was found to be the cause of cystitis in a patient who was noted to pass whitish greasy masses in the urine.[4]

Other Opportunistic Mycoses

Torulopsis glabrata was isolated in concentrations greater than 10^5 from the urine of seven patients with symptoms of urinary tract infection.[15] Moreover, one case each of pyelonephritis due to *Geotrichum* and *Sporotrichum* have been described in the older literature.[20]

Pathogenic Mycoses

Although renal involvement by *Histoplasma, Coccidioides,* and *Cryptococcus* has been occasionally noted in patients with disseminated disease, *Blastomyces dermatitidis* is frequently associated with severe disease of the entire genitourinary system.[18, 27, 30] Epididymitis and prostatitis are most common; however, 9 of 11 cases in a recent series demonstrated considerable renal involvement characterized by parenchymal abscesses, necrosis, and tubular casts composed of *Blastomyces* organisms.[27]

Actinomyces has been reported as the causative organism in 11 cases of pyelonephritis[6] and in one case of cystitis.[16]

Diagnosis

It is well accepted that the diagnosis of fungal disease must include not only the demonstration of the presence of the offending organism by smear or culture but also proof that the fungus is invading tissue and producing disease. Many diabetic patients, for example, may intermittently have small numbers of *Candida* organisms in the urine without urinary tract disease.

Patients having symptoms and signs of urinary tract infection in the absence of bacteriuria and those patients who either fail to respond or have an aggravation of their symptoms during antimicrobial therapy should be suspected of having a fungal infection. Every effort should be made to demonstrate fungi in the urine by direct examination and by culture. Although it is probable that *Candida* organisms are significant only when their concentration exceeds 10^5 per ml. or perhaps even 10^3 per ml. of urine, nothing is known concerning urine concentrations of other fungi.[15] Proof of infection should be confirmed by pyelography, cystoscopy, and biopsies of the bladder and kidney.

Disseminated fungal disease must always

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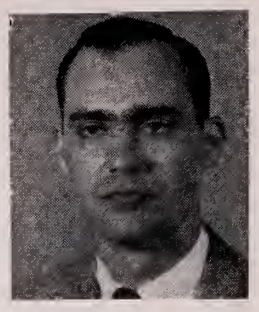

be ruled out. The renal parenchyma is most often involved in disseminated fungal infections whereas primary disease frequently spares the parenchyma and involves the pelves and calyces.

Treatment

The treatment of fungal infections leaves much to be desired. Penicillin is the treatment of choice for actinomycosis; however, experience in drug therapy of the opportunistic fungal diseases is limited. Amphotericin B is the only commercially available antifungal agent which may be of value for *Candida, Aspergillus, Torulopsis,* and the pathogenic fungal infections. The drug should be administered intravenously for infections of the kidney and may be given topically in cases of primary bladder involvement. Surgical excision of localized disease in combination with specific drug therapy offers the best chance of permanent cure.

Conclusion

It is apparent that fungal infections of the urinary tract will probably become more common. Patients who have symptoms of a urinary tract infection in the absence of bacteriuria or who fail to respond to the usual antibiotic therapy should be suspected of having fungal infections. Early recognition and prompt therapy with the newer antifungal agents is imperative for successful management.

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Methenamine Mandelate-Sulfamethizole Combination

Evaluation in Management of Urinary Tract Infections

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RESEARCH & INVESTIGATION

MANAGEMENT OF the acute uncomplicated case of urinary tract infection rarely presents a problem. In fact, a cure rate of 80 to 90 per cent can be achieved if adequate treatment with an appropriate antimicrobial agent is employed.[12] Chronic recurrent pyelonephritis, however, has often defied all chemotherapeutic measures. Long-term cure rates, even in cases without demonstrable obstruction or foreign body, have rarely exceeded 10 to 20 per cent.[4] Even if a cure is not achieved, there is increasing clinical evidence that prolonged suppression of active infection impedes the progress of renal functional loss.[11, 15] In the search for antibacterial agents more suitable for long-term therapy, the report of Garvey and Murray[8, 9] encouraged a trial of methenamine mandelate-sulfamethizole (Mesulfin) combination.

Sulfamethizole, a sulfonamide which is highly soluble, rapidly absorbed and excreted, and least inactivated of the sulfonamides, is most active at a low urine pH, a property which renders it uniquely suitable for this combination.

Methenamine is active when excreted in an acid urine by virtue of the liberation of formaldehyde, while mandelic acid escapes oxidation in the body and is therefore excreted in the urine as an acidifying agent. Lowering the pH of the urine has an inhibitory effect on bacterial multiplication. Studies dealing with the antibacterial effect of acidifying agents in urinary infections readily attest to their chemotherapeutic value.[6]

The purpose of this report is to present further clinical experiences with the combination of methenamine and sulfamethizole in the treatment of 37 patients with acute and chronic urinary tract infections.

Materials and Methods

Fifty-two adult patients with symptoms of acute or chronic pyelonephritis were treated with Mesulfin. Of these, 37 returned for adequate follow-up and are included in this report. Criteria for selection of patients included the presence of symptoms referable to the urinary tract and the demonstration of pyuria and/or significant bacteriuria.

Clean-voided midstream urine specimens were collected by house officers or trained nurses for culture and bacterial colony counts prior to, during, and after the treatment. The colony counts were performed by the pour-plate method immediately after collection. Bacteriuria was considered significant when a colony count of 100,000 or greater per milliliter of urine was obtained.

In acute pyelonephritis, a uniform dosage schedule of 1.0 gm. of Mesulfin initially and then 1.0 gm. four times a day was employed. The total dose was 20 to 50 gm. In cases of chronic pyelonephritis, the dosage ranged from 1.0 to 4.0 gm. per day, the total dose having been in the range of 40 to 1500 gm. Of the total 37 patients, 22 had chronic pyelonephritis, 15 of whom received Mesulfin only. The remaining seven had a favorable response to another antibiotic initially and were then started on Mesulfin for long-term suppressive therapy.

The authors evaluated the results of methenamine mandelate-sulfamethizole in the treatment of 37 patients with symptoms of acute or chronic pyelonephritis. No serious side effects or toxic reactions were observed and the drug was well tolerated by all patients. Response to therapy ranged from good to fair with one patient showing poor results.

TABLE 1.—*Clinical and Laboratory Data on Patients Treated with Mesulfin.*

Patient	Diagnosis	Daily Dose	Total Dose	1961-1962	Urine Culture	Bacterial Colony Count Neg.	<10^5	>10^5	Previous Therapy	Results
1.	Acute pyelonephritis	4 gm.	50 gm.	11/24	Enterococci			x	None	Good
				12/8		x				
2.	Acute pyelonephritis	4 gm.	50 gm.	3/19	*Aerobacter*			x	None	Good
				3/26		x				
3.	Acute pyelonephritis	4 gm.	50 gm.	3/9	*E. coli*		x		None	Good
				3/16		x				
4.	Acute pyelonephritis	4 gm.	50 gm.	3/2	Paracolon			x	None	Good
	Diabetes mellitus			3/9		x				
5.	Acute pyelonephritis	4 gm.	50 gm.	11/24	*E. coli*			x	None	Good
				12/1		x				
6.	Acute pyelonephritis	4 gm.	50 gm.	12/9	*E. coli*			x	None	Good
				12/22		x				
7.	Acute pyelonephritis	4 gm.	50 gm.	8/18	*S. aureus*		x		None	Fair
				9/1	Diphtheroids		x			
8.	Acute pyelonephritis	4 gm.	50 gm.	2/9	*S. aureus*		x		None	Good
				2/23	Diphtheroids	x				
9.	Acute pyelonephritis	4 gm.	30 gm.	7/28	*S. aureus*		x		None	Fair
				8/4			x			
10.	Acute pyelonephritis	4 gm.	50 gm.	10/13	*E. coli*			x	None	Good
				10/20		x				
11.	Acute pyelonephritis	4 gm.	20 gm.	7/18	*Proteus*			x	None	Good
				7/21		x				
12.	Acute pyelonephritis	4 gm.	50 gm.	12/8	Negative	x			None	Fair
				12/15		x				
13.	Acute pyelonephritis	4 gm.	50 gm.	3/16	*S. aureus*		x		None	Fair
				3/23	Diphtheroids	x				
14.	Acute pyelonephritis	4 gm.	50 gm.	9/29	*Aerobacter*			x	None	Good
	Diabetes mellitus			10/13	Paracolon	x				
15.	Acute pyelonephritis	4 gm.	50 gm.	7/19	*E. coli*			x	None	Good
	Anemia			7/26		x				
16.	Chronic pyelonephritis	4 gm.	50 gm.	3/23	*S. aureus*		x		None in	Fair
				3/30	Diphtheroids	x			2 years	
17.	Chronic pyelonephritis	4 gm.	50 gm.	12/26	Diphtheroids		x		None in	Fair
				1/3		x			6 months	
18.	Chronic pyelonephritis	4 gm.	50 gm.	12/27	Negative	x			None in	Fair
				1/4		x			2 years	
19.	Chronic pyelonephritis	4 gm.	50 gm.	9/8	*S. aureus*		x		None in	Fair
				9/22	Diphtheroids		x		10 months	
20.	Chronic pyelonephritis	4 gm.	50 gm.	10/20	Diphtheroids		x		None	Fair
				10/27		x				
21.	Chronic pyelonephritis	4 gm.	50 gm.	7/7	*Proteus*			x	Nitrofur-antoin	Fair
	Renal stones			7/21			x			
22.	Chronic pyelonephritis			7/19	*Klebsiella, E. coli*			x	Kanamycin	Fair
	Anemia, diabetes mellitus	4 gm.	58 gm.	7/27	*Pseudomonas**		x		Tetracycline	
23.	Chronic pyelonephritis	4 gm.	50 gm.	8/23	*E. coli*			x	None in	Good
				9/1		x			6 months	
24.	Chronic pyelonephritis	4 gm.	50 gm.	11/13	*Aerobacter*		x		Penicillin	Fair
				11/21		x			Tetracycline	
25.	Chronic pyelonephritis	4 gm.	90 gm.	9/20	*Proteus*			x	None in	Good
	Multiple myeloma			10/12	*Aerobacter*	x			1 year	

*Change in flora with treatment.

TABLE 1.—*Clinical and Laboratory Data on Patients Treated with Mesulfin (Continued).*

Patient	Diagnosis	Daily Dose	Total Dose	1961-1962	Urine Culture	Bacterial Colony Count Neg.	$<10^5$	$>10^5$	Previous Therapy	Results
26.	Chronic pyelonephritis	4 gm.	40 gm.	8/14	*E. coli*		x		None	Fair
	G. I. bleeding			8/21		x				
27.	Chronic pyelonephritis	2-4 gm.	424 gm.	11/6	*E. coli*		x		Kanamycin	Poor
	Renal stones			11/16				x	Coly-Mycin	
28.	Chronic pyelonephritis	4 gm.	50 gm.	9/6	*Proteus*		x		None in	Fair
				9/22	*S. aureus**		x		2 years	
29.	Chronic pyelonephritis	4 gm.	50 gm.	8/4	*S. aureus*		x		None in	Fair
				8/11		x			6 months	
30.	Chronic pyelonephritis	4 gm.	50 gm.	9/29	*Aerobacter, S. aureus,* Diphtheroids,		x		None in 2 years	Fair
				10/6	*E. coli** and Diphtheroids		x			
31.	Chronic pyelonephritis	4 gm.	50 gm.	3/16	*E. coli*		x		None in	Fair
				3/30		x			3 months	
32.	Chronic pyelonephritis	4 gm.	50 gm.	3/23	Diphtheroids		x		None	Fair
				3/30		x				
33.	Chronic pyelonephritis	4 gm.	50 gm.	10/28	Enterococci, diphtheroids		x		None in 1 year	Fair
				11/3	*S. aureus** and *Aerobacter*		x			
34.	Chronic pyelonephritis	4 gm.	250 gm.	11/31	*E. coli*			x	Kanamycin	Fair
				12/13		x				
35.	Chronic pyelonephritis	4 gm.	50 gm.	4/9	*Aerobacter*		x		None in	Fair
				4/16		x			3 months	
36.	Chronic pyelonephritis	1.0 gm.	360 gm.	8/9	*E. coli*			x	Nitrofurantoin, novobiocin, erythromycin, sulfadimethoxine, kanamycin, Coly-Mycin	Fair
(See Fig. 1. Patient still on Mesulfin.)				10/21	*E. coli, Proteus*		x			
				1/19/62	*E. coli*		x			
				6/26	*E. coli*		x			
37.	Chronic pyelonephritis	4.0 gm.	1500 gm.	6/29	*E. coli*		x		Kanamycin	Fair
(See Fig. 2. Patient still on Mesulfin.)					Diphtheroids		x		Tetracycline	
				10/30	*S. aureus,** diphtheroids			x		
				4/19	*E. coli,* diphtheroids		x			
				8/13	*E. coli*		x			

*Change in flora with treatment.

A hemogram, complete urinalysis, and a blood urea nitrogen determination were performed before and after therapy. None of the patients included in this study had significant azotemia. Urine pH was also determined on the urine specimens collected for culture during the course of therapy.

The results of the treatment are reported as good, fair, and poor. A good result is defined as symptomatic response within two to three days, cessation of pyuria, and a significant reduction of bacteriuria when present. A fair response is defined as diminished pyuria and bacteriuria but with no symptomatic response until the completion of at least one week of therapy. A poor response is defined as no improvement of symptoms or of urinary findings. Patients who experienced an exacerbation of their disease soon after a course of therapy are also included in this group.

Results

The major clinical and laboratory data in 37 patients treated with Mesulfin are pre-

TABLE 2.—*Frequency of the Various Organisms Isolated from Initial Urine Cultures.*

Organism	Acute	Chronic
E. coli	4	8
Proteus species	1	3
A. aerogenes	2	4
Paracolon	2	–
K. pneumoniae	–	1
Enterococci	2	–
S. aureus (Coagulase negative)	4	4
Diphtheroids	3	8

TABLE 3.—*Bacterial Colony Count of Initial Urine Cultures in 35 Patients.**

Organisms		$<10^5$ colonies/ml.	$>10^5$ colonies/ml.
Gram-positive			
	Acute	4	1
	Chronic	7	–
Gram-negative			
	Acute	–	9
	Chronic	8†	6

*Two additional patients had negative urine cultures.

†Three of this group had antibiotics just prior to Mesulfin therapy.

sented in Table 1. A number of organisms were isolated from the pre-treatment urine specimens. Tables 2 and 3 present the frequency with which the organisms were encountered and the incidence of significant bacteriuria on initial urine cultures. The therapeutic responses observed with reference to acuteness and chronicity of the disease as well as to etiologic organisms are shown in Tables 4 and 5.

Gram-negative organisms with a predominance of *Escherichia coli* accounted for the infections in two-thirds of the group. Fifteen of 23 quantitative urine cultures were positive for significant colony counts. Only eight patients infected with gram-negative organisms had initial urine colony counts of less than 100,000 organisms per milliliter. All eight were cases of chronic pyelonephritis, three of them having received antibiotics just prior to starting Mesulfin therapy. Response to therapy was good in 11, fair in 11, and poor in one patient.

Twelve cultures grew gram-positive cocci. Only one initial urine colony count revealed a significant bacteriuria. *Staphylococcus aureus,* coagulase negative, and diphtheroids were recovered in ten of these cultures. Even though the pathogenicity of these two organisms in the urine is questionable and they are often considered contaminants, the patients harboring them in the urine were included because of the presence of urinary

TABLE 4.—*Results of Therapy in Acute Versus Chronic Pyelonephritis.*

Response	Pyelonephritis—Acute	Pyelonephritis—Chronic
Good	11	2
Fair	4	19
Poor	0	1
Total	15	22

TABLE 5.—*Results of Therapy with Reference to Etiological Organisms.*

	Good	Fair	Poor
Gram-negative	11	11	1
Gram-positive	2	10	–
Total*	13	21	1

*Two patients had negative initial urine cultures.

tract symptoms and pyuria. Two of the 12 patients infected with gram-positive cocci had prompt symptomatic improvement and disappearance of pyuria with Mesulfin therapy. The remaining ten patients had a slow response.

An analysis of the therapeutic response with relation to acuteness and chronicity of pyelonephritis revealed a good response in 11 of 15 patients with acute pyelonephritis and a fair response in most of the cases of chronic pyelonephritis with respect to the criteria established for this study.

Urine pH during the course of therapy was found to be in the range of 4 to 6 on repeated determinations. The only side effect observed was urine turbidity, which was noted in all patients.

Emergence of resistant organisms or a change in flora during the course of therapy was not a major problem, having occurred only in five patients. All five of these were in the group with chronic pyelonephritis, and only two showed development of resistant strains.

Discussion

The evaluation of the therapeutic merits of an antibiotic or a drug combination in the management of chronic pyelonephritis is a difficult task. This is as much due to lack of definitive diagnostic criteria as it is to inadequate parameters for evaluation of response to therapy. The diagnosis and treatment of the acute uncomplicated case, however, is simple and methods for evaluation of response to therapy are relatively uniform and reliable.

In chronic pyelonephritis, particularly in the asymptomatic form, the high degree of correlation that has been found to exist between significant bacteriuria and active pyelonephritis has engendered much confidence in the diagnostic value of bacterial colony counts of the urine.[7, 13, 14] It has also been suggested that reduction in the magnitude of the bacteriuria has a salutary effect not only on symptoms but also on the progress of this potentially fatal disease.

In this study the results observed in the treatment of acute pyelonephritis were favorable. This was anticipated since during the last decade trials with sulfamethizole alone had been encouraging.[1, 2, 3, 5, 10, 16, 17] The use of a sulfonamide singly or in combination with an acidifying agent is therefore considered an effective and relatively safe therapy in acute urinary tract infections.

Of greater interest are the beneficial effects observed with Mesulfin in the treatment of chronic pyelonephritis. The symptomatic relief achieved coincident with reduction in bacterial colony counts and pyuria was definite although slow. Mesulfin appeared effective in preventing recurrent attacks of chills, fever, and systemic symptoms in patients with chronic pyelonephritis. Some of the patients had experienced such recurrent episodes prior to Mesulfin therapy. Figs. 1 and 2 illustrate the effect of long-term Mesulfin therapy in two such patients (cases 36 and 37 respectively).

No patients with severe renal insufficiency were included in this study and none of the patients treated developed any evidence of acidosis secondary to Mesulfin therapy.

The antibacterial activity of sulfamethizole appears to be enhanced by the lowering of the urine pH by methenamine mandelate.

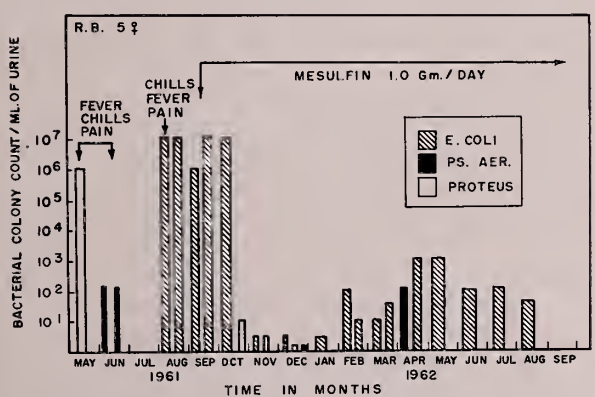

Fig. 1. The effect of Mesulfin in a 5-year-old girl with a recurrent pyelonephritis. Symptomatic improvement occurred with reduction of bacteriuria.

The relative nontoxicity of Mesulfin and the infrequency of emergence of resistant organisms with its use were observed in this study.

Turbidity of the urine was present in all patients but had no effect on the therapeutic response. This turbidity appeared to be related to pH: the lower the pH the greater was the turbidity. Qualitative analysis of the sediment showed it to contain ammonium, urate, calcium, oxalate, and phosphate ions.[6]

The favorable results reported in this study indicate that methenamine mandelate-sulfamethizole combination should be a useful addition to the list of agents used in the long-term suppressive therapy of chronic pyelonephritis. Moreover, it appears to have an advantage over other agents because of its suppressive antibacterial effect in the interstitial tissue of the kidneys as well as in the urine.

Summary

Methenamine mandelate-sulfamethizole combination (Mesulfin) was evaluated in the treatment of urinary tract infections. Of the 15 patients with acute pyelonephritis, a good response was obtained in 11 and a fair response in 4. Only two patients with chronic pyelonephritis had a good response to therapy while 19 had a fair response. One patient with chronic pyelonephritis due to *E. coli* had a poor response with an acute exacerbation of symptoms while on Mesulfin therapy.

No serious side effects or toxic reactions

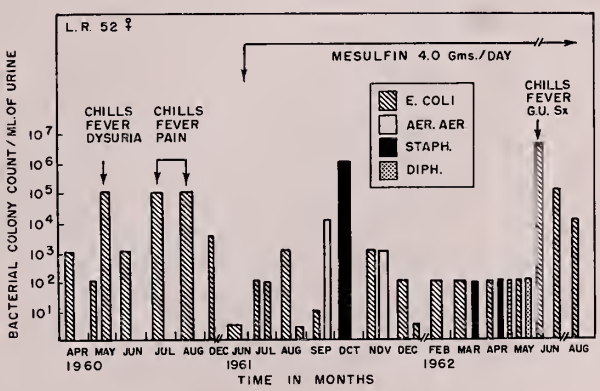

Fig. 2. Symptomatic improvement and reduction of bacteriuria achieved with prolonged Mesulfin therapy in a 52-year-old woman with recurrent pyelonephritis. A relapse occurred when the drug was temporarily discontinued in June.

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were observed. Mesulfin was well tolerated by all patients.

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Doubt, Disbelief Accompany Clinical Progress

Not infrequently, it takes years and sometimes decades of widespread clinical experience to evaluate the relative merit of a drug in given conditions, according to Dr. Theodore G. Klumpp of New York City, president of Winthrop Laboratories. "From such long experience," he told an audience at the Conference of Professional and Scientific Societies in Chicago June 28, "a medical consensus generally emerges but even then some qualified physicians refuse to go along with their colleagues. History teaches that authoritarian bodies often have been guardians of orthodoxy rather than champions of progress. Medical experts rejected Jenner's smallpox vaccine, Pasteur's anthrax vaccine, Lister's theory of antisepsis and Semmelweis' discovery of the cause of childbed fever. Cod liver oil was once rejected as worthless by the Council on Pharmacy and Chemistry of the American Medical Association. When Prontosil, the first sulfa drug, was introduced in the United States, it was greeted as another quack remedy by an outstanding American authority on infectious diseases. In the early 1930's, the same authority dismissed early English reports on penicillin as incredible and refused to employ for clinical testing a culture of *Penicillium* that had been brought to him by one of his associates. He poured it down the sink."

Disorders of Coagulation Associated with Surgery of the Prostate

TERRY D. ALLEN, M.D.

CASE REPORTS

THE IMPORTANCE of the blood clotting mechanism is never appreciated quite as much as when it fails during operative procedures. While such abnormalities have followed a variety of surgical efforts, notably hepatic, pancreatic, and pulmonary, the urologist is most keenly aware of those associated with surgery of the prostate, particularly transurethral prostatic resections. Recent investigations of fibrinolytic mechanisms in surgery[5, 14, 17, 20] and experimental studies of hematologic factors in irreversible shock[6, 7, 16] have added much to the understanding of the syndrome of sudden unexplained and uncontrolled bleeding associated with prostatic surgery but they have not lessened the seriousness of this complication.

Three cases are presented herein to illustrate some of the clinical syndromes which may be encountered, to discuss the current concepts of the mechanism of this interesting phenomenon, and to review the prophylactic and active treatment of the disorder.

Case Reports

Case 1: A hematologist hospitalized a 58-year-old white man for evaluation of a hemorrhagic diathesis of two weeks' duration. The patient was easily bruised and had ecchymosis and bleeding from the gums following minimal trauma. The prostate was indurated with a hard fixed right lobe. Acid phosphatase was 15.2 and 26 Bodansky units and roentgenograms showed multiple metastatic deposits in the bone compatible with prostatic carcinoma. A urologic consultation was obtained, and the patient was started on stilbestrol, 10 mg. daily. He was discharged from the hospital by the hematologist, with a notation that the clotting mechanism had tested out "normal" and that the cause of the hemorrhagic diathesis was obscure. The patient's symptoms promptly and dramatically subsided on stilbestrol therapy, and he was readmitted three months later for a bilateral orchiectomy at which time he was completely asymptomatic. Platelet count was 638,590, bleeding time 1 min., and clotting time 3 min., 30 sec. He was released after an uneventful surgical procedure and postoperative course.

Comment: This case illustrates that spontaneous hemorrhagic manifestations may occur in prostatic carcinoma even in the absence of surgical procedures upon the gland. It may be argued that the mechanism was never conclusively established in this patient but unfortunately the true nature of the disease process was not appreciated except in retrospect. The typical clinical picture, failure to find another explanation, and dramatic response to stilbestrol warrants its inclusion in this series.

Case 2: An 85-year-old white man entered the hospital with a two-year history of progressive obstructive prostatic symptoms and three episodes of hematuria. Originally, the clotting time was 20 min., 58 sec., but was 16 min., 36 sec. and 15 min., 30 sec. on repeat examination. Prothrombin time was 62 per cent, platelet count was 636,270. He underwent a transurethral resection of 28 gm. of benign tissue, but on two occasions in the postoperative period was unable to void upon removal of the catheter. Consequently, about two weeks after his original surgery an additional 12 gm. of benign tissue was resected transurethrally. In his enthusiasm to clean out the prostatic fossa, the surgeon entered the capsule in several areas. Upon return to his room, the patient began to bleed moderately and steadily. He was returned to surgery where cystoscopy failed to disclose any arterial bleeders. Because there were no blood clots in the bladder, 5 cc. of blood was drawn, placed in a tube and taped to the bed. It was observed to clot normally, but complete liquefaction of the clot occurred in two and one-half hours. Fibrin-

Excessive fibrinolytic activity may initiate uncontrolled bleeding associated with prostatic disease. The mechanism and treatment of this syndrome are discussed.

ogen was 200 mg. per 100 cc., platelet count 262,800, and prothrombin time was 62 per cent. He was treated with five units of blood (three of them fresh whole blood), one unit of fresh frozen plasma, vitamin C, and Mephyton. He continued to bleed for about 12 to 18 hours but this gradually subsided and, by 24 hours, bleeding had ceased. During the next few days his urinary output fell to a low of 500 cc. for 24 hours and his blood urea nitrogen (BUN) rose to a high of 96 mg. per 100 cc. He recovered from this mild renal shutdown, but suffered a transient stroke to his right side. He was finally discharged voiding well, after approximately six weeks of hospitalization.

Comment: It would have been easy to pass this case off as "excessive sinus bleeding" had not the surgeon been alerted by the absence of blood clots in the bladder of a patient plagued with annoying post-resection bleeding. The simple observation of a tube of the patient's blood unequivocally established the diagnosis when liquefaction of the clot at two and one-half hours occurred. Treatment, consisting principally of blood replacement, allowed recovery.

Case 3: One year previously, a 64-year-old white man was diagnosed by needle biopsy as having adenocarcinoma of the prostate. He had been treated in the interim with stilbestrol. He entered the hospital with lower tract obstructive symptoms and 110 cc. residual urine which was grossly infected. He had also had intermittent hematuria which had responded to occasional Premarin injections given by another physician. The prostate was hard (3+) and fixed, and small ecchymotic areas were present on the back of the hands; these were not appreciated at the time of admission. Acid phosphatase was normal. The BUN was 17 mg. per 100 cc., and hematocrit was 39. An excretory urogram revealed grade 1 ectasia of the upper urinary tracts bilaterally, secondary to obstruction in the region of the ureterovesical junction. It was felt that he had sufficient obstruction to warrant a transurethral resection of the prostate as a primary procedure with the idea of performing an orchiectomy in the postoperative period. Although he was apprehensive, he was reassured and taken to surgery for a transurethral prostatic resection. Difficulty was encountered in inserting the resectoscope through the hard prostatic urethra, and the resection was plagued with technical problems and a small bladder capacity, thus extending the length of the procedure to about 1 hour and 45 minutes. Near the conclusion of the resection, sudden excessive bleeding was noted. No arterial bleeders could be identified and as the intravesical pressure rose, the bleeding ceased, only to recur briskly upon emptying the bladder. Traction on the catheter failed to exert any appreciable effect. He received two units of blood in the operating room and was returned to his room where 5 cc. of blood was drawn, placed in a tube, and taped to his bed. The blood failed to clot. Platelet count was 300,000; fibrinogen was 100 to 200 mg. per 100 cc. and Quick's prothrombin test failed to produce a clot. The patient continued to bleed through his catheter without forming clots. He developed oozing from puncture wounds and cut-down sites, massive ecchymoses over his body, and bleeding from the gums. He was given prednisolone, Benadryl, vitamin K, vitamin C, Polybrene, Cedilanid, oxygen, Premarin, eight units of blood, and two units of fibrinogen. Following this, his fibrinogen was reported as less than 100 mg. per 100 cc. By 24 hours after surgery, the patient was moribund. His blood now clotted and his bleeding had slowed considerably, but he was jaundiced and it was necessary to maintain his pressure with Aramine. He died 48 hours after surgery. No autopsy was obtained.

Comment: This is an example of the syndrome in its most severe form. From the history of hematuria responding to Premarin and the presence of small ecchymotic areas on the back of the hand, it is likely that even prior to surgery, excessive fibrinolytic activity was present. Failure to be aware of this was compounded by technical problems at the time of surgery, and as a final *coup de grace,* time spent looking for bleeders served only to wash more activating enzymes into the circulation. In retrospect, treatment probably was inadequate, reflecting the inexperience of the operator in dealing with this problem. However, the prognosis has been uniformly poor in the presence of blood which fails to clot at all.

Mechanism

A review of the clotting mechanism reveals that the human body is endowed with an ingenious system designed to preserve blood volume in the face of disruption of the vascular tree. The efficiency of this system is such that the clotting time can be lowered so rapidly that in phlebotomies the blood removed last clots first. Without a counter system, however, the vessels of the body would ultimately become so occluded with clots resulting from minor trauma that circulation would cease. To prevent this, nature has graciously provided the body with the fibrinolytic system, whose function it is to digest away clots after they have served their purpose, thus re-establishing circulatory continuity. This fine balance between blood clotting and clot liquefaction is illustrated in Fig. 1.

The fibrinolytic system has been the subject of increasing attention in recent years, and is centered around a proteolytic enzyme, fibrinolysin or plasmin. The precursor of

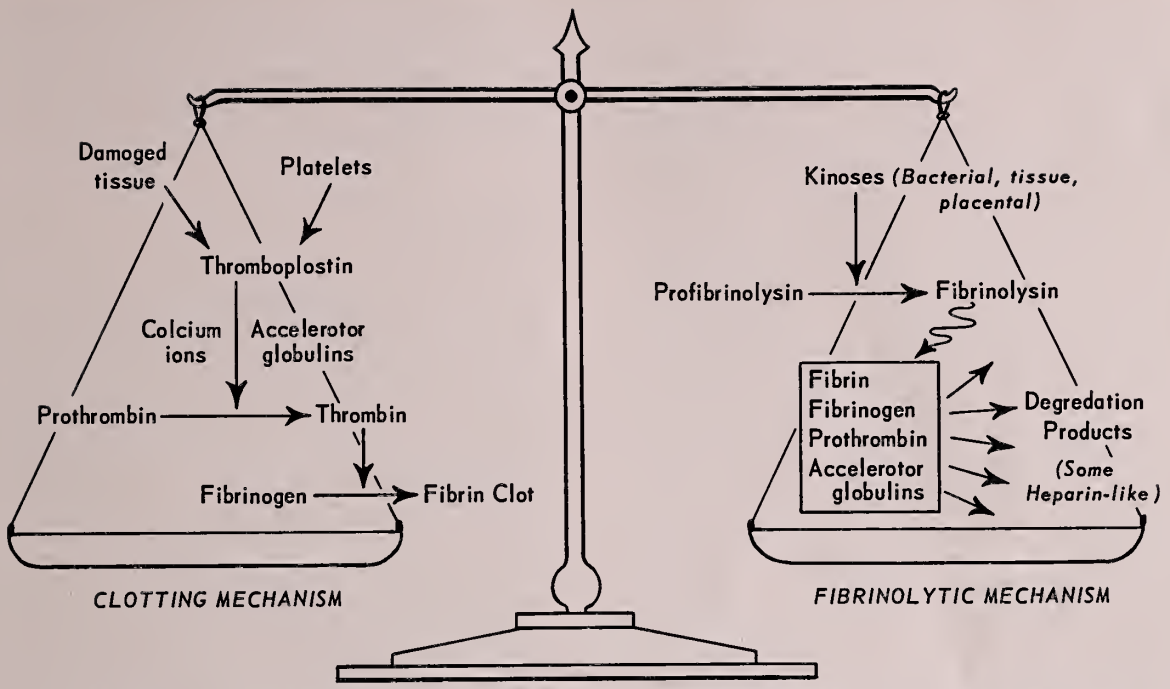

Fig. 1. The balance which exists between the clotting mechanism on the left and the fibrinolytic mechanism on the right is illustrated.

this enzyme, profibrinolysin, is synthesized by the liver and converted to fibrinolysin by a variety of activators known as kinases. Fibrinolysin attacks not only the fibrin clot, but also a number of clot precursors, particularly fibrinogen, depending to some extent upon the particular kinase which has led to its activation.[3] Ordinarily, fibrinolysin is kept rather inactive in the plasma by the binding power of an anti-enzyme, antifibrinolysin, but under certain circumstances its concentration may be sharply increased by activation of its precursor, whereupon fibrinolysin becomes clinically effective. This activation occurs physiologically in response to the formation of clots, but it may be seen also in a variety of pathologic states, particularly whenever trauma and shock are common denominators. Thus severe burns, asphyxiation, anaphylactic and endotoxic shock, transfusion reactions, adrenalin infusions, and even severe exercise and anxiety[2] are among a long list of insults known to produce increased fibrinolytic activity. The role of fibrinolysin in the reproductive process, menstruation, placental separation, lysis of pneumonia, and a variety of other states is interesting but beyond the scope of this article.

The exact sequence of events leading to the hemorrhagic diathesis seen in prostatic disease has not been conclusively clarified, but mounting evidence appears to indict two factors which cannot be completely divorced, namely excessive circulating fibrinolysin and a concentration of clot precursors inadequate to compensate for it. Low concentrations of clotting factors probably play a lesser role, since they are often within normal limits even in the face of frank bleeding. Even when low values are present, they may simply be a manifestation of destruction by fibrinolysin rather than of any separate pathologic process. This coincides with the conclusion of Phillips and associates[14] in investigations of acquired hypofibrinogenemia, but is by no means universally accepted. Stefanini,[17] in studying the spontaneous hemorrhagic manifestations in a series of patients with carcinoma of the prostate failed to find evidence of increased fibrinolysin, but found instead consistently low platelet counts and evidence of an accelerated clotting mechanism. He concluded that the basic process is one of intravascular clotting with resultant depletion of the clotting factors, and that fibrinolysin activation occurs late and is of lesser importance.

In spite of Stefanini's work, most of the evidence supports the prominent role of fibrinolysin. In almost every reported case, liquefaction of formed blood clots has been an important feature, and occasional reports illustrate the ability of the patient's serum

to liquefy even a normal clot.[8] Tagnon and associates[19] found increased fibrinolytic activity in 6 of 48 cases of prostatic carcinoma studied, and in a somewhat analogous situation, Grossi and others,[5] in a study of 53 patients with cirrhosis undergoing portacaval shunt, found spontaneous fibrinolytic activity preoperatively in 60 per cent of them.

The role of the prostate in upsetting this fine balance between clotting and clot lysis is not clear, but since equivalent degrees of trauma to other organs are not often associated with such coagulation defects, the prostate itself may be a key factor in the development of the syndrome. Prostatic tissue, particularly carcinomatous prostatic tissue, is a rich source of proteolytic enzymes, tissue kinases, and thromboplastins. It might be expected that infusion of these substances into the circulation could be associated with striking systemic alterations. That such may be the case is suggested by the work of Verrilli and associates[21] who injected prostatic extracts intravenously in dogs, producing profound shock and even death. The theoretical pathways by which the clotting mechanism might be altered as a result of prostatic surgery are diagrammatically illustrated in Fig. 2.

Clinical Picture

The hemorrhagic diathesis associated with prostatic disease fortunately confronts the urologist infrequently, but, in view of Lombardo's[8] report of 5 cases in 302 consecutive resections, the problem may be encountered more frequently than has been appreciated. Perhaps some difficulty in recognizing the complication has occurred as a result of the great variation in its clinical picture.

Urologically, spontaneous hemorrhagic manifestations are almost exclusively limited to patients with prostatic carcinoma. They consist of ecchymoses, bleeding from the gums, hematuria, or gastrointestinal bleeding. Oozing from needle puncture wounds may be seen. Mild cases may be overlooked until minor procedures such as orchiectomy

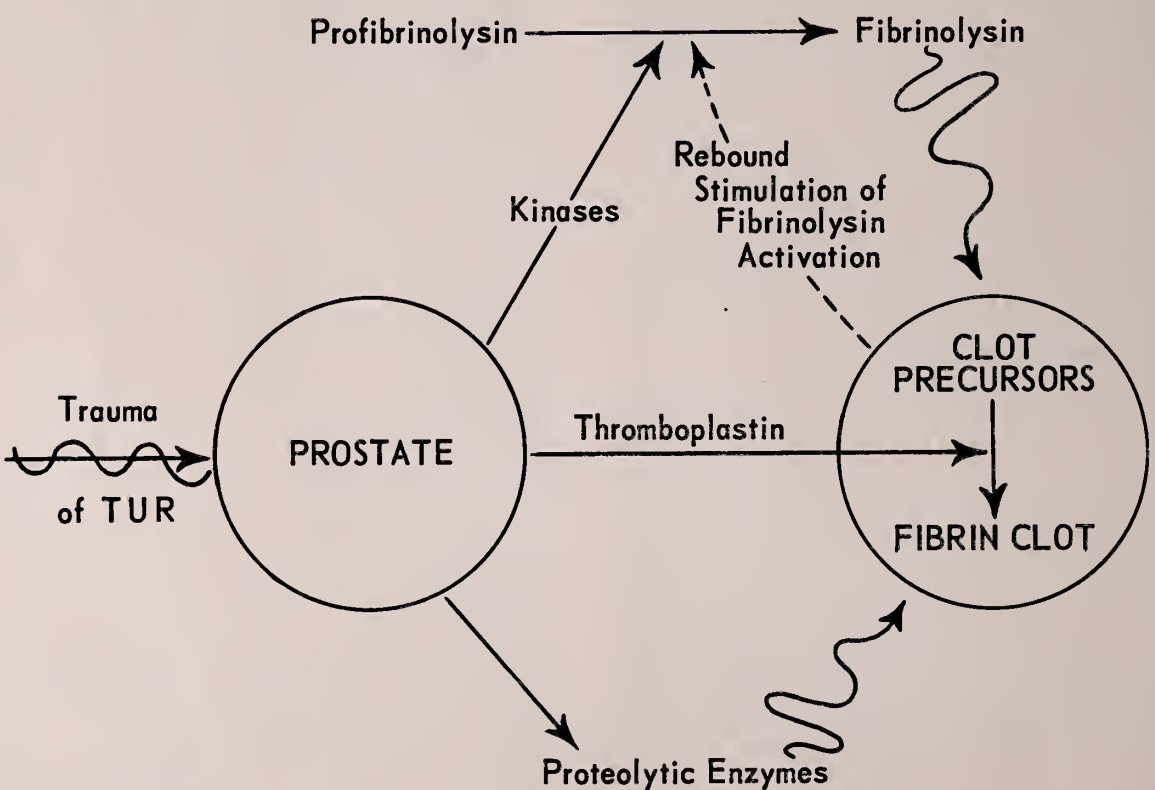

Fig. 2. Diagrom illustroting the three routes by which prostotic surgery might precipitate o bleeding crisis. Below, proteolytic enzymes responsible for semen liquefaction and lysis of the cervicol plug might directly hydrolize clots. Center, infusion of thromboplostins might precipitate introvosculor clotting with rebound fibrinolytic activation. Above, infusion of kinases might activote the fibrinolytic system directly.

spotlight them by activating sufficient fibrinolysin to produce uncontrolled oozing from the wound or massive ecchymoses around the operative site. Major surgical procedures in such patients could be disastrous.

The development of the syndrome in conjunction with surgical procedures has been reported in transurethral resections of bladder carcinoma and in suprapubic prostatectomy, but the vast majority of the cases has followed transurethral resections of the prostate gland for both benign and malignant lesions, suggesting again that infusion of prostatic enzymes into the circulation may indeed play a major role in the precipitation of the clinical picture. Although surgery initiates the chain of events ultimately leading to the coagulation defects, serial measurements of fibrinogen concentrations[9] indicate that the fall in clot precursors may not reach its maximum until four hours after surgery. Thus, not uncommonly, bleeding may not become clinically evident until several hours after the operative procedure has been completed. As a generalization, the earlier the bleeding appears, the more serious the disease process.

It is usually this uncontrolled bleeding from the prostatic bed that alerts the surgeon to the possibility of a hemorrhagic diathesis. This may range from mild annoying bleeding, continuing or recurring over a period of days or even weeks, to the explosive and dramatic massive exsanguination. Clots may be present in the bladder but the absence of clots in the face of troublesome bleeding is a significant clue when seen. In severe cases uncontrolled bleeding from the prostatic fossa may be followed in rapid succession by evidences of a fibrinolytic crisis as manifested by oozing from needle puncture wounds, massive ecchymoses, gastrointestinal bleeding, circulatory collapse, and death unless the process is checked by immediate and effective therapy.

Routine hematologic studies are of limited value in predicting or prognosticating this syndrome. Preoperatively they may be completely within normal limits, though minimal increases in clotting time and low to low-normal fibrinogen levels and platelet counts are not unusual. Levels of fibrinogen below 200 mg. per 100 cc. are of particular significance since Grossi and associates,[5] in their study of cirrhotic patients, found that spontaneous fibrinolytic activity was always present under those circumstances. The presence of such activity preoperatively, as demonstrated by whole clot or euglobulin clot lysis, indicates that the patient is a likely candidate for gross coagulation defects should the contemplated surgery be carried out. Unfortunately, the converse is not necessarily true, since negative tests for fibrinolytic activity are no assurance that bleeding problems will not develop.

Fibrinolysin levels increase during the surgical procedure and bleeding may first become evident at this time. Patients with spontaneous fibrinolytic activity preoperatively do not show this same rise,[5] possibly because fibrinolysins are already maximally activated. With the increase in fibrinolytic activity, there is a gradual fall in the concentration of clot precursors and a severe depletion of these factors may develop. Because this lags behind the clinical picture, however, alarming hemorrhage may be seen while clotting factors are still within limits of normal. A systemic blood specimen usually will clot, though the clot is "softer" and subsequently undergoes liquefaction, but in severe cases, the blood may fail to clot at all.

The surgeon, unfamiliar with this complication and faced with the task of trying to diagnose an unexplained bleeding problem, may be confused by the battery of hematologic tests available, and by their interpretation. It should be remembered that measurements of clot precursors are a poor index to the disease[20] and that the key to diagnosis rests upon the demonstration of fibrinolytic activity. Though euglobulin clot lysis tests are quicker, simple observation of a clot of the patient's blood for the development of subsequent liquefaction is an easy method which can be performed anywhere. A normal blood clot will not liquefy even after several days, but in the presence of fibrinolytic activity, lysis may appear within 30 minutes to several hours. The patient's own clot is more easily lysed since it is composed of depleted or altered clotting factors, but when high levels of fibrinolysin are present, the patient's serum will lyse even normal clots.

Prophylaxis

It would be fortunate indeed were the surgeon able to predict with certainty the development of such a complication and thus compensate for it before it occurred. Unfor-

Dr. Allen, who is with the Division of Urology of the University of Texas Southwestern Medical School and Parkland Memorial Hospital, Dallas, presented this paper before the South Central Section of the American Urological Association in November, 1962.

tunately this is seldom possible. Nonetheless, an occasional candidate may arouse suspicion of impending disaster, particularly patients with prostatic carcinoma or cirrhosis who present with ecchymoses or histories of recurrent bleeding. Such patients should be carefully evaluated for the presence of adequate clotting factors and the absence of spontaneous fibrinolytic activity.

When an imbalance in the clot-clot lysis mechanism seems imminent, several avenues are available to prepare the patient for surgical procedures. Most are based upon methods designed to increase the concentration of clot precursors. Estrogens increase fibrinogen levels and may produce dramatic coagulation responses in patients with prostatic carcinoma. Lombardo[9] reported that fibrinogen levels were maintained better also in patients given toluidine blue compounds preoperatively. A high fat diet[12] appears to improve the clotting mechanism, but the manner by which it does this is not completely clear. Transfusion of specific clotting factors may be indicated and there is a sizable list of vitamins and other medications designed to protect or replenish specific clot precursors. A slightly different approach is taken by the use of epsilon aminocaproic acid (EACA) about which more will be said later. This compound, by blocking the proteolytic action of fibrinolysin, allows the clotting factors to accumulate spontaneously.

Lastly, it appears logical to suppose that gentleness in instrumentation, reduction of operative time to a minimum, and avoidance of venous sinuses during transurethral resection might reduce the incidence of such a bleeding crisis. However, rational though such a statement may be, it is difficult to demonstrate statistically.

Treatment

The aims of treatment are to replenish clot precursors, block fibrinolytic activity, and maintain homeostasis with blood, fluids, and supportive measures. Blood plays a critical role in this effort since, not only is it important in maintaining circulatory volume, but it supplies necessary clotting factors as well. For the latter role, fresh whole blood should be emphasized since many of these clotting factors deteriorate rapidly upon storage. Other sources for clot precursors are fresh frozen plasma, serum albumin, and packaged fibrinogen. Fibrinogen is of particular value since it will neutralize fibrinolysin directly. It is supplied in 1 gm. units and is expensive. Dosage depends upon individual requirements, but in severe bleeding problems, probably 5 gm. should be given initially with more to follow as needed. Other compounds such as Premarin, toluidine blue, Polybrene, Mephyton, and protamine improve the clotting mechanism, but probably are of limited value in a hemorrhagic crisis.

Recently, great interest has arisen in a new compound, EACA, a synthetic amino acid which has been shown to block the activation of profibrinolysin to fibrinolysin as well as to block fibrinolytic activity directly.[15] The advantage of such a drug is obvious since it would allow a rapid build up of clotting factors which might otherwise be destroyed as fast as they could be infused. While still experimental, EACA has had a number of clinical trials[5, 10, 13, 15, 18] in the treatment of spontaneous and surgically precipitated bleeding abnormalities associated with hepatic, prostatic, pulmonary, and pancreatic surgery. The results so far have been impressive. In clinical trials, 4 to 6 gm. have been used initially with maintenance doses ranging from 2 gm. daily[5] to 6 gm. every four hours.[13] No toxic effects have been noted in humans, though subendocardial hemorrhages have been reported in dogs following prolonged administration of large doses. Steroids, too, have a direct antifibrinolytic effect and may thus be of value.

Other modes of therapy are less well established, but Neft and associates[12] reported good results with the use of intravenous fat emulsions. On the grounds that intravascular clotting may be a precipitating factor, heparin might have a theoretical place early, but its use after the appearance of uncontrolled bleeding would seem somewhat hazardous. Nonetheless, Gormsen and Hansen[4]

reported a case in which heparin for a pulmonary embolus resulted in the cessation of troublesome bleeding from the prostatic bed in a patient who had undergone a suprapubic prostatectomy.

Prognosis

While many examples of this syndrome are mild and improve spontaneously, a hemorrhagic crisis is a serious condition. In Lombardo's[8] report of five such cases, there was only one survival. With better understanding of the pathologic process and the development of new drugs, the outlook for the future should be brighter.

Summary

Uncontrolled bleeding associated with prostatic disease may be caused by a defect in the coagulation mechanism, primarily as a result of excessive fibrinolytic activity. Three cases are presented and current concepts of the mechanism of the syndrome and its treatment are discussed.

The author wishes to thank Dr. Harold O'Brien for the use of Case 1, Dr. Foster Fuqua for the use of Case 2, and Drs. Ellen Loeb and Harry Spence for their helpful suggestions.

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Good Riddance to "Good Old Days"

Anybody who wishes for the return of "the good old days" so as to get things cheaper will realize that the price he has to pay is too great, says an article, "An Rx for a Dime," in New York State *General Practice News*, November-December, 1963. This was the age of rickets and deformities. Maternal mortality was high. Each infant born had to struggle just to survive. Each adult was lucky if he could live to the age of 40. The great scourges were tuberculosis, pneumonia, typhoid fever, diphtheria, measles, scarlet fever, smallpox, mastoid and brain abscess, meningitis and many other infections too numerous to mention, but all conquered now. Want to go back to "the good old days"? Would you want the penny prescription of yesteryear for the dollar ones of today, or the life of a century ago for the luxuries of today? Would you trade modern wonder drugs for old-fashioned, home-spun medicaments? People should know that particularly in health matters, as elsewhere "cheap is cheap."

Clinicopathologic Conference

ROBERT E. ROSSMAN, M.D., JOHN M. KNOX, M.D.,
ROBERT G. FREEMAN, M.D., AND MONTAGUE LANE, M.D.

This is the sixth in a series of clinicopathologic conferences from Baylor University College of Medicine in Houston. Guest editor of the series is Dr. S. H. Frazier. Participating in the conference this month are Dr. Robert E. Rossman, assistant in dermatology; Dr. John M. Knox, associate professor of dermatology and associate professor of dermatopathology; Dr. Robert G. Freeman, assistant professor of dermatopathology in the Department of Dermatology and assistant professor of pathology; and Dr. Montague Lane, associate professor of pharmacology and medicine.

DR. ROBERT E. ROSSMAN: This 49-year-old Negro woman was first examined in the Skin Clinic on Nov. 28, 1960, upon referral from the local Venereal Disease Clinic. Several hundred umbilicated, nodular, and crateriform lesions were scattered over her scalp, face, shoulders, and anterior chest (Fig. 1). Many darkly pigmented and confluent varioliform scars were interspersed among the active nodules. According to the patient, the initial eruption appeared spontaneously on normal skin approximately three months before her first Clinic visit. The original lesions occurred on the scalp and ears and gradually spread to adjacent skin. Spontaneous involution of a few nodules left residual atrophic scars. Progressive hoarseness had been present for several months. There was no history of any similar episode or of any known familial skin disorder.

Unknown quantities of Miles Nervine had been ingested for "nervousness" caused by the conspicuous eruption, but the patient denied having taken any other drug. There had been no known exposure to arsenic or other systemic carcinogen.

The patient had been seroreactive for syphilis for 15 years. Treatment for late latent syphilis had been given at the Venereal Disease Clinic of the Houston City Health Department in 1946. A low titer reactive serologic test for syphilis persisted, and the patient received 2,400,000 units of penicillin intramuscularly from private physicians in 1952 and again two months before her initial Clinic visit.

Physical Findings.—The pertinent physical findings were limited to the skin with the exception of small nodules involving the right vocal cord observed during laryngoscopy. Skin lesions in several stages of development were present (Fig. 2). The earliest visible change appeared to be a tiny follicular papule which rapidly progressed to a larger dome-shaped nodule. Umbilication resulted in a crater containing a pigmented hyperkeratotic center. More advanced and involutional changes were characterized by gradual flattening and subsequent extrusion of the keratotic plug from the central crater. Many pigmented atrophic scars with overhanging crenelated borders marked the sites of previously active lesions. In several areas individual lesions had coalesced to form large irregular plaques. A few nodules were present on the external genitalia. The distal phalanx of the left middle finger was tender, grossly deformed, and draining foul purulent material. No lymphadenopathy, hepatosplenomegaly, or evidence of weight loss was noted.

Laboratory Results.—Results of a complete blood count, urinalysis, blood urea nitrogen (BUN), and fasting blood sugar were normal. The VDRL serologic test was reactive at a 1:2 Cq dilution. The initial serum bromide level was 98 mg. per 100 ml. Arsenic was not detected in specimens of hair and nails. Routine chest roentgenograms were normal, but X-rays of the hands revealed a lytic area in the distal phalanx of the left middle finger and destruction of the associated interpha-

Fig. 1. Appearance of the patient several months after onset of the eruption.

langeal joint. Results of skin testing with tuberculin, coccidioidin, histoplasmin, and blastomycin were negative. Bronchoscopy, sigmoidoscopy, and cystoscopy were negative. Repeated laryngoscopy revealed little change in the nodules on the vocal cord over a period of several months. The original skin biopsy revealed pseudoepitheliomatous hyperplasia compatible with bromoderma, although squamous cell carcinoma could not be ruled out. No organisms were seen in the microscopic sections.

Clinical Course.—Multiple skin biopsies were taken of lesions in various stages of development. Repeated laryngeal biopsy several months later was followed by a partial laryngectomy.

Six weeks after discontinuing Nervine, the serum bromide level was normal and many lesions had involuted; however, new skin nodules continued to appear. A tendency for individual lesions to fuse into large plaques became apparent on the trunk. New hyperkeratotic craters appeared on the palms, fingers, and soles, as did subcutaneous nodules over the dorsa of the fingers.

The course of this patient has continued

Fig. 2. Close-up view of the patient's back illustrating lesions in all stages.

unabated for the past two and one-half years with some nodules resolving spontaneously over a period of a few months only to be replaced by other similar lesions in adjacent skin. Her general health has remained excellent except for anxiety created by the cosmetic handicap of extensive residual scarring.

Differential Diagnosis

DR. JOHN M. KNOX: This is an interesting and unusual patient and several diagnostic possibilities should be considered, including halogen drug eruption, metastatic carcinoma, infectious granulomata, and multiple keratoacanthomas.

The acute onset, distribution pattern, gross and microscopic descriptions of the lesions are certainly compatible with bromoderma. This is well supported by the elevated serum bromide level of 98 mg. per 100 ml., although the minimum bromide concentration prerequisite for the appearance of such lesions varies from patient to patient. It should be kept in mind that most patients with severe bromide intoxication do not have associated skin lesions in spite of serum bromide levels exceeding 200 mg. per 100 ml. Patients' drug histories are often unreliable, yet the available information indicated that the actual drug ingestion took place after the eruption began to produce cosmetic problems. The chief evidence against this diagnosis is, however, the continued appearance of many new lesions after the drug was discontinued. The rapid fall of serum bromide concentration and the return to normal levels in six weeks would confirm cessation of drug intake.

Metastatic carcinoma should also be considered. Undoubtedly something suspicious

was found on the laryngeal biopsies which preceded the partial laryngectomy, and in a middle-aged patient with progressive hoarseness, a primary carcinoma of the larynx would be a good consideration. This is further supported by a lytic lesion in the patient's finger, although such an isolated metastatic focus in a phalanx would be very rare.

The feature that seems incompatible with metastases is the curious spontaneous resolution of her multiple skin nodules. Certainly by this time other visceral metastases should be apparent if metastatic disease is to be seriously considered. Regional lymphatic involvement would also be expected.

The infectious granulomas, especially blastomycosis, coccidioidomycosis, and syphilis are additional possibilities. The last one can mimic any clinical situation, but, in view of the more than adequate treatment administered and her very low serologic titer, syphilis would seem to be a very remote consideration. The persistence of the low titer seroreactivity is not a sufficient indication of active disease because a reactive serology may persist for many years in spite of very intensive treatment. Many injections of penicillin and repeated courses of alternate broad spectrum antibiotics are wasted each year trying to erase such reactivity. Persistently reactive serological tests can be difficult to evaluate. If syphilis can be ruled out, false positive reactions always raise the possibility of diseases such as Hansen's disease or a latent collagen disease.

Blastomycosis and coccidioidomycosis could also mimic this clinical situation. Such extensive cutaneous involvement would imply the grave disseminated forms of these infections, and the patient should have much more evidence of serious systemic involvement. Pseudoepitheliomatous hyperplasia would fit with either type of infection, but the responsible organisms are easily visualized on microscopic sections. Negative skin test results do not rule out deep fungus infections for, in patients with little inherent resistance and widespread dissemination, skin tests are frequently negative; when coupled with a rising titer of complement-fixing antibodies, a poor prognosis is usually indicated. There is little support for a diagnosis of a disseminated deep fungus infection in the patient.

The remaining and most likely consideration is some variant of keratoacanthoma. These curious, but by no means rare, skin tumors are characterized by rapid growth followed by spontaneous resolution and residual scarring. Most frequently these lesions occur as isolated nodules which umbilicate and appear to be crateriform. Histologically, they may be indistinguishable from well-differentiated squamous cell carcinoma and can certainly account for the pseudoepitheliomatous hyperplasia noted in this patient's biopsy. The chief point in differentiating them from carcinoma is their capacity to heal spontaneously.

Multiple keratoacanthomas have been described, although only a few active lesions are usually present at any given time and a strong familial tendency has been noted in most of the patients. This is the type referred to by J. Ferguson Smith as "self healing primary squamous cell carcinoma of the skin."[3]

Two case reports have appeared in the literature; one by Grzybowski[1] and a second by Witten and Zak,[4] describing patients with hundreds of keratoacanthomas as seen in this patient. Such rare cases would best be classified as unusual variants of multiple keratoacanthomas.

There are several unique features about the present case that bear further consideration. Most keratoacanthomas appear to derive from hair follicles, although a few have appeared on mucous membranes. Perhaps our histopathologist, Dr. Freeman, can help explain the palmar, plantar, and finger lesions, as well as the laryngeal involvement.

Pathology and Histopathogenesis

DR. ROBERT G. FREEMAN: Histologic examination of several skin lesions from this patient support the diagnosis of keratoacanthoma. Because of the myriads of tumors this case would be classified as the multiple or eruptive type. Classically, keratoacanthomas appear as raised firm lesions with rounded "shoulders" and a central crater filled with keratin debris. The wall of the crater shows hyperplastic and dyskeratotic squamous epithelium which may simulate squamous cell carcinoma. In fact, prior to the description of keratoacanthoma in the American literature in 1954, these lesions were called squamous cell carcinomas. Surveys of such older material indicate that

keratoacanthomas comprise approximately 17 per cent of all lesions originally diagnosed as squamous cell carcinoma. Since 1954, keratoacanthoma has been widely recognized and accepted as an entity. The gross and microscopic architecture are as important as the cytologic details in making a diagnosis of keratoacanthoma. The history of short duration and rapid growth is helpful but not always obtainable. Keratoacanthoma can be differentiated reliably from squamous cell carcinoma in most cases. In a few cases, however, particularly when tangential sectioning is done or when a small portion of a tumor is submitted, the differentiation between these two conditions may not be possible.

The clinical and microscopic appearance (Fig. 3) is dependent upon the stage of development of the tumor. All stages are beautifully illustrated by the series of biopsies

Fig. 3. Microscopic picture of a typical fully developed keratoacanthoma.

taken from this patient. In early lesions the site of origin could be seen in the upper third of the hair follicle. As the lesion progresses, a crater forms and the follicle is obscured by epithelial proliferation. In this stage, the typical crateriform appearance of keratoacanthoma is seen. During regression the crater widens and becomes shallow and the dyskeratosis fades. Some regressing lesions resemble an epidermal cyst.

In this case and in lesions produced experimentally in animals, the keratoacanthomas have arisen in hair follicles. This obviously cannot explain the occurrence of such lesions on the palm or sole or on mucous membranes such as the oral cavity, conjunctiva, or possibly larynx. Other epithelial appendages are, of course, present in these sites, but early lesions from these areas have not been examined to determine the site of origin.

One unusually interesting feature in this patient is the occurrence of laryngeal lesions concomitantly with her skin eruption. A review of these biopsies indicates hyperplastic and dysplastic epithelium which does not show malignant change with absolute certainty but which could possibly represent keratoacanthoma or pseudoepitheliomatous hyperplasia. The coincidental onset at the time of skin eruption, the relatively benign microscopic appearance, and the lack of progression of the lesion following conservative surgical treatment strongly suggest that this lesion was indeed a keratoacanthoma of the larynx, although squamous cell carcinoma must still be considered.

Treatment of Keratoacanthomas

DR. ROSSMAN: Most isolated keratoacanthomas are removed surgically or destroyed by electrodesiccation and curettage. If done properly, this results in much less scarring than occurs in lesions which are permitted to heal spontaneously. It is important to submit adequate tissue, preferably including a portion of the central crater, for histopathologic confirmation.

The large number of active keratoacanthomas in this patient plus a natural reluctance to submit to frequent surgical procedures rendered individual local destruction infeasible. A recent report of successful local cytostatic drug therapy for keratoacanthoma by Klein and associates at Roswell Park suggested this possibility.[2] We consulted Dr. Lane and the chemotherapy group with regard to systemic use of these drugs.

Chemotherapy of Keratoacanthomas

DR. MONTAGUE LANE: Therapy of malignant skin tumors such as mycosis fungoides and advanced squamous cell carcinoma have been within the purview of cancer chemotherapists for some time. Only with the initial reports of the response of psoriasis to the folic acid antagonist Aminopterin (4-aminopteroyl glutamic acid) has there been interest in the systemic use of cancer chemotherapeutic agents in benign skin disorders. These agents tend to be specific only in that they predominantly affect rapidly

proliferating tissues. Generally, the bone marrow, the oral and gastrointestinal epithelia, and rapidly growing tumors are the preferential targets of these drugs. Amethopterin (Methotrexate) has been shown to interfere with the proliferation of cells in hair follicles.[4]

Recently, alkylating agents such as nitrogen mustard, and antimetabolites such as fluorouracil, have been used topically and by direct injection into benign and malignant skin lesions with some success. Fluorouracil and nitrogen mustard have not been used systemically for widespread benign skin disorders. Since we have had some success with therapy of advanced squamous cell cancer of the head and neck region with large oral doses of Methotrexate (4-amino-N^{10}-methylpteroylglutamic acid) (20-25 mg. per day for five days), administration of a smaller dose (20 mg. per day for four days) was suggested for this patient. If this therapy proves ineffective, a reduced dosage program of fluorouracil might be considered.

Summary and Conclusions

DR. KNOX: Combining the clinical and histopathological findings, this patient appears to have the rare eruptive type of multiple keratoacanthomas. The large number of active lesions with a widespread distribution and relatively late age of onset contrasts sharply with the ordinary multiple type in which a few lesions appear during early adult life, slowly involute and periodically recur over many years.

It is unusual but not rare to find keratoacanthomas in a Negro patient. Other unique features include the palmar and laryngeal lesions. Although the laryngeal nodule was originally thought to be a well-differentiated squamous cell carcinoma, this diagnosis has been questioned. The time of onset of laryngeal symptoms, the lack of invasion, the histological appearance, and the benign postoperative course favor an interpretation of keratoacanthoma.

Gradual recognition of the many variations of keratoacanthoma has produced an ever-enlarging clinical concept, and better appreciation of the entity may prevent unnecessary surgery. Despite previous descriptions of viral-like nuclear inclusions, attempts to transmit the lesions using tumor tissue homogenates have been unsuccessful. Topically applied carcinogens have often been successfully employed to produce tumors indistinguishable from keratoacanthomas in laboratory animals. The etiology of these spontaneously regressing neoplasms in man, however, remains unknown.

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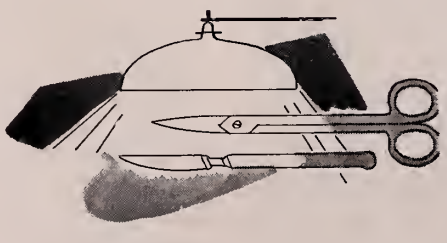


NEWS

January Conference Attracts 574

SPRINGLIKE WEATHER after several days of ice and snow was a fortunate development for Jan. 18, the day of the Texas Medical Association Conference on Medical Service and Legislation and the Orientation Program. Held in Austin at the Headquarters Building, the meeting was attended by 574 physicians, Auxiliary members, and guests, the highest total attendance to date for a TMA January meeting.

Four simultaneous conferences were held during the day. These were a symposium for county medical society secretaries, a conference on medical service and legislation, an orientation program, and a meeting of the Woman's Auxiliary. On Friday preceding the meeting, executive secretaries of county medical societies participated in a conference at the Stephen F. Austin Hotel. Registration figures showed that 70 physicians attended the orientation program and 101 physicians' wives, the Auxiliary program.

Lunch was served at the Building for those attending the conferences, and a hospitality hour at the Driskill Hotel concluded the day. Texas Medical Association was host for the lunch, and Employers National Life Insurance Company affiliated with Texas Employers Insurance Association and Employers Casualty Company, Dallas, was host for the hospitality hour.

All groups joined to hear five principal speeches. A sixth address listed on the program was not heard because the speaker, Dr. Franklin F. Ham of Van Nuys, Calif., could not secure air transportation to Austin in time for the meeting.

The three speakers heard during the morning session were Dr. Robert Mayo Tenery of Waxahachie, President of Texas Medical Association; Thomas J. Anderson of Nashville, Tenn., president of Farm and Ranch Publications; and Capt. Kenneth W. Ryker of Fort Worth, executive director of the Fort Worth Freedom Center.

The late afternoon session included the addresses of Dr. Milford O. Rouse of Dallas, Speaker of the AMA House of Delegates, and Walter E. Rogers of Pampa, Congressman from Texas' 18th District. Dr. Rouse, because of laryngitis, was unable to give his speech, and it was read by Darrell Coover of Park Ridge, Ill., director of the Community Relations Department of the AMA Communications Division.

Physicians pause for coffee during the all-day conference.

A report of the five principal speeches follows:

Farm and Ranch publisher Thomas J. Anderson of Nashville asserts, "I may die in a dictatorship, but I do not intend to live in one."

Tenery: *What's Ahead For Us in 1964?*

"The answer is one big fight," said the President of Texas Medical Association; and these were his first words after he was introduced and the subject of his address was announced.

"No one can say for sure what is going to happen in the next three months," he continued, "but indications are that the big push is on."

Dr. Tenery believes that the House Ways and Means Committee currently stands with physicians in their fight against medicare, but he pointed out that President Johnson has indicated "three times now" that he is for health care for the aged via the social security mechanism.

"If he means what he says, we are in for a fight again," he said.

"And we will have to do the job which should have been done before," he declared.

Dr. Tenery urged his audience to assimilate as much information as possible for use in preventing medicare. He suggested that physicians make sure that information available from the AMA on this subject is in their waiting rooms.

Paramedical groups are working with Texas physicians, said Dr. Tenery, but this is not the situation in some other states. He urged physicians to call on their friends in paramedical groups to help wage the battle against the King-Anderson bill.

He reminded the audience to work for passage of the Constitutional amendment next November to add the Medical Aid to the Aged provision to the Texas Kerr-Mills program.

"We must improve our image," he stated, adding that physicians have an obligation to keep fees reasonable. He called for improvement in relations with news media and stated that medical societies should establish realistic publicity codes.

Regarding other areas, he called for support of a program to keep teenagers from taking up the smoking habit and for improved nursing home accreditation.

He urged promotion of the medicine and religion program. This has been the best

Capt. Kenneth W. Ryker, USAF Retired, points out some apparently unrelated occurrences of the past five years to show why he feels the United States is headed down the wrong track.

Dr. Milford O. Rouse of Dallas, a victim of laryngitis, hands his speech to Darrell Coover of the American Medical Association who read it before a late afternoon audience.

received program TMA has started, judging from the volume of newspaper clippings which have come in concerning it, he said. "People want it, we need it, and it will certainly help our patients."

Anderson: *Straight Talk*

"The issue today is not South versus North. The issue is not health, education, welfare, taxes, economics, or Catholicism. The issue is not Republican versus Democrat. The issue is not segregation versus integration. The issue is freedom. Not freedom *from*, but freedom *to*. Not forced freedom, but freedom of *choice*. Freedom to *choose* which doctor is going to have to wait to get paid. Not legislated freedom, but God-given freedom. For free men are not equal, and equal men are not free," began Thomas J. Anderson of Nashville, president of Farm and Ranch Publications.

He came before his audience after a brief introduction made by Dr. R. H. Bell of Palestine who pointed out that many physicians today own cattle and read a lot of farm literature including Anderson's. Dr. Bell said he had found Anderson's editorials to be particularly good.

A graduate of Vanderbilt University, Anderson spoke in the modulated tones characteristic of his native region, spicing his address with homey anecdotes, but the burden of his message was forceful and direct.

Both parties are taking us down the road to socialism, he said. "Socialism is the halfway house to communism."

The crowd in Washington is trying to bring on socialism, Anderson believes. "The State Department has been infiltrated by the

enemy." It is imperative that we investigate the State Department, he said. "I'm not worried about the Communists—it's the liberal mice I'm worried about," he explained.

Quoting Dante, he said, " 'The hottest fires in Hell are reserved for those who in a period of moral crisis maintain neutrality.' " This is a period of moral crisis, he stated, and it is impossible to know the truth and not be held responsible.

"The great issue of our time is not education versus ignorance in spite of what the eggheads try to tell us. Some of the most educated people in the world are the most wicked. Empty bellies are not, as our materialists say, the cause of Communism or of wars. Germany started two wars on a full belly. The Communists and fellow travelers in America and throughout the world are mostly full bellied. As Robert Welch has said, 'There are more Commies with Harvard accents than there are in overalls.' "

Anderson said he was amazed by people who say they can't afford to get mixed up in politics.

He urged physicians to get into politics on all fronts, not just as it regards medicine. He said doctors in Tennessee helped re-elect Kefauver by opposing him. "People didn't like the idea of doctors jumping into the political fray merely because their own ox was being gored," he said.

It has become controversial to be for the Constitution, but not controversial to be for worldwide socialism, he declared. "We're in this position because we have defaulted."

"I may die in a dictatorship, but I do not intend to live in one," he concluded.

Ryker: *The Issue Is Freedom*

"To throw some light on the 'big picture' " Capt. Kenneth W. Ryker, USAF Retired, fitted pieces of a jigsaw puzzle in place on a flannel board before his audience of phyicians.

Each piece of the puzzle represented a seemingly unrelated occurrence. When fitted together, Capt. Ryker, who is executive director of the Fort Worth Feedom Center, explained, "the picture gradually emerges."

This picture is "dismal," he says. It shows how the American people are being railroaded down a track today which can mean the end of "the American way of life."

Events represented by pieces of the puzzle included Khrushchev's speech before the U. N. General Assembly, Sept. 18, 1959; the Asilomar Conference in April, 1960, which was a national strategy seminar jointly sponsored by the Army, Navy, and Air Force; the sixth Pugwash Conference, November-December, 1960, (the name Pugwash comes from the place in Nova Scotia where the conferences were held) ; the Moscow Manifesto, December, 1960; the Bay of Pigs fiasco, April 17, 1961; the Fulbright memorandum, June, 1961; State Department Document 7277, Sept. 25, 1961; the Reuther Memorandum, Dec. 19, 1961; Rostow Papers, June, 1962; news management, October, 1962; fairness doctrine, July 26, 1963; Nuclear Test Ban Treaty, September, 1963; and the sale of wheat to the U.S.S.R., October, 1963.

The major premise upon which Captain Ryker based his reasoning with regard to the Soviet Union is that of J. Edgar Hoover, director of the F.B.I.: "We are at war with Communism and the sooner every red-blooded American realizes this the safer we will be." Using deductive reasoning, Captain Ryker drew this conclusion from his major premise:

"If we are at war with Communism it follows we are at war with the Soviet Union, for that country is the international headquarters of Communism. If we are at war with the Soviet Union, it follows that the sale of wheat to the Soviet Union is treason as defined by Article III, Section 3 of the United States Constitution as giving 'aid and comfort' to the enemy."

Captain Ryker emphasized at the end of his address that he does not think it is too late "to reverse the tide." He urged physicians to support "those on the front lines of the battlefield of freedom in your communities. . . . And above all, remember—the issue is freedom!"

Rouse: *American Medicine—Its Current Problems, Future*

After expressing a brief greeting to his audience, Dr. Milford O. Rouse, Speaker of the House of Delegates of the American Medical Association, handed his prepared text to Darrell Coover, an AMA staff member, who read the speech. Dr. Rouse had contracted a case of laryngitis a few days earlier.

The first part of the speech was devoted to answering the question "How are policies of the American Medical Association formulated?" It was stressed that the county medical society is the autonomous group of medicine and that the state and AMA organizations might be termed service organizations. "The balance of power and authority for policy-making decisions rests with the organization as close to the individual doctor as possible so that he can have responsive, democratic representation."

If the county medical organization has the right to decide medical policy, the audience was told, it follows as a necessary corollary that it should have responsibilities. "One of these is to accept responsibility for following the programs, including the legislative program, of the state and national organizations and implementing them at the grass roots. These programs are set by your chosen representatives in the House of Delegates."

It was then emphasized that it is not enough for the individual physician to give tacit support to the efforts of his local and state societies or to those of the AMA.

"He must be the articulate voice of his profession, wherever he is. He must be dedicated to the well-being of his community. In matters of health, he must be willing to assume responsibility and discharge it as the expert he is. He must be citizen as well as doctor, plaintiff's attorney for his patients, missionary for the individual and for the individual's rights," Dr. Rouse proposed.

Dr. Rouse, through the text of his speech, then took up a second proposition: There has been a breakdown of communications between physician and physician, between physician and patient, and between community and physician.

"The public image of the physician has been unjustifiably tarnished because of our failure to communicate properly," he claimed.

He called for effort to remedy this situation, particularly in the areas of patient-physician and community-physician relationships.

TMA President Dr. Robert Mayo Tenery talks with Congressman Walter E. Rogers of Pampa. Both were speakers.

Rogers: *Issues Facing The Congress in 1964*

Three domestic issues expected to be foremost in 1964 — taxation, civil rights, and medicare—were discussed by Congressman Walter E. Rogers of Pampa who represents the 18th District. He touched on the subject of certain administrative agencies of government to which certain regulatory powers have been delegated, and which he feels are attempting to invade the legislative field. He concluded his address with a discussion of world problems.

Regarding taxation, Congressman Rogers said the tax reduction and reform legislation passed by the House during the past session and presently pending in the Senate provides the opportunity to prove that relaxation of government controls and a reduction of the tax burden will encourage the free enterprise system to provide the goods, the services, and the jobs needed in increasing abundance.

"It represents the first effort in our history to stimulate economic development by freeing capital," he stated.

The intense emotional heat that has been associated with the "omnibus civil rights bill"* and like measures during the past 20 years has not decreased, it is the opinion of Congressman Rogers. He had expected debate on the subject matter of the present bill to start before the full House of Representatives in the near future. He expected contention to be greatest over the public accommodations section of the bill. This section involves certain basic legal and constitutional questions.

"If the basic fundamentals of contract law are to be changed, and there is to be a redefinition of interstate commerce to accomplish one specific purpose, the people of this country are entitled to know what the impact of such action will be on their own rights as well as the rights of the business people directly concerned," stated the Congressman.

An intelligent decision cannot be made until the approximate length and breadth of this impact is known, he said.

Regarding medicare, Congressman Rogers said, "I am sure that all of you are familiar with the proposals that have been made on the subject in recent years as well as the arguments pro and con concerning a national program of health insurance."

He continued, "I would warn that there appears to be a growing sentiment throughout the country for a realistic attack on whatever problems exist. Unless the situation is faced on a purely factual basis, and the people generally are made to understand it, demagogues will be afforded a source of strength for false appeals to the public."

"These could possibly result in the acceptance of unworkable plans that could do damage, if not violence, to the medical profession. This we must avoid at all costs," he said.

Regarding administrative agencies which are attempting to invade the legislative field, Congressman Rogers said that a number of Congressmen are trying to "cure these attempts" for unless this practice is stopped, rights will be lost that may never be regained. These agencies to which he referred are using their "rule-making" powers, he said, to "adopt rules and regulations under the guise of a procedural format which, in fact, have the same effect upon the businesses regulated as substantive law."

"I do not charge that this is being done with malice aforethought, but its effect is nonetheless dangerous," he stated.

Ed. Note: The House passed this bill on Feb. 10 as the *Journal* was going to press.

68 Loans Made in 1963 From Sam Thompson Fund

The Board of Trustees has reported that 68 loans in the total amount of $29,450 have been awarded from the Dr. S. E. Thompson Fund. Loans were made to students at the University of Texas Medical Branch and Southwestern Medical School.

The Trustees now have made a total of 280 loans in the amount of $118,000 since the loan program was placed in operation late in 1959. These loans are available to needy and deserving Texas residents who are enrolled in the second, third, and fourth years of study at medical branches of the University of Texas.

Commitee, Council Recommend Oral Polio Boosters

The Committee on Public Health and the Council on Public Relations and Public Service suggest that county medical societies and individual physicians now give a booster of trivalent oral polio vaccine, the method of administration to be determined at the local level.

Physician-insurance company relationships were discussed by a panel including (standing, left to right) Dr. Charles D. Bussey of Dallas, Dr. Everett C. Fox of Dallas, the moderator, (seated, left to right) Russell B. Donovan of Dallas, Frank J. McGurl of Houston, Charles D. Scott of Dallas, and Dr. Charles E. Oswalt, Jr., of Fort Stockton who introduced the group.

Insurance, Texas Story Are Topics for Discussion

PHYSICIANS attending the Conference on Medical Service and Legislation in Austin Jan. 18 had the opportunity to hear two panel discussions of general interest to the medical profession.

During the morning the topic was "Physician-Insurance Company Relationships" and during the afternoon, "The Texas Story—Medical Care for the Aged."

Physician-Insurance Company Relationships

Dr. Charles E. Oswalt, Jr., of Fort Stockton, Chairman of the Board of Councilors, introduced the men who discussed the morning topic. Moderating the discussion was Dr. Everett C. Fox of Dallas, Chairman of the Liaison Committee with Blue Shield. Members of the panel were Dr. Charles D. Bussey, Dallas, vice chairman, Hospitals-Insurance-Physicians Joint Advisory Committee of Texas; Russell B. Donovan, Dallas, representative to HIPJAC from Texas Health Insurance Council; Dr. Frank J. McGurl, Houston, medical director, Prudential Insurance Company of America; and Charles D. Scott, Dallas, president of Great American Reserve Insurance Company.

Dr. Bussey.—After discussing the history and purpose of the HIPJAC Committee, Dr. Bussey stated that most of the cases referred to the Committee for review are not deliberate abuses of health insurance but indicate a lack of knowledge on the part of one of the parties.

"In those cases of actual abuse I must say, in all fairness, that the insurance segment of the committee has . . . been more effective in bringing an insurance company into line than the hospital and physician segment of the committee has in bringing a hospital or physician into line," said Dr. Bussey.

He believes this is true because often the hospital or physician in question is not in or is not active in either the Texas Hospital Association or the Texas Medical Association. He concluded his remarks with a discussion of the responsibility of the individual physician, the county medical society, and the Texas Medical Association in the matter of voluntary health insurance.

The Texas story on medical care for the aged was told by this group. Left to right are Dr. A. Rex Kirkley, Dallas; Fred R. Higginbotham, Dallas; Dr. Harvey Renger, Hallettsville, (standing); H. Lewis Rietz, Houston; and Joe Belden, Dallas.

Physicians who spoke before the group attending the symposium for county medical society secretaries include (left to right) Drs. Walter C. Barnes, Jr., Texarkana; Dick Cason, Hillsboro; and James H. Sammons, Highlands.

Mr. Donovan.—"In the administration of policy benefits," said Mr. Donovan, speaking for the insurance industry, "we are solely concerned with the proper discharge of our contractual obligation to the policyholder. Our interest in the medical aspects of the case—diagnosis, treatment, past history, etc. —is only incident to and in furtherance of that objective. We do not view the physician as either an advocate or an adversary in claim transactions. We ask him to serve only as an expert witness to establish the facts upon which our liabilities to our insured may be determined."

Dr. McGurl.—In commenting on the relationships between the insurance company medical director and physicians in private practice, Dr. McGurl said medical directors are frequently asked whether, in their opinion, a professional fee is fair or excessive. "In making this determination, we consider the type of surgery (it is most often a surgical case), the professional status and background of the physician, the locale, the income of the insured, and the presence of any complications," he said.

In answer to the question of how medical directors arrive at medical fees for reports completed by physicians, Dr. McGurl stated that this fee should be commensurate with the clerical time necessary for its completion, subject, of course, to certain minimums and maximums.

Mr. Scott.—"Not to minimize the impact of the evolution in medical care practices and the socioeconomic trend, the fact remains that the practicing physician bears a grave responsibility in controlling health care costs, costs which are reflected in higher health care insurance premiums," said the insurance executive.

In speaking of the responsibility of the writers of health insurance, he said there must be an awareness on their [the insurers'] part of the evolution that is proceeding in the provision of medical care and to design and administer insurance coverages to meet these changing needs.

The Texas Story—
Medical Care for Aged

Dr. A. Rex Kirkley of Dallas, Chairman of the Committee on Health Insurance, moderated the afternoon panel discussion. Taking part were Fred R. Higginbotham, Dallas, director of professional and public relations for Blue Cross-Blue Shield of Texas; H. Lewis Rietz, Houston, president of Texas 65 Health Insurance Association; Joe Belden, Dallas, president of Belden Associates; and

Dr. Harvey Renger of Hallettsville, Past President of TMA.

Mr. Higginbotham.—A review of history of the Kerr-Mills Act in Texas was presented by Mr. Higginbotham who credited Commissioner John Winters and his staff in the Texas Department of Public Welfare with foresight in asking insurance companies to bid on the health insurance program for Old Age Assistance recipients. Some other state governments have tried to handle this themselves—and not too successfully, he said. Mr. Higginbotham then showed slides to illustrate statistically the success of the Kerr-Mills OAA program in Texas.

Mr. Rietz.—In a period of a little more than 30 years, some 75 per cent of the people in the United States have come to carry some form of health insurance, stated Mr. Rietz. He then described the Texas 65 plan under which last fall more than 50,000 Texans, with an average age of 73, were enrolled. The Texas-licensed companies who grouped to offer the Texas 65 plan designed the policy so that premiums are in a range most people can afford. They also took one precaution in issuing the policy—provision for a reasonable waiting period to protect themselves against claims that already existed.

"Many of us think this program will phase out," he said. As time goes on people will carry other insurance into retirement years and the need for Texas 65 will not exist, he explained.

Mr. Belden.—Mr. Belden reported on a survey conducted by Belden Associates, Dallas, in 1962 for Blue Cross-Blue Shield of Texas on the medical care needs of the aged population of Texas.

"We set out to measure three wide areas: (1) opinion on ways to meet medical needs, (2) actual medical needs and expenses of the aged during the year 1961, and (3) the resources the aged now have to meet medical needs—that is, their own finances, help from the family, their living arrangements, and insurance coverage," Mr. Belden said.

It was concluded from the data obtained in the survey that approximately 3 per cent of all aged Texans (excluding those receiving outside help in the form of Blue Cross-Blue Shield coverage as part of their OAA benefits) need and want outside help.

Dr. Renger.—If the King-Anderson bill

A group of county society executive secretaries gathered for lunch and an afternoon meeting in Austin on Friday before the all-day conference on Saturday. They are (standing, left to right) S. E. Cockrell, Jr., Bexar county; Bill E. Robertson, Harris county; George S. McKay, Harrison county; Richard Rodgers, Lubbock-Crosby counties; Roy Cates, Travis county; (seated, left to right) Marion D. Barrett, Hidalgo-Starr counties; Jean Oliver, Nueces county; and Mary Tom Rasco, Potter-Randall counties.

were the law, it would cost the Texas Ninth Congressional District taxpayers $4,000,000 to cover a $600,000 hospital bill for the aged in that district, according to Dr. Renger. Dr. Renger recently conducted a survey in which he received statistics from 24 of 34 hospitals in the district. These figures show that only 21.3 per cent of the aged hospitalized were not covered by some form of insurance. Projecting this percentage figure to cover the total 34 hospitals of the district, the amount not covered would be $600,000.

"This $600,000 will easily be completely eliminated when, in November, the citizens of Texas will vote on the MAA phase of the Kerr-Mills Act," Dr. Renger stated. If passed in Texas, this phase of the law will be more than adequate to take care of this portion of the aged unable to cover their entire bill for medical care, he explained.

101 Women Attend Auxiliary Workshop

One hundred-one members of the Woman's Auxiliary to the Texas Medical Association attended the 1964 Legislative Workshop in Austin Jan. 18 at the Headquarters Building.

A portion of the day was spent in joint session with Texas Medical Association, and the Auxiliary members were guests of TMA at its luncheon.

Hostesses were members of the Woman's Auxiliary to the Travis County Medical Society.

The women's program, planned by Mrs. John J. Andujar, Fort Worth, legislative chairman, and Mrs. Warren Binion, Fort Worth, legislative co-chairman, was aimed at familiarizing Auxiliary members with issues facing Congress in 1964. Mrs. Aaron E. Margulis, Chicago, American Medical Association Field Representative to Women's Organizations, addressed those at the Workshop on the subject "Women *Will* Listen." Capt. Kenneth W. Ryker, USAF, Ret., of Fort Worth presented dangers confronting the United States in his speech entitled "Which Way, America?" The "Texas story" on legislation was presented by C. Lincoln Williston, Executive Secretary of TMA, in his speech entitled, "Texas Leads the Way, Let's Tell It."

Presiding was Mrs. Hamilton Ford, president of the Woman's Auxiliary.

A question and answer session was afforded listeners by Mrs. Margulis, Captain Ryker, and Mr. Tom Anderson, Nashville, Tenn.

Mrs. Aaron E. Margulis, AMA Field Representative to Women's Organizations, spoke to members of the Woman's Auxiliary.

The Auxiliary program also included showing of films and discussion of material and information to be used by legislative chairmen at the county auxiliary level.

Rural Health Conference To Be Held in Columbus, Ohio

The American Medical Association Council on Rural Health will present its 17th national conference on rural health March 6-7 at the Columbus Plaza Motor Hotel in Columbus, Ohio.

Keynote address will be given by Paul A. Miller, Ph.D., president, West Virginia University.

Additional information may be secured by writing to the Council on Rural Health, American Medical Association, 535 North Dearborn St., Chicago, Ill. 60610.

Executive Board Receives Reports; Adopts Those Needing Immediate Action

THE EXECUTIVE Board on Jan. 19 approved in principle two pieces of proposed legislation which involve the health of children in Texas—the child abuse bill and the thallium bill. The Board also called for publication in the *Texas State Journal of Medicine* the Board of Councilors' opinion regarding lay-owned laboratories (p. 179), pointed to the need for a Department of Corrections hospital in Galveston, and recommended certain revisions in the minimum standards for nursing homes.

The Board also approved a resolution opposing the assumption by the Department of Labor of certain regulatory authority in the field of ionizing radiation which is already delegated to the Atomic Energy Commission and certain states.

In approving the recommendation of the Council on Medical Jurisprudence regarding the thallium and child abuse bills, the Board asked for the assistance of the Woman's Auxiliary, Texas Pediatric Society, the Texas Chapter of the American Academy of Pediatrics, and the Texas Academy of General Practice in presenting the problems to the public.

Most of the reports heard by the Board were received for information and will receive final action from the House of Delegates in April. Those items requiring immediate action were adopted by the Board.

The Board stood for a moment of silence

Sen. Walter Richter of Gonzales, chairman of the Governor's Committee on Aging, stresses a point in talking to members of the TMA Committee on Aging.

Members of the Committee on Disaster Medical Care heard Dr. Robert Klein of Gainesville, Fla., discuss procurement of blood during emergency situations. Seated around the table, left to right, are Drs. T. June Melton, Midland; W. D. Nicholson, Freeport; O. A. Fly, Jr., Houston; Joe R. Shaeffer, San Antonio; and Ben A. Merrick, Dallas.

after hearing a resolution from the Board of Trustees regarding the death on Oct. 9, 1963, of Dr. Troy A. Shafer of Harlingen, former Chairman of the Board of Trustees.

TMA Library Receives Valuable Rare Book

The TMA Memorial Library has been presented the second edition of the work by Andreas Vesalius on anatomy. This rare book was given to the Library by Dr. Hampton Robinson of Houston.

The book was prepared by Vesalius himself and was published in 1555. Copies of the second edition are scarce, and the value of this copy is estimated to be several thousand dollars.

The book is in excellent condition. It was rebound during the last century, and its pages show very few water marks. Vesalius' work has never been translated into English, but the Russians have translated it into their language.

The book is particularly interesting because of its illustrations which were printed from wood blocks.

Benevolent Fund Receives 100% Support of 9 Societies

Nine county medical societies have already achieved 100 per cent participation among their members in contributing to the Physicians Benevolent Fund. More than $5,000 has been donated and additional contributions are being received daily.

Recently a special committee has conducted fund-raising activities for money designed to help needy physicians, both young and old, with insufficent incomes and who are unable to work. The fund was started in 1962 by Dr. May Owen of Fort Worth, TMA Past President, who contributed $2,500.

Recent cash gifts will be invested, and it is hoped that as the fund grows, dividends eventually will be adequate for the purpose

Mrs. Barbara Wray of Dallas, assistant to Dr. Samuel A. Shelburne, left, and Mrs. Barbara Dickerson of Fort Warth, assistant to Dr. William H. Morgan, center, receive certificates designating them among the natian's first Certified Medical Assistants. Presenting the certificates is Mrs. Judy Coleman of Dallas, assistant to Dr. Charles D. Bussey, and president of the American Association af Medical Assistants.

of the program.

County societies which have contributed 100 per cent include Van Zandt, DeWitt, Jasper-Newton, Colorado Basin, Coryell, Denton, Permian Basin, Johnson, and Hopkins-Franklin.

Councilors Issue Opinion On Lay-Owned Laboratories

The Board of Councilors on Jan. 18 rendered the following opinion concerning lay-owned laboratories:

"It is unethical for a member of the Texas Medical Association to do business with lay-owned laboratories which are operated without the supervision of a licensed Doctor of Medicine. The Attorney General has held that such unsupervised laboratories are in violation of the Medical Practice Act. Reference is made to Section 4 of the 'Principles of Medical Ethics' which reads in part, 'Physicians should observe all laws. . . .' "

The Board of Councilors followed the dictates of the House of Delegates in considering the question. At its meeting in Dallas April 27-30, 1963, the House recommended that the Board of Councilors take cognizance of the problem regarding lay-owned laboratories from an educational viewpoint among the members of the TMA and take steps to correct this educational deficiency.

Ninth District Society To Meet in Huntsville

The Ninth District Medical Society will meet in Huntsville March 19 at the Student Recreation Center.

Scientific presentations will include "Tracheostomy, Indications, Technique, and Aftercare," Dr. Fred Aves, Houston; "Diabetic Care and Emergencies," Dr. Leonard Robbins, Houston; "Ob-Gyn Emergencies," Dr. L. E. Lundgren, Houston; "Urology and Abdominal Pain," Dr. James Boren, Houston; "Management of Severe Burns," Dr. S. Baron Hardy, Houston; "Management of Infant Diarrhea," Dr. John K. Glen, Houston; "Diseases of the Thyroid, Medical Aspects," Dr. Thomas G. Vandivier, Houston; and "Diseases of the Thyroid, Surgical Aspects," James Allen Chamberlin, Houston.

Dr. Max Johnson of San Antonio will be the luncheon speaker.

Blood Banks Association To Meet March 27-28

The South Central Association of Blood Banks will meet in Oklahoma City March 27-28 at the Skirvin Tower Hotel.

Two Texas physicians are listed on the program. Dr. Arthur C. Beall, Jr., of Baylor University College of Medicine, Houston, will speak on "Open Heart Surgery Eliminating the Need for Fresh Heparinized Blood." Dr. William C. Levin of the University of Texas Medical Branch, Galveston, will speak on "The Interrelationship of Serum Immunoglobulins and Isohemagglutinins."

Any member of the medical profession, administrative or technical personnel, or members of biological or chemical sciences interested in blood banking are invited to register, according to Mrs. Florence Del Prete, secretary of the South Central Association of Blood Banks, 3600 Gaston Avenue, Dallas 10.

Diabetes To Be Topic Of Bowie-Miller Seminar

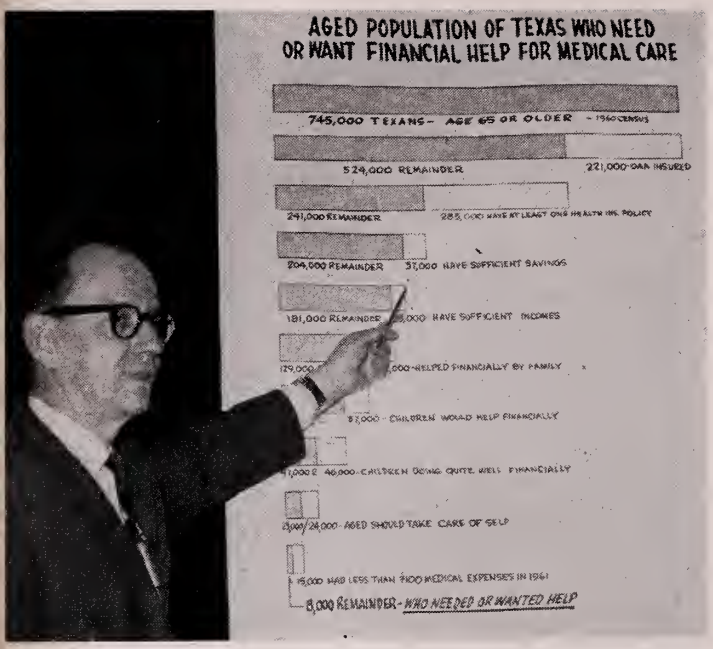
"Diabetes Mellitus" will be the conference subject of the Bowie-Miller Counties Medical Society annual medical seminar Saturday, March 21, at the Holiday Inn in Texarkana.

Speakers for the program will be Dr. Theodore C. Panos, Little Rock, Ark., chairman of the Department of Pediatrics, University of Arkansas School of Medicine; Dr. George L. Jordan, Jr., Houston, associate professor of surgery, Baylor University College of Medicine; Dr. Harold Brown, Houston, professor of medicine, Baylor University College of Medicine; and Dr. Harry S. Lipscomb, Houston, associate professor of physiology and medicine, Baylor University College of Medicine.

The medical auxiliary will also have a program.

Cost of the seminar and luncheon is $15. Speaker for the banquet on Saturday at the Texarkana Country Club will be Jeff Williams, Oklahoma philosopher and humorist. The cost of the seminar plus the banquet is $25.

Reservations for the seminar and banquet may be made with Mrs. James Pryor, secretary, Texarkana-Bowie Counties Medical Society, Box 1288, Texarkana.


AGED POPULATION OF TEXAS WHO NEED OR WANT FINANCIAL HELP FOR MEDICAL CARE
745,000 TEXANS - AGE 65 OR OLDER - 1960 CENSUS
524,000 REMAINDER
221,000 OAA INSURED
241,000 REMAINDER
285,000 HAVE AT LEAST ONE HEALTH INS. POLICY
204,000 REMAINDER
37,000 HAVE SUFFICIENT SAVINGS
181,000 REMAINDER
HAVE SUFFICIENT INCOMES
179,000
HELPED FINANCIALLY BY FAMILY
CHILDREN WOULD HELP FINANCIALLY
46,000 CHILDREN DOING QUITE WELL FINANCIALLY
24,000 AGED SHOULD TAKE CARE OF SELF
15,000 HAD LESS THAN $100 MEDICAL EXPENSES IN 1961
8,000 REMAINDER - WHO NEEDED OR WANTED HELP

Joe Belden of Belden Associates, Dallas, illustrates the results of his investigation of the medical care needs of the aged population of Texas. Mr. Belden was testifying before the House Ways and Means Committee during recent medicare hearings.

Personals

Jaime N. Delgado, Ph.D., University of Texas associate professor of pharmaceutical chemistry, discussed "Recent Developments in Steroids" at the January meeting of the Travis County Pharmaceutical Association. *Dr. Delgado* is a regular contributor to the "Recent Advances in Drug Research" section of the *Journal.*

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Dr. James Stanton Bussell of Marlin and *Dr. Alva Rex Kirkley,* medical director of Blue Cross-Blue Shield in Dallas and formerly of Belton and Temple, received the highest award in Scouting, the Silver Beaver. The award was presented at the annual banquet of the Heart O' Texas Council, Boy Scouts of America, Dec. 17 at James Connally Air Force Base in Waco.

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Dr. McIver Furman of Corpus Christi was elected president of the Chamber of Commerce recently.

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Dr. Erwin G. Pink of Frisco, Texas, has been named the outstanding ex-student of Midwestern University. The award will be presented at the spring graduation ceremonies.

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Dr. A. Ford Wolf was recently elected president of the Scott and White Memorial Hospital. New trustees named to the Scott and White Memorial Hospital and Scott, Sherwood and Brindley Foundation were *Dr. Walker A. Lea, Jr.,* and *Dr. J. H. Greenwood.*

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Maj. Gen. Aubrey L. Jennings has received the Legion of Merit with first Oak Leaf Cluster. General Jennings who retired Jan. 1, received the award for outstanding service to the United States as Director of Professional Services, Office of the Surgeon General, USAF, from Aug. 1, 1959, to Oct. 31, 1963. *General Jennings* is a native of Bartlett.

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The Hill County Medical Society recently heard a paper on "Recognition of Heart Defects in the Newborn" presented by *Dr. Andrew Megarity* of Corsicana.

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The Harris County chapter of the Academy of General Practice has elected these officers: *Dr. Charles W. Klanke,* president; *Dr. W. M. Wallis,* president elect; *Dr. Jack Gerdes,* vice president; *Dr. Russell Turner,* secretary; *Dr. Antonio Vlahakos,* treasurer; and *Dr. Joe T. Ainsworth,* member of the board of directors.

Allergists to Meet In Bal Harbour, Fla.

The American College of Allergists will hold its 20th annual congress and graduate instructional course in allergy at the Americana Hotel, Bal Harbour, Miami Beach, Fla., March 1-6.

The overall program chairman is Dr. Boen Swinny of San Antonio, president-elect of the College.

Texas physicians who will contribute to the scientific program include Drs. Bernard T. Fein, San Antonio; John P. McGovern, Houston; Homer E. Prince, Waco; Herschel E. Whigham, McAllen; and Orville C. Thomas, Houston.

Baylor Receives Grant For Cancer Research

A resource grant of over $300,000 for research in cancer—particularly leukemia and blood malignancies—in children has been awarded to the Department of Pediatrics at Baylor University College of Medicine and to Texas Children's Hospital in Houston.

Presented by the National Cancer Institute of the Public Health Service, the grant provides $113,000 for one year and guarantees $98,000 for each of two additional years. It will expand research begun at Texas Children's Hospital in 1957 as a cooperative program with the Southwest Cancer Chemotherapy Study Group.

"The grant is unrestricted in the sense that it will allow us to investigate multiple problems related to cancer in children," said Dr. Donald J. Fernbach, principal investigator of the project. Dr. Fernbach is an assistant professor of pediatrics at Baylor and the chief of hematology at Texas Children's Hospital.

Provisions are made in the grant for additional personnel and equipment. An eight-room laboratory in the residency building adjoining TCH is being adapted for use in the study. Dr. Fernbach said he hopes to move into these quarters by March 1.

The work at the hospital includes participation in the Human Cancer Virus Task Force studies at Baylor into the role of viruses in leukemia. This project is a collaborative program with Dr. Matilda Benyesh-Melnick and Kendall O. Smith, Ph.D., of Baylor's Department of Virology and Epidemiology. Dr. Fernbach and his associates will provide material for injecting newborn baboons of a special primate colony, to be established at Baylor in 1964.

"Our ultimate hope, of course, is to determine what causes malignant disease in children," Dr. Fernbach said.

"If we never find the ultimate causes, we would still like to know more about the clinical problems of the diseases and how to treat them more effectively," he said.

To take maximum advantage of the material available and to coordinate the investigation, Dr. Fernbach's group will use an IBM electronic data processing system. A wide variety of information—such as family background, previous illnesses, previous exposure to toxic substances, blood counts, and response to drug and blood component therapy—will be stored in the computer.

Clinical studies, already in progress, will be expanded when the new laboratory is occupied. These studies include a survey of the epidemiology of leukemia; an evaluation of blood component therapy in the treatment of anemia and bleeding complications of leukemia; a study of the function of the leukemic white blood cell; an evaluation of newer antibiotics in dealing with infections in leukemic children, and other similar studies.

"Have you thought of trying to bowl left-handed?"

Houston Symposium Set For Neurologists

The 12th annual scientific meeting of the Houston Neurological Society and the Department of Neurology, Baylor University College of Medicine, will be held in the Texas Medical Center, Houston, March 12-14.

This symposium, entitled "Intracranial Aneurysms and Subarachnoid Hemorrhage," with emphasis on pathologic anatomy, angiography, and medical and surgical therapy, will encompass the experimental and clinical presentations of 21 internationally known authorities from Canada, England, Finland, Sweden, and the United States.

Further information may be obtained from Dr. William S. Fields, professor and chairman, Department of Neurology, Baylor University College of Medicine, Houston 77025; or Dr. Adolph L. Sahs, professor and head of the Department of Neurology, State University of Iowa College of Medicine, Iowa City.

Advice on How To Quit—Smoking, of Course

A smoker who is "personally convinced" *can* give up the habit.

This is the conclusion of Dr. George William Ware, Washington, D.C., offered in the February *Today's Health* magazine, published by the American Medical Association.

Summing up results of his studies, Dr. Ware said no one stops smoking because of newspaper stories, requests by his wife, or half-hearted intentions concerning next week.

Most smokers cannot cut down, according to the physician. A voluntary restriction of cigarettes is likely to last only a few days. The smoker must be stopped completely, not even one cigarette per day permitted.

The person who can stop at any given moment is rare, according to the article. The usual smoker should pick a target date, such as a weekend away from smoking associates. He should publicly state his intentions to stop on this day.

On D-Day, the patient should realize that the first eight hours are the most important. Gum and candy are helpful substitutes. Cooperation of the smoker's wife must be obtained, since she will bear the brunt of the patient's irritation. The hardest step will be to abstain when in the company of smoking friends, when the desire for cigarettes is greatest. Hence it is important that the smoker inform his friends that he is about to stop—and then do so.

Dr. Leo H. Bartemeier, medical director of Seton Psychiatric Institute, Baltimore, also was quoted in the magazine concerning his study on what makes a heavy smoker.

Oversmoking, overeating, and overdrinking, he found, all stem from the basic emotional need to obtain satisfaction, relieve tension, and protect against anxiety. The food, the cigarette, the alcohol is a sedative. Where one is used to excess and it is taken away, the individual usually turns to some other form of self-indulgence.

Athletic Problem Comments Requested

The Committee on Athletics invites suggestions or comments relative to athletic problems which may confront the school, team, or practicing physician in Texas. All questions or inquiries will be answered, according to Dr. Robert G. McCorkle, chairman of the Committee on Athletics.

Information for Authors

Material for the *Texas State Journal of Medicine* may be sent to the Managing Editor, 1801 North Lamar Blvd., Austin 78701. It must be offered solely to this journal. Each paper is reviewed by a consultant specialist and an editorial board, and is accepted or rejected on the basis of its individual merit and the availability of other material.

Manuscripts should be typed double-spaced with ample margins on firm paper. Carbons are unacceptable, but the author should keep a copy.

The *title* should include the words most suitable for indexing the article in "Index Medicus," should stress the main point, and should be brief. Subtitles should not be connected grammatically with the main title.

The *authors' names* and degrees should follow the title, and their mailing addresses should be listed at the end of the article. A glossy picture of the senior author is needed.

The *text* should be narrative with complete sentences, *few abbreviations*, and logical subheadings. For *spelling and usage* the editors follow "Dorland's Illustrated Medical Dictionary," 23rd edition, and "Webster's Third New International Dictionary, Unabridged." *Quotations* must give credit to both author and source. A concise *summary* or conclusion is of professional value.

Reference lists, alphabetized by authors, should contain in this order (1) author's last name, initials, (2) title of article or book, (3) name of journal, (4) volume and first page numbers, written 33:156, (5) month of issue, (6) year. For books, the title should be followed by place of publication, name of publisher, and year.

Illustrations should be black and white drawings or positive photographs, with neat, uniform, fairly large lettering. A label pasted to the back of each illustration should indicate its number, top, author's name, and title of article in brief. *Legends* should be in complete sentences, numbered, and typed on a separate sheet. *Color illustrations* are authorized if they are of special value and if the author arranges to pay part of the extra expense incurred.

Tables should be typed on separate sheets. Column headings should show points of similarity; side headings, points of difference.

All material is subject to *editing*, but authors receive proofs to check before publication. Factual and typographical errors should be corrected, but because resetting type is costly, further revisions should be kept to a minimum. Prompt return of proofs to the *Journal* office avoids delay in publication.

Reprints are available at cost plus 2 per cent sales tax. Authors automatically receive order blanks when their articles are published.

Permission should be obtained from the publishers and the authors for any previously published material (extensive textual matter, illustrations, tables) used. Short verbatim quotations in the text may be used without permission, but should be quoted exactly with the source credited in the bibliography. Copies of permission letters should be submitted with the manuscript.

18th Symposium on Cancer Research To Be Held at M. D. Anderson in Houston

A SYMPOSIUM TO consider radiation effects in the cell and possible implications for cancer therapy will be held March 2-4 at the Shamrock Hilton Hotel in Houston.

Sponsored by The University of Texas M. D. Anderson Hospital and Tumor Institute and cosponsored by The University of Texas Graduate School of Biomedical Sciences at Houston, Texas Division of the American Cancer Society, and the National Cancer Institute, the topic of the 18th annual symposium on fundamental cancer research will be "Cellular Radiation Biology." Chairman of the symposium is Robert J. Shalek, Department of Physics, M. D. Anderson Hospital.

Registration will begin Sunday, March 1, at 2 p.m. in the Shamrock Hilton Hotel. The registration fee is $5.00 payable in advance or at the time of registration. The fee will be waived for students, including residents and fellows who preregister prior to March 2.

Papers to be presented at the three-day symposium include "The Production of Photoproducts of Thymine, Uracil, and Their Derivatives," Harold E. Johns, Toronto, Canada; "Repair of Ultraviolet Photoproducts and Radiation Products in DNA," Paul Howard-Flanders, New Haven, Conn.; "Transient Species Formed by Radiation," John W. Boag, Northwood, England; "Effects of Ionizing Radiation on Macromolecules, In Vitro and In Vivo Evidence," Franklin Hutchinson, Ph.D., New Haven, Conn.

"The Effects of UV Upon Protein and Nucleic Acid Synthesizing Systems," Lawrence Grossman, Waltham, Mass.; "Ultraviolet Light Effects on Deoxyribonucleic Acid Replication," Charles O. Doudney, Houston; "Radiosensitivity of Nuclear ATP Synthesis and Its Relation to Inhibition of Mitosis," H. M. Klouwen, Rijswijk Z. H., Netherlands; "Radiation Effects Related to Chromosome Damage, Restitution, Structure, and Function," Sheldon Wolff, Oak Ridge, Tenn.

"Disassembly of Mitotic Organelles with Subcellular Microbeams," Raymond E. Zirkle, Ph.D., and Robert B. Uretz, Chicago; "Relationship Between Chromosome Volume and Radiation Sensitivity in Plant Cells," Arnold H. Sparrow, Ph.D., Long Island, N. Y.; "Studies on Halogenated DNA as Related to the Nature of the Critical Radiosensitive Structures," Waclaw Szybalski, Madison, Wis.; "Differences in the Radiosensitivity of Cells: Role of Critical Structures Versus Recovery and Intracellular Protection Phenomena," Peter Alexander, London.

"Interactions of Modifying Treatments and the Light They Throw on Targets for Cell Death," Tikvah Alper, London; "Physicochemical Bases of Radiation Sensitivity in Cells," E. L. Powers, Argonne, Ill.; "The Mechanism of Action of the S-Containing Radiation Protective Agents," L. Eldjarn, Oslo, Norway.

"Radiosensitivity and Recovery in Relation to the Cell Cycle," William C. Dewey and Ronald M. Humphrey, Houston; "Changes in X-Ray Sensitivity of HeLa S3 Cells During the Division Cycle," L. J. Tolmach, St. Louis; "Recovery Phenomena," G. F. Whitmore, Toronto, Can.; "Recovery Phenomena and Their Implications for Radiation Therapy of Malignant Disease," Dr. Mortimer M. Elkind, Bethesda, Md.

"The Importance of Cell Population Kinetics in Determining Response to Irradiation of Normal and Malignant Tissue," L. G. Lajtha, Manchester, England; "The Kinetics of Tumor Cell Proliferation," Dr. Mortimer L. Mendelsohn, Philadelphia; "Radiation Response of C3H Mouse Mammary Carcinoma Interpreted in Terms of Cellular Sensitivity," Dr. Herman D. Suit and Robert J. Shalek, Houston; "Physical, Biological, and Clinical Aspects of Fast Neutron and Other High LET Radiations and Implications for Fast Neutron Beam Clinical Radiotherapy," Dr. J. Robert Andrews, Bethesda, Md.; and "Clinical Potentialities of Recent Advances in Cellular Radiobiology," Dr. Henry S. Kaplan, Palo Alto, Calif.

The Bertner Foundation Lecture and the Bertner Foundation Award for an outstanding contribution related to cancer research will be presented March 3.

Information regarding the 18th symposi-

um on Fundamental Cancer Research may be obtained from the Publications Department, M. D. Anderson Hospital, Texas Medical Center, Houston 25. The proceedings of the symposium will be published under the title, "Cellular Radiation Biology."

Annual Session To Feature Curbstone Consultations, Films

CURBSTONE CONSULTATIONS and the showing of scientific films are again features of the TMA Annual Session.

The Houston meeting scheduled for April 23-26 will also include 32 guest speakers, 14 refresher courses, 11 scientific sections, 2 general sessions, 200 scientific and technical exhibits, 27 related and specialty group meetings, several special programs, and a variety of entertainment.

Requests for hotel or motel reservations are now being accepted. These should be addressed to the housing bureau at the Shamrock Hilton Hotel. A tear-out post card is enclosed in this issue of the *Journal* for the convenience of readers who wish to make reservations. The card may be found opposite page 166.

Approximately 75 gastroenterologists are expected to attend Dallas and Houston meetings and many will fly to Mexico City for a third meeting near Annual Session time. The American Gastroenterological Association is meeting in Dallas on April 23, 24, and 25; TMA's Section on Digestive Diseases will sponsor a program on April 26 in Houston; and the Mexican Association of Gastroenterology has planned a program for April 28 and 29 in Mexico City. Special arrangements have been made for air transportation so that physicians may attend all three meetings. Complete details of the air itinerary and the process for making reservations was published in the November, 1963, issue of the *Journal* on p. 1099. Additional information may be obtained by writing Dr. John R. Kelsey, 6655 Travis, Houston.

The Curbstone Consultations will be held Friday evening, April 24, from 8 to 9:30 in the Shamrock Hilton Hotel. These will be person-to-person conversational discussions of cases or problems between the round-table "experts" and the physicians visiting the tables. Eleven tables are planned at which 11 separate subjects of medical interest will be discussed. Topics of discussions will be "Present Status of Surgery of the Heart," "Inhalation Therapy in Lung Disease," "Modern Concepts in the Management of the Post Menopausal Patient," "Use of Oral Agents in Diabetes Mellitus," "Backache," "Vaginitis," "Diarrhea," "Newer Concepts in Immunizations," "Adolescent Problems," "Psychiatric Problems in Clinical Practice," and "Cardiac Emergencies."

The Annual Session Film Program is scheduled for Friday and Saturday, April 24-25, during the hours 10 a.m. to 1 p.m. Fourteen films have been selected and include four by Texas physicians.

Films by Texas physicians are "The Painful Breast" by Dr. James L. May of Irving, "Principles of the Local Burn Wound" by Dr. Truman G. Blocker, Jr., of Galveston, "Early Plastic Repair of Finger Tip Injuries in Children" by Dr. Bromley Freeman of Houston, and "Penetrating Wounds of the Large Intestine" by Dr. George C. Morris, Jr., of Houston.

Other films to be shown are "Clinical Medical Electronics," "Technique of Total Abdominal Hysterectomy," "Emergency Airways-Crisis in Action," "Stroke—Early Restorative Measures in Your Hospital," "A New Diagnostic Technique for Stroke: Retrograde Cerebral Angiography," "The Doctor Is Cross-Examined," "Football Injuries —Cause and Prevention," "One Who Heals," "Is Smoking Worth It?" and "Human Growth."

U. S.-Mexico Border Group To Meet March 2-5

The United States-Mexico Border Public Health Association will hold its annual meeting in Monterrey, N. L., Mexico, March 2-5.

Texas physicians who will give scientific presentations include Dr. James E. Peavy of Austin, Commissioner of Health, and Dr. David M. Cowgill, director of the Lubbock City Health Department.

Dallas Southern Clinical Society To Hold 33rd Spring Conference

THE DALLAS Southern Clinical Society will hold its 33rd annual spring clinical conference March 16-18 at the Statler Hilton Hotel in Dallas.

Acceptable for 15 hours credit, American Academy of General Practice, the conference will include general assemblies, scientific exhibits, panel discussions, round-table luncheons, technical exhibits, a dinner dance, postgraduate lectures, informal breakfast discussions, and grand rounds.

Guest speakers include, on pediatrics, Dr. John D. Bridgers, High Point, N. C., "Child Health and Development: The Two Sides of the Pediatric Coin," "Pediatric Interviewing as Disciplined Dialogue," and "Management of 'Stress-sensitive' Disorders in Children";

On surgery, Dr. Bentley P. Colcock, Boston, "The Indications for Surgery in the Treatment of Thyroid Disease" and "The Indications for Surgery in Diverticulitis"; Dr. Edwin H. Ellison, Milwaukee, "Acute and Chronic Pancreatitis" and "Pathogenesis, Recognition and Management of the Zollinger-Ellison Syndrome"; and Dr. Orvar Swenson, Chicago, "Intestinal Obstruction in Children," "The Diagnosis and Management of Congenital Megacolon," and "Appendicitis in Children";

On urology, Dr. J. Hartwell Harrison, Boston, "Management of Carcinoma of the Prostate" and "The Diagnosis and Treatment of Tumors of the Kidney";

On dermatology, Dr. John R. Haserick, Cleveland, "Dermatologic Conditions Associated with Internal Disorders" and "The Acne Problem";

On ophthalmology, Dr. William F. Hughes, Chicago, "Treatment Dangers to the Cornea," "Management of Keratitis," and "Diagnosis and Treatment of Corneal Dystrophies"; and Dr. John M. McLean, New York City, "Diagnostic Appraisal of Glaucoma," "Surgical Management of Glaucoma," and "Surgical Management of Congenital Cataract";

On internal medicine, Dr. John P. Merrill, Boston, "Transplantation of the Kidney" and "Renal Hypertension"; and Dr. George E. Schreiner, Washington, D. C., "Nephrotoxic Nephritis" and "Renal Biopsy, Correlation of Structure and Function";

On obstetrics and gynecology, Dr. Louis M. Hellman, Brooklyn, "Premature Separation of the Placenta" and "Urinary Incontinence"; and Dr. Ben M. Peckham, Madison, "Management of Advanced Ovarian Carcinoma" and "Current Management of Uterine Bleeding";

On orthopedics, Dr. Daniel C. Riordan, New Orleans, "Treatment of the Arthritic Hand," "Carpal Tunnel Syndromes," and "Principles in the Care of Acute Hand Injuries";

On otolaryngology, Dr. Jerome A. Hilger, St. Paul, "Sudden Deafness," "Bell's Palsy," and "Laryngopharyngeal Cancer"; and Dr. William H. Saunders, Columbus, "Surgical Treatment of Atrophic Rhinitis," "Differential Diagnosis of Meniere's Disease," and "Septal Dermoplasty for Hereditary Hemorrhagic Telangiectasia and Certain Other Nasal Conditions";

On psychiatry, Dr. Martin Stein, New York City, "Psychosomatic Aspects of Asthma," "Psychotherapy in Medical Practice," and "A Consideration of the Etiology of Depression";

On pathology, Col. Frank M. Townsend, USAF (MC), San Antonio, "Polyps of the Colon—Problem and Prognosis," "The Physician's Role in Accident Evaluation and Prevention," and "Carcinoma in Situ of the Cervix—Two Decades' Experience at the Armed Forces Institute of Pathology";

And on radiology, Dr. Stanley M. Wyman, Boston, "The Problem of the Enlarged Pulmonary Hilus" and "Radiology of the Acute Abdomen."

Biliary tract disease will be discussed by Dr. Bentley P. Colcock and Col. Frank M. Townsend on grand rounds.

Members of the panel on renal failure include Dr. J. Hartwell Harrison and Dr. John P. Merrill.

Dr. Edwin H. Ellison and Dr. Stanley M. Wyman will be on a panel to discuss the complicated peptic ulcer.

Dr. John R. Haserick and Dr. George E. Schreiner will be on the panel on systemic lupus.

Members of the panel on toxemia of pregnancy include Dr. Louis M. Hellman and Dr. Ben M. Peckham.

Physicians, School Administrators Hear Talk on Interprofessional Relations

TO DISCUSS the problem of classifying school children for physical education and to suggest possible solutions, 233 school administrators and physicians met Jan. 9 at the Conference on Total Fitness of the School Child in Austin. The meeting was sponsored by the Texas Medical Association and the Texas Association of School Administrators, in cooperation with the Texas Education Agency.

In his address, "Interprofessional Relationships in Fitness," Dr. Donald A. Dukelow, assistant director in the Department of Community Health and Health Education of the AMA, told administrators and physicians that communication problems in school health programs can seriously affect children.

"Health to a physician is the absence of disease or pathology. . . . I have heard an educator define health as a positive quality, as 'something without which kids aren't in school'."

"If the school administrator, the health officer, and the physician in practice each attempts to carry out his own prerogatives as he sees them, without an understanding of the other fellow's responsibilities, and without agreement among themselves as to the responsibility of each and the relation of each to the other, it is very easy to develop a situation where the irresistible force meets the immovable body," Dr. Dukelow said.

Problems in health appraisal of students concern to what extent teacher observation is used and teacher-nurse conferences are carried on as a means of evaluating student problems; who should examine the child; and what techniques are used, and by whom, to inform parents of a child's need for medical care.

To define the problem of classifying children for participation in physical education, a school superintendent and a physician gave their points of view.

Dr. Irby B. Carruth, superintendent of Austin schools, said that the school administrator finds this is a question mostly of attitude of physicians and parents. To get cooperation from both physicians and parents for a school health program, Dr. Carruth suggested that schools form health committees composed of doctors and laymen.

Dr. P. E. Luecke, a Dallas pediatrician, said that physicians should be aware of school health programs in order to make intelligent decisions regarding classification of students for participation.

Following the addresses, physicians and school administrators formed panel groups to discuss the problem of classifying students for physical education and school health programs.

Nuclear Medicine Society Sets Houston Meeting for March 6-8

The Southwestern Chapter of the Society of Nuclear Medicine will hold its ninth annual meeting at the Shamrock Hilton Hotel in Houston on March 6-8.

The scientific program will present new developments in the field of radioisotopes in the field of medicine and biology, discussions of current clinical application and established uses of radioisotopes in medicine and biology, and the industrial application of radioisotopes which have medical or biological significance.

Technical exhibits will be on display at the hotel during the meeting. The annual banquet will be March 7.

All physicians and others interested in nuclear medicine may attend. Additional information may be obtained by contacting Dr. J. R. Maxfield, Jr., secretary, 2711 Oak Lawn, Dallas.

Dallas Hospital Announces Staff Publication

The first edition of a publication carrying articles by members of the medical education department at Methodist Hospital of Dallas has been issued by the hospital's medical staff.

The *Bulletin of the Methodist Hospital of Dallas Medical Staff,* it is anticipated, will be an annual publication, reports Dr. James E. Gleichert, chairman of the editorial committee. Others on his committee are Drs. Charles E. Van Cleave and Joseph C. Ogle.

New Orleans Assembly Announced for March 2-5

The 27th annual meeting of the New Orleans Graduate Medical Assembly will be held March 2-5 at the Roosevelt Hotel.

Nineteen outstanding guest speakers will take part, and their presentations will be of interest to both specialists and general practitioners. The program will include 55 discussions on topics of current medical interest, in addition to clinicopathologic conferences, symposiums, medical motion pictures, round-table luncheons, and technical exhibits.

Following the meeting in New Orleans, arrangements have been made for a clinical tour to Europe leaving via air on March 7. The itinerary includes visits to Lisbon, Madrid, Rome, Vienna, Berlin, and Paris. The group will return on March 28.

Details of the New Orleans meeting and the tour are available at the office of the Assembly, 1430 Tulane Avenue, New Orleans, La. 70112.

Postgraduate Courses

Obstetrics and Gynecology, Oklahoma City, March 12-13.—Emphasis of the symposia will be placed on the indications, contraindications, and techniques for operative procedure in gynecology and obstetrics.

The course was developed by the Oklahoma Medical Center's Department of Gynecology and Obstetrics and the Department of Continuing Education.

Guest faculty will include Dr. Reuben H. Adams, associate professor of obstetrics and gynecology, Southwestern Medical School; Dr. Raphael B. Durfee, associate professor of obstetrics and gynecology, University of Oregon School of Medicine; Dr. Isadore Dyer, professor of obstetrics, Tulane University School of Medicine; and Dr. John D. Thompson, professor and chairman of gynecology and obstetrics, Emory University School of Medicine.

Registration information and a program may be obtained from the Office of Postgraduate Education, University of Oklahoma Medical Center, 801 NE 13, Oklahoma City 4, Okla.

Trauma, San Antonio, March 9-13, 1964.—The course will be held at Brooke General Hospital, Brooke Army Medical Center, Fort Sam Houston.

The program is designed to present current trends and newer concepts in the management of trauma as well as elaboration upon previously accepted principles.

"He's really a very modern and up-to-date doctor . . . even the magazines in his waiting room are dated 1964."

Among the subjects to be discussed are shock and resuscitation, wound infections and antibiotics, management of burn injury, abdominal trauma, thoracic and cardiovascular injuries, combined thoracic-abdominal injuries, injuries of the pancreas, spleen, and liver, injuries to the brain, spinal cord, and peripheral nerves, urological injuries, maxillofacial and ocular trauma, and multiple injuries.

There will be a panel and lectures on various orthopedic injuries, including fractures, sprains, back, and tendon injuries.

Army medical service officers on active duty and civil service physicians may apply to SGO, DA, Washington 25, D. C., Attn: MEDPT-TC, eight weeks prior to the course.

Reserve and National Guard may apply through Headquarters, U. S. Army Corps Commander, or to the Chief, National Guard Bureau, Washington, not later than eight weeks prior to the date of the course.

Civilian physicians or dentists may apply to CG, Brooke General Hospital, Attn: Chief, Department of Surgery.

Information may be obtained from Commanding General, Brooke General Hospital, Attn: Chief, Department of Surgery.

Laboratory Workshops, Austin, March and April.—The Texas State Department of Health will sponsor two workshops during March for laboratory technologists.

Enteric bacteriology, March 9-13, will deal with the various enteric pathogens such as *Salmonella, Shigella,* pathogenic *E. coli* serotypes, and commonly encountered paracolons. These organisms will be considered both from a biochemical and serological standpoint. The technologist should have some experience in bacteriology as a prerequisite.

Introductory virology, March 30, 31, April 1, and 2, will be concerned with representative procedures useful in diagnostic virology. Only a small group can be accommodated.

There is no registration fee for the workshops, but admittance is by advance registration.

Anson Jones Award Winners Announced

Winners have been announced for the Texas Medical Association's 1964 Anson Jones Award.

The *Fort Worth Star-Telegram* and its medical reporter, Blair Justice, have taken top honors in metropolitan daily newspaper reporting of health and medical information. Winner of the TMA award the first year it was established in 1957, Mr. Justice has now won the honor five times in the past eight years. His entry included medical coverage of President Kennedy's assassination, plus medical stories sent directly from such places as Boston, New York, Philadelphia, Atlantic City, New Orleans, La Jolla, Calif., and throughout the state of Texas.

The *Dallas Morning News* and feature writer, Helen Bullock Callaway, received honorable mention in this category. This marks the first time TMA has given an honorable mention award since separate categories were established four years ago. Mrs. Callaway won top honors in 1958 when she was medical writer for the *News.* Her entry consisted of five feature articles published in 1963 on (1) child abuse, (2) a ten-year-old girl born without arms, (3) advance of modern psychiatry, (4) drug therapy, and (5) hormone treatment of dwarfs.

The *Odessa American* and staff writer, John Sliney, are winners for reporting in smaller daily newspapers. Sliney's entry consisted of ten articles of medical interest in the Odessa area, including such topics as the harmful effects of sun, a two-pound baby's fight for life, drug treatment for cystic fibrosis, protecting eyes from viewing the sun eclipse, radiation treatment of cancer, pathology, heart disease, anesthesiology, eye transplants, and emergency room treatment.

The *San Marcos Record* and its news editor, Tom Buckner, were first in weekly newspaper reporting. Buckner's entry consisted of a six-article series on alcoholism, written by a local member of Alcoholics Anonymous who once had a promising writing career. The series had great impact on the community and resulted in a significant increase in the membership of the local AA chapter.

The *Humble News,* edited by Gene D. Legler, won for magazine reporting. The Humble Oil and Refining Company publication is a monthly magazine that goes to some 50,000 employees in Humble's operating areas. Their entry consisted of feature articles on pesticides, water pollution, poison control centers, cancer check-ups, emotional disturbances, flu protection, and the body's need for water.

Twenty-six entries were received in the award competition this year. Divided into four categories, the award is given for excellence in reporting health information to the public. Categories are: I—daily newspapers in cities of 300,000 population or more; II—daily newspapers in cities of less than 300,000 population; III—weekly newspapers; and IV—magazines, company publications, and trade journals in non-health fields.

All winners receive an award certificate, and the four top-place winners' publications each receive an engraved plaque for the role they played in making the coverage of medical news possible. Cash prizes for the winners total $600.

The Anson Jones Award was created by the Texas Medical Association in 1956. It is named in honor of Dr. Anson Jones, a physician, statesman, writer, and last President of the Republic of Texas.

Judges this year were Norris G. Davis, Ph.D., professor of journalism, the University of Texas; Moselle Boland, medical reporter, *Houston Chronicle,* and last year's Category I Anson Jones Award winner; Wilbourn McNutt, science editor, *Austin American-Statesman,* and Category II Anson Jones winner last year; and Mrs. Marilyn Baker, Managing Editor, *Texas State Journal of Medicine.* A special group of physicians gave final approval to the winners selected.

Benzodiazepines

THESE compounds constitute a relatively new chemical class of psychotherapeutic agents which are unrelated in structure to the phenothiazines or meprobamate types of psychotropic drugs. Although their precise mode of action at the molecular level is obscure and few generalizations can be made in this regard, with respect to their gross physiologic manifestations these agents produce anti-anxiety, sedative, and muscle relaxant effects.

Chlordiazepoxide (Librium)

One of the benzodiazepine derivatives which is presently employed as a psychotherapeutic agent is chlordiazepoxide hydrochloride (Librium). Chemically the compound is 7-chloro-2-methylamino-5-phenyl-3 H-1, 4-benzodiazepine 4-oxide and may be described structurally as shown in Fig. 1.

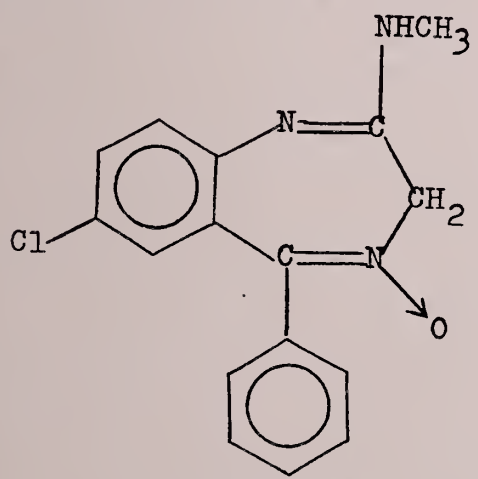

Fig. 1. Chlordiazepoxide.

One might speculate that despite its apparent structural dissimilarity to the phenothiazine tranquilizers, certain common structural features may be visualized. For example, in the formula shown in Fig. 1 there is a basic moiety, (the $NHCH_3$ group) separated by an appropriate distance from the two large aromatic rings, a situation somewhat analogous to that in the phenothiazines where the distance between the aromatic rings and the amine function has been shown to be necessary for tranquilizing activity. However, despite this structural similarity, present evidence of differences in the overall spectra of activity of the drugs makes it questionable whether chlordiazepoxide and the phenothiazines act by a similar mechanism.

Although the site of action of chlordiazepoxide has not been clearly defined, there are some indications that it may act on the reticular system and its hypothalamic connections. In contrast with reports concerning the action of meprobamate, this drug does not act interneuronally; however, it appears to have similar muscle relaxant properties[1]. In an attempt to localize the action of the drug on higher portions of the central nervous system, animal studies indicate that depressant activity on the limbic system occurred at lower doses than on the reticular activating system. Since the former system and particularly the amygdala are believed to be concerned with emotional responses, it appears possible that the depressant effects of the psychotherapeutic agent on these areas could be related to the anti-anxiety effects in human subjects.[12,16] The work of Randall and others shows that chlordiazepoxide produces muscle and sedative effects in mice. It depresses avoidance responding and spontaneous locomotion activity of normal rats and has a calming effect on rats made irritable by lesions in the septal area of the brain. Ataxia is produced in dogs at doses below those causing sleep. In monkeys, a loss of aggressive behavior was observed at doses which did not depress general activity or avoidance behavior.[11] Randall and others also showed that chlordiazepoxide has in common with meprobamate, chlorpromazine, and phenobarbital anticonvulsant properties in mice and the ability to produce muscle relaxation in mice and cats. It differs

from meprobamate and phenobarbital in lacking hypnotic effect in high doses in animals and resembles chlorpromazine in this respect. Animals show ataxia but not true hypnosis at high doses of the drug. Chlordiazepoxide has not been observed to have the powerful autonomic blocking effects as do chlorpromazine and reserpine.[10,11,12] According to Boris,[3] this psychotherapeutic agent, unlike the phenothiazines, exhibits no gonadal hormone-like activity, anti-gonadal hormone activity, or anti-goitrogenic activity; nor does it appear to interfere with normal functioning of the pituitary gonadal axis.

Chlordiazepoxide is reported to be absorbed readily from the gastrointestinal tract, and this may partially explain the drug's quick clinical response. Blood levels show a half-life of two and one-half days in humans and four hours in rats. Only small amounts appear in the urine as unchanged drug.[12] An inspection of the molecule discloses that embodied is an amidine group. Normally the high basicity of this moiety would indicate that the compound might be present in the salt form in the gastrointestinal tract, and this would preclude the rapid absorption of the drug. However, there is present also in the molecule an electron-withdrawing amine oxide group which is in conjugation with this amidine group and which accordingly may lower the basicity of the latter group. This structural arrangement may promote the molecular species which is rapidly absorbed.

Chlordiazepoxide has found extensive clinical application in psychiatric disorders. The study of Tobin and others on the evaluation of the drug in the treatment of anxiety reactions shows that it has two different pharmacologic actions, namely, an anti-anxiety and a sedative effect. In addition, the drug may exert a psychostimulating effect which becomes manifested by a feeling of well-being, increased interest in social activities, verbal productivity, appetite, and in the lifting of anxious depressions.[19]

Although chlordiazepoxide has been found to be effective in long standing anxiety and phobic reactions, Bowes found it to be specifically indicated in acute situational anxiety tension states.[4]

McFarland reported the use of this psychotherapeutic agent for spasticity in a double blind study. The results showed that the drug was statistically more effective than a placebo in relieving spasticity; however, this investigator states that the spasmolytic effectiveness of chlordiazepoxide compared to other preparations remains to be demonstrated.[9]

In a study using the drug in the treatment of psychotics with convulsive disorders, Barsa[2] showed that it has definite anticonvulsant properties, as shown by control of seizures and by electroencephalogram changes; however, it is frequently not as effective as the combination of Dilantin and phenobarbital. As a tranquilizer its greatest usefulness is in those cases of less severe anxiety or those cases where preservation of mental alertness is important because of patients' intense fear of losing control of themselves.

In alcoholic patients, chlordiazepoxide has been employed successfully as an aid to psychotherapy in long-term rehabilitation. It is administered parenterally to control the acute withdrawal symptoms, delirium tremens, and hallucinosis.[5]

Diazepam (Valium)

Diazepam is a newly developed psychotherapeutic agent of the 1, 4-benzodiazepine class. This analogue of chlordiazepoxide is reported to have an action qualitatively similar to that of the parent compound, but is more potent in most laboratory animal tests.[13] It has approximately five times the potency of chlordiazepoxide as a tranquilizer and muscle relaxant as shown by standard animal tests. In acute toxicity it is reported to be about equal to chlordiazepoxide.[13] The new derivative is a colorless crystalline compound, insoluble in water, and has the chemical name 7-chloro-1-methyl-5-phenyl-3H-1,4-benzodiazepin-2(1H)-one. Its structure may be represented shown in Fig. 2.

A comparison of this structure with that of chlordiazepoxide reveals a gross similarity; however, upon close inspection, differences in the molecules may be discerned. In place of the amidine moiety found in chlordiazepoxide, there is an amide group. Consequently, it is not surprising that pharmacologic tests have indicated an action which is qualitatively different from the parent product.

In an extensive study where the new drug was compared with chlordiazepoxide, it was found that diazepam is generally more potent than Librium in many pharmacologic tests

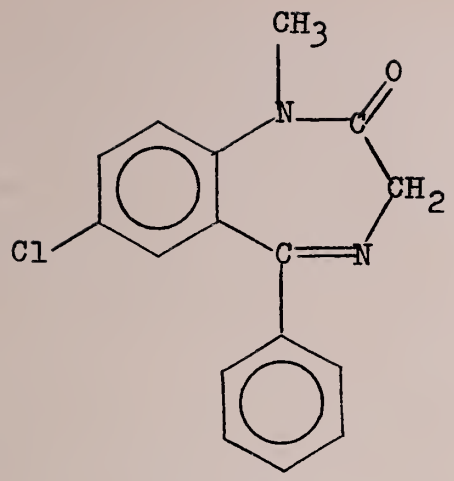

Fig. 2. Diazepam.

for central nervous system activity. It is more potent than the parent drug in anticonvulsant tests as tested against strychnine, Metrazol, or electroshock convulsions in mice. It also is more potent as a muscle relaxant in mice and cats and more potent in reducing fighting in mice induced by electroshock. In addition, it is more active in relaxing decerebrate rigidity in cats, depressing electroencephalograms of cortex and the limbic system, and in inducing sedation and ataxia. In rats there is an equivalence of efficacy between the two psychotherapeutic agents. This is observed in the locomotor activity, irritability of septal rats, and in numerous conditioned avoidance tests in which the animals were required to press a lever to avoid electroshock. The results show that the drug is less active than the parent agent and chlorpromazine, about the same as pentobarbital, and more potent than meprobamate in reducing locomotor activity in rats. Diazepam and chlordiazepoxide are equally active in the monkey, both producing "taming" as defined by the aggression/activity ratio at doses well below those which produce ataxia.[13]

Nine patients included in a clinical study were under treatment for the following neuropsychiatric disorders: three, anxiety reaction; three, involutional syndrome; one, anxiety with depression; one, psychotic reaction; and one, schizophrenia. Those patients with anxiety reactions, anxiety with depression, and involutional syndromes had an excellent clinical response to diazepam; however, the two remaining subjects were not improved. The investigators state that such a response would be anticipated in patients with these diagnoses. Although seven patients showed minor subjective side effects such as dizziness, incoordination, fatigue, and rash, in many cases the side effects disappeared when the medication was continued.[13]

Sussex and others administered diazepam in divided doses of 6 to 30 mg. daily to 13 patients all selected as representing a syndrome characterized by paranoid and affective features and dominated clinically by symptoms of anxiety and depression. Previously all the patients had received a variety of treatment from various sources and none, with the exception of two patients who had been hospitalized, had shown any significant improvement even for short periods of time. Improvement was observed in all symptomatic areas in 12 patients, but one subject was clearly unimproved after reaching dosage levels of 60 mg. daily for a period of almost four months. However, this patient was the only one of the group whose illness, although of acute recent onset, showed prominent catatonic features. The most obvious clinical difference in the improved patients was the marked decrease in both anxiety and depression.[18]

In a comparative study of diazepam and chlordiazepoxide in psychoneurotic patients, Darling found that both drugs were similar in action and results and interchangeable in almost all cases; however, diazepam appeared to be smoother-acting and easier to handle. He stated that in his opinion neither drug is an antischizophrenic agent. In the case of a few alcoholics and drug addicts, diazepam seemed remarkably effective in controlling anxiety during and after the withdrawal period much more so than chlordiazepoxide. However, in these cases, it was a two-edged sword, because when patients resumed drinking or taking drugs the concomitant use of diazepam greatly potentiated their intoxications.[6]

In a clinical study by Farb, 34 patients seen in private psychiatric practice for various refractory disorders including phobic reaction, depressive reaction, involutional syndrome, schizophrenic reaction, hysterical reaction and others were administered diazepam intravenously as a pre-interview medication. The findings indicate that the new drug is highly effective as a pre-interview medication to reduce emotional symptoms during the psychotherapy session. The side effects noted do not appear to adversely affect symptomatic response.[7]

Although the spectrum of this new drug overlaps that of the parent compound be-

cause amelioration of anxiety and tension has been noted in a variety of neuroses in which these symptoms were prominent, it also appears to be of value in the relief of muscle spasm in cerebral palsy and athetosis.[14, 15] The manufacturer of diazepam has indicated that the drug is of no value in dealing with psychotic patients manifesting anxiety and should be avoided when there is reason to believe the patient is psychotic. In elderly or debilitated patients, it is important to limit the dosage to the smallest effective amount to preclude the development of ataxia or oversedation (not more than 1 mg. one or two times daily initially, to be increased gradually as needed and tolerated).[8]

The drug's usual daily dose is as follows:

Mild to moderate psychoneurotic reactions: 2 to 5 mg. 2 or 3 times daily.

Severe psychoneurotic reactions: 5 to 10 mg. 3 or 4 times daily.

Muscle spasm, associated with cerebral palsy or athetosis: 2 to 10 mg., 3 or 4 times daily.

Alcoholism: 10 mg. 3 or 4 times during the first 24 hours; reducing to 5 mg., 3 or 4 times daily as needed.

Newer Benzodiazepines

In further studies the synthesis of a number of chlordiazepoxide and diazepam analogues has been reported. According to preliminary pharmacologic data the 7-nitro analogue of diazepam is a potent muscle relaxant, sedative, and anticonvulsant, being approximately seven times as active in the mouse and 20 times as active in the cat as chlordiazepoxide.[17]

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Four-Day Meet Set in March By American College of Surgeons

Surgeons and graduate nurses are invited to the tenth annual joint meeting of the American College of Surgeons in New Orleans, March 16-19. Headquarters hotel for doctors will be the Roosevelt, and for nurses, the Jung.

This is the College's only four-day meeting for 1964 and the only meeting with a program for nurses.

Texas physicians taking part on the program include the following:

From Houston, Drs. George L. Jordan, Murray M. Copeland, John S. Stehlin, Jr., E. Stanley Crawford, Everett L. Goar, George R. Kolodny, Edwin E. Garrett, Frederick R. Guilford, Herbert H. Harris, Thomas D. Cronin, Bromley S. Freeman, Denton A. Cooley, George C. Morris, Jr., J. W. Harris, Russell Scott, Jr., John A. Wall, C. Denton Kerr, and Felix N. Rutledge.

From Galveston, Truman G. Blocker, Jr., Edgar J. Poth, Stephen R. Lewis, and Charles A. Hooks.

From Dallas, Robert S. Sparkman, Donald A. Corgill, Lyle M. Sellers, Donald L. Paulson, Robert J. Rowe, William K. Clark, Tom E. Smith, Harry M. Spence, and Vanda A. Davidson.

From McAllen, Dr. Berton F. Frink.

From San Antonio, Drs. James L. Mims, Jr., Charles W. Tennison, and John J. Hinchey.

From Corpus Christi, Dr. L. David Wright.
From Temple, Dr. G. V. Brindley, Jr.

Advance housing registration forms, giving hotel rates and registration hours, may be obtained by writing to the College headquarters: Mr. T. E. McGinnis, American College of Surgeons, 55 East Erie Street, Chicago 11.

Santa Rosa Sets Conference

The Children's Hospital of Santa Rosa Medical Center, San Antonio, will hold its annual teaching conference March 14-15.

Speakers will be Drs. Irving Shulman, Jersey City, and Orvar Swenson, Chicago. There is no registration fee, according to Dr. Cornelius H. Nau, chairman, Department of Pediatrics.

New Films in TMA Library

The Painful Breast.—Sound, color, 12 minutes, 1963.
Basic Principles of the Local Burn Wound.—Sound, color, 24 minutes, 1963.

A PROGRAM IS outlined for management of the local burn wound which consists, in essence, of protection of the wound; mechanical cleanliness by repeated soap-and-water washing combined with conservative debridement; employment of large sheets of skin, where possible for grafts, and simplified techniques for handling of both transplants and donor sites.

New Books in TMA Library

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Menninger, K.: Vital Balance, New York, Viking Press, 1963.

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Cartwright, G. E.: Diagnostic Laboratory Hematology, 3rd ed., New York: Grune & Stratton, 1963.

Bryson, J. G.: Reminiscences of a Country Doctor, Waco, Texian Press, 1963.

Schreiner, G. E., ed.: Transactions American Society for Artificial Internal Organs, vol. 9, 1963, Washington, D. C., Georgetown University Printing Department, 1963.

Akademiya Nauk U.S.S.R., Works of the Pavlov Institute of Psychology, vol. 10, Problems of Physiology of Higher Nervous Activity and of the Nervous System, Moscow, Academy of Sciences of the U.S.S.R., 1963.

Works of the Scientific Research Institute of Biology and the Faculty of Biology, vol. 29, Physiology and Biochemistry of Aging, Jerusalem, Israel Program for Scientific Translation, Ltd., 1963.

Miracle of Life, Chicago, American Medical Association, 1961.

Wonderful Human Machine, Chicago, American Medical Association, 1961.

DeStevens, G.: Diuretics, Chemistry and Pharmacology, vol. 1, New York, Academic Press, 1963.

Smart, J. V.: Elements of Medical Statistics, Springfield, Charles C Thomas, 1963.

University of Texas M. D. Anderson Hospital and Tumor Institute, Viruses, Nucleic Acids, and Cancer, Baltimore, Williams & Wilkins Co., 1963.

Recent Deaths

DR. N. L. BLACKWELL

Dr. Nan L. Gilkerson Blackwell, the first woman physician to practice in the Panhandle, died in an Amarillo hospital of uremia and congestive heart failure on Nov. 29 after a long illness.

The wife of Dr. Ben T. Blackwell since their marriage in 1928, she was a resident of Amarillo and had practiced there from 1927 to 1952. Her specialty was eye, ear, nose, and throat treatment.

Before coming to Amarillo, Dr. Blackwell had practiced in Lubbock where she was the first physician for Texas Technological College.

A native of Guthrie, Tex., and the daughter of George W. and Mary Johnson Gilkerson, Dr. Blackwell was born Oct. 9, 1898. She received her medical degree from the University of Texas Medical Branch in Galveston. Prior to going to medical school she attended Stamford College (now McMurray College) in Abilene, Southern Methodist University, and the University of Texas.

She interned at Jefferson Davis Hospital in Houston and served her residency at Bellevue Hospital Center in New York City. She also did postgraduate work in New York Eye and Ear Infirmary in New York City and the University of Pennsylvania School of Medicine in Philadelphia.

"Dr. Nan" as she was called by patients in the High Plains area was a tireless worker in the American Association of University Women and Gamma Phi Beta Sorority which she had served as international membership chairman since 1960.

She was a Presbyterian.

The first woman president of Potter-Randall Counties Medical Society, she was elected through it to inactive membership in Texas Medical Association in 1955. She also belonged to Southern Medical Association and the American Medical Association.

She is survived by her husband of Amarillo and three sisters, Mrs. Charles Bacon, Mrs. Jimmie Wilson, and Mrs. George Langford, all of Lubbock.

DR. C. F. MONTGOMERY

Dr. Charles F. Montgomery, Jr., an orthopedist in Houston for 24 years, died at his home there on Nov. 17.

DR. C. F. MONTGOMERY

The 55-year-old physician was born and reared in Greenfield, Mo. He was a graduate of the University of Missouri at Columbia and received his M.D. degree from Duke University School of Medicine at Durham, N. C.

Dr. Montgomery interned and served a residency at Henry Ford Hospital in Detroit. Later he did postgraduate work in London, England, and at Duke University. Before coming to Houston he had practiced in Roswell, N. M., and in Baltimore, Md.

An avid hunter and fisherman, Dr. Montgomery suffered a stroke while duck hunting three years ago and had not practiced actively since that time.

He was a clinical associate professor at Baylor University College of Medicine. A member of Alpha Omega Alpha, he was affiliated with other medical groups including Harris County Medical Society, Texas Medical Association (elected to inactive membership in 1961), the American Medical Association, and the Houston Orthopedic Club.

He had published articles in the *American Journal of Surgery* and *Southern Medical Journal.*

Dr. Montgomery was a past president of the Houston alumni group of Duke University; he was a charter member of St. Luke's Methodist Church; and he was a member of Kappa Alpha fraternity.

He is survived by his wife, Mrs. Jean Bar-

num Montgomery whom he married in Detroit on Sept. 10, 1937, and two daughters, Misses Mary and Bonnie Montgomery, all of Houston.

DR. H. H. VOET

Dr. Henriette Hanna Voet, who came to the United States from Holland in 1939, just before Hitler's hordes invaded her homeland, died in an Amarillo hospital on Dec. 11 at the age of 55 from a ruptured aneurysm of the anterior communicating artery.

Her husband, Andries Voet, Ph.D., is senior research chemist with the J. M. Huber Corporation in Borger. They were married in Holland on Jan. 5, 1932, where two of their four children were born.

Dr. Voet was born in Arnhem, Holland, on Dec. 7, 1908, the daughter of Louis and Sophia van Coevorden Mogendorff.

Before she left her native country, Dr. Voet had earned her M.D. and Ph.D. degrees at the University of Amsterdam, and had been certified by the Netherlands Board of Ophthalmology. She had done postgraduate work at the University Eye Hospital in Amsterdam on the subject of dark adaptation, published in a thesis. After immigrating into the United States, she was invited to join the staff of the New York Eye and Ear Infirmary, a position she held until she moved to Texas. From 1943 to 1949, she was ophthalmologist for the City of New York.

Dr. Voet practiced ophthalmology in New York City and in Rockville Centre, N. Y., before coming to Borger.

Listed in "Who's Who in American Women," Dr. Voet was a member and officer of the Borger Branch of the American Association of University Women, Altrusa Club, and Conamour Club. She was a member of the American Medical Association and the Texas Medical Association through the Gray-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society, of which she was a past officer.

Survivors are her husband; two daughters, Mrs. Louise Blumberg, wife of Dr. E. J. Blumberg of Arlington; Miss Marion Voet of San Francisco, Calif.; two sons, Donald Voet of Cambridge, Mass., and Martin Voet, of Berkeley, Calif.; two grandchildren; a brother, Martin Mogendorff of Arnhem, Holland; and a sister, Mrs. Anne Frank, of Ra'ananah, Israel.

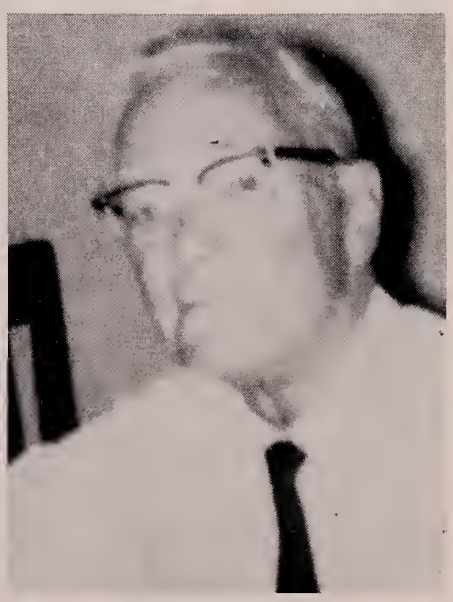
DR. H. H. VOET

DR. L. E. HAMILTON

Dr. Lawrence Edwin Hamilton of Rogers died at home from a coronary attack on Dec. 8. The 65-year-old physician had practiced in Rogers for 17 years.

A native of Dallas born Dec. 1, 1898, he was the son of Luther E. and Willie P. Hall Hamilton. He attended Powell Training School in Dallas, the University of Texas, and graduated from the University of Texas Medical Branch in 1924.

He interned at Texas Scottish Rite Hos-



DR. L. E. HAMILTON

pital for Crippled Children in Dallas and served a residency at Knickerbocker Hospital in New York City.

On July 23, 1945, he married Mary Elizabeth Miller of Beeville in a San Antonio ceremony.

Before coming to Rogers, Dr. Hamilton practiced in Dallas, Chicago, Lubbock, and San Antonio.

A veteran of World War I, he was a member of the Rogers Community Service Club, a Mason, and the Rogers city health officer. Since his childhood, he had been a member of the Westminster Presbyterian Church in Dallas, but he had attended the Methodist church during the years he lived in Rogers.

He belonged to the American Cancer Society, American Heart Association, American Medical Association, Texas Medical Association, and the Milam County Medical Society.

He is survived by his wife; a son and daughter of a former marriage, Lawrence E. Hamilton, Jr., of Dallas and Ann McGivney Hamilton of Galveston; and a brother, William Hamilton of Dallas.

DR. B. F. FRINK

Dr. Berton F. Frink, McAllen surgeon and ophthalmologist, died unexpectedly of a stroke on Nov. 18. He was 58.

A resident of McAllen since 1950, Dr. Frink was a graduate of Baylor University College of Medicine. He was born in DeWitt, Iowa, the son of George B. and Florence B. (Goff) Frink. He attended school in Tacoma, Wash., and was a student at Washington State College and Baylor University. After medical school he interned at Baylor University Hospital in Dallas and served his residency at the New York University Post Graduate Medical School in New York City and at the Veterans Administration Hospital in Houston. He was certified by the American Board of Ophthalmology in October, 1952.

He was the author of an article published in 1955 in the *American Journal of Ophthalmology.*

DR. B. F. FRINK

During World War II he served as a major in the U. S. Army.

He married Vernon C. LaChance of Puyallup, Wash., on Nov. 7, 1928, at Vancouver, Wash. She died in July of 1957. Dr. Frink and Mrs. Margaret Edens Carter were married June 10, 1960, in San Antonio.

Dr. Frink was an Episcopalian and a Mason.

Medical organizations to which he belonged included Hidalgo-Starr Counties Medical Society, Texas Medical Association, American Medical Association, Phi Alpha Sigma, Alpha Omega Alpha, Academy of International Medicine, Texas Society of Ophthalmology and Otolaryngology, American Academy of Ophthalmology and Otolaryngology, Pan American Ophthalmological Foundation, the Association of American Physicians and Surgeons, and he was a fellow of the American College of Surgeons.

His hobbies were photography and golf.

He is survived by his wife, Mrs. Margaret Edens Frink; his mother, now Mrs. Ira West of Puyallup, Wash.; a sister, Mrs. P. M. Cunningham, also of Puyallup; a brother, L. G. Frink of Medford, Ore.; one stepson, John Edens Carter of Galveston; one stepdaughter, Mrs. Ernest E. Rowe of Corrigan, Tex.; and two grandchildren, both of Corrigan.

Coming Meetings

American Medical Association, Annual Meeting, San Francisco, Calif., June 21-25, 1964. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Houston, Annual Session, April 23-26, 1964; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

FEBRUARY

American Academy of Allergy, San Francisco, Feb. 7-12, 1964. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American College of Radiology, Tucson, Ariz., Feb. 2-8, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American Society of Clinical Pathologists, New Orleans, Feb. 19-23, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

Texas Association of Obstetricians and Gynecologists, Corpus Christi, Feb. 29, 1964. Dr. H. W. Savage, 815 Fifth Ave., Fort Worth, Sec.

Texas Public Health Association, Austin, Feb. 23-26, 1964. J. N. Murphy. Jr., P.O. Box 5192, Austin 31, Sec.

Texas Urological Society, Austin, Feb. 8-9, 1964. Dr. B. C. Bates, 1108 Nueces, Austin, Sec.

First District Society, El Paso, Feb. 1, 1964. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Twelfth District Society, Temple, Feb. 8, 1964. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

MARCH

American College of Allergists, Bal Harbour, Fla., March 1-6, 1964. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

South Central Association of Blood Banks, Oklahoma City, March 27-28, 1964. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southwestern Society of Nuclear Medicine, Houston, March 6-8, 1964. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Texas Tuberculosis Association, Austin, March 19-21, 1964. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

United States-Mexico Border Public Health Association, Monterrey, Mexico, March 2-5, 1964. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

Ninth District Society, Huntsville, March 19, 1964. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Dallas Southern Clinical Society Conference, Dallas, March 15-18, 1964. Dr. W. O. Tschumy, 1616 Medical Arts Bldg., Dallas, Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 2-4, 1964. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

National and Regional

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, Atlantic City, April 11-17, 1964. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, Philadelphia, April 20-22, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Association of Plastic Surgeons, Chicago, May 13-16, 1964. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N.Y., Sec.

American Association for Thoracic Surgery, Montreal, Canada, April 27-29, 1964. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Chest Physicians, San Francisco, Calif., June 18-22, 1964. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Obstetricians and Gynecologists, Miami Beach, Fla., May 17-22, 1964. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, Atlantic City, April 6-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Hawaii, June 27-July 2, 1964. Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Sec.

American Gastroenterological Association, Dallas, April 24-25, 1964. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C.

American Gynecological Society, Hot Springs, Va., May 25-27, 1964. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Medical Women's Association, Roanoke, Va., May 31-June 2, 1964. Dr. Jessie Brodie, 1790 Broadway, New York 19, Exec. Sec.

American Neurological Association, Atlantic City, June 15-17, 1964. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Philadelphia, May 10-14, 1964. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Surgical Association, Hot Springs, Va., April 1-3, 1964. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., Chicago, April 3-4, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons, Vienna, Austria, May 11-16, 1964. Dr. W. F. James, 1615 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico. Dr. C. Kalil, 200 East Monterey Way, Phoenix, Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum, Hot Springs, Ark., April 5-7, 1964. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., October, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Surgical Congress, San Antonio, April 26-29, 1964. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, Corpus Christi, May 15-16, 1964. Dr. Sanford Glanz, 2425 Medical Center, Corpus Christi, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, Houston, April 23-26, 1964. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Public Health Physicians, Houston, April 23, 1964. Dr. W. S. Brumage, 2600 Westover Rd., Austin, Sec.

Texas Chapter, American Academy of Pediatrics, Houston, April 24, 1964. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, Houston, April 26, 1964. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Houston, April 25, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, Houston, April 26, 1964. Dr. John W. Chriss, 2436 Morgan, Corpus Christi, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, Houston, April 23, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, Houston, April 26, 1964. Dr. E. I. Bruce, Jr., 1014 Strand, Galveston, Sec.

Texas Ophthalmological Association, Houston, April 23-26, 1964. Dr. Harold Hunt, 150 Eighth St., S.E., Paris, Sec.

Texas Orthopaedic Association, Houston April 23-26, 1964. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, Houston, April 23-26, 1964. Dr. J. P. Moran, 3154 Reid Dr., Corpus Christi, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, Houston, April 23-26, 1964. Dr. D. Hoehn, Doctor's Bldg., Fort Worth, Sec.

Texas Proctologic Society, Houston, April 23, 1964. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Radiological Society. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, Houston, April 26, 1964. Dr. L. Duflot, 173 San Marino, Galveston, Sec.

Texas Society of Athletic Team Physicians, Houston, April 23, 1964. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, Houston, April 23, 1964. Dr. W. C. Sellman, Jr., 610 Medical Arts Bldg., Dallas, Sec.

Texas State Board of Examiners in Basic Sciences, April, 1964. Mrs. Maria P. Hathaway, 1012 State Office Bldg., Austin, Exec Sec.

Texas State Board of Medical Examiners. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Surgical Society, Waco, April 5-7, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Houston, April 23, 1964. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

District

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, Tex., Spring, 1964. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Thirteenth District Society. Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

International Medical Assembly of Southwest Texas, San Antonio. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 412 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals. Mr. R. L. Heath, Malone-Hogan Clinic, Big Spring, Sec.

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What Is Pediatric Surgery?

PEDIATRIC SURGERY is the surgical care of patients in the pediatric age group, and particularly involves the correction of congenital defects, many of which are life-threatening. Well-established in the major medical centers of western Europe for the past two generations, there has been tremendous growth of this specialty in the last 20 years in the United States and Canada.

The father of pediatric surgery in the United States is Dr. William E. Ladd of Boston. Most practicing pediatric surgeons in this country are first, second, or third generation "Ladd-trained". The 140 members of the surgical section of the American Academy of Pediatrics are the qualifying body for pediatric surgeons in the United States and Canada. In recent years the Board of Regents of the American College of Surgeons decided that the classification of surgery (pediatric) should be granted only when the applicant had obtained substantial training in that field, in addition to that which would qualify him in general surgery, and he must limit his practice to pediatric surgery.

Because of the rapid pace of advancement in all phases of medicine and surgery, including pediatric, it is no longer possible in a reasonable period of time, to acquire the necessary knowledge and experience to expertly practice pediatric surgery without formal training. Most training programs require general surgery board certification or eligibility before a two-year training experience can be started in pediatric surgery. This training consists of diagnosis, preoperative, operative, and postoperative management of infants, children, and adolescents with head and neck, thoracic, abdominal, and urologic problems. The pediatric surgical resident learns embryology and fetal development; the relationship of growth and development to surgical treatment; the importance of properly administered

pediatric anesthesia; and frequently participates in research programs directed toward improving infant and child surgical care. He must learn to think in the terms in which a child thinks and to deal sympathetically and calmly with a child's reaction to illness and trauma. He learns to be thoroughly familiar with the techniques especially developed for surgery of the very young.

The vexing problem of who should do what has historically involved all growing specialties. That a surgeon can resect lung cancer or an adult pulmonary lobe or segment for tuberculosis is no reason to assume he can, with equal facility, recognize and repair a tracheo-esophageal fistula or a congenital diaphragmatic hernia in a four-pound infant without additional training and experience. Nor can it be assumed that the excellent endoscopist of adult urinary bladders and resectionist of adult prostate glands can, with equal facility, perform retrograde pyelography and anastomose a six-pound infant's ureter or intestine, or repair an extrophied bladder, and expertly manage the total care of these patients postoperatively. Likewise, a pediatric surgeon who has had considerable training and experience with esophageal and anal atresias, congenital diaphragmatic hernias, and infant diagnostic endoscopies cannot necessarily expertly correct a transposition of the great vessels or perform transurethral prostatectomy without additional special training. These problems are being solved to the patient's benefit by the communities whose physicians ask: Who has the training and experience to do what? And: Who can do what well?

There is considerable likelihood that in the next decade every community in the United States with a population of over 250,000 will have trained pediatric surgeons. In Texas, this has already been realized in Dallas, Houston, and San Antonio.

It is hoped that the symposium in this month's Journal on some common and uncommon surgical problems of infancy and childhood will be of use to physicians engaged in caring for this age group.

I am very grateful to my colleagues Drs. Luke Able and Carl Haggard of Houston, and Drs. George Dorman and Theodore Votteler of Dallas for their advice and assistance in the preparation of this symposium.

—Irving A. Ratner, M.D.,
San Antonio.

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House of Delegates Faces Diverse Issues

The proposed creation of a nominations board for TMA may cause debate on the floor of the House of Delegates, and most certainly it will be subject to praise, criticism, and suggestions for alterations when it comes before the reference committee.

Other items sure to arouse interest are a proposal giving the Board of Councilors explicit authority to divide a multi-county society into one or more societies, and a proposal to increase the dues of the Association, which are much lower than the average for state medical societies. These are among the reports to be published in the 1964 "Handbook for Delegates."

Obtaining the best talent available by the fairest means possible is a problem which the Council on Constitution and By-Laws has studied intensively for the past three years. Last year, the House approved in principle the idea of a nominations board, but directed the Council to present a more specific recommendation in 1964.

The new proposal calls for a five-member board elected by the House. No member could hold any other elective position by the House or have served on the board before, and no two members could be from the same councilor district. To provide continuity one board member, elected by the House, would serve one additional year only; all others would be limited to one-year terms.

The board would have a full year to evaluate the records of available persons, and during this time it might draw upon the experience of any member and/or officer of

the Association for advice and counsel as to the abilities of potential candidates. After the board presented its proposed slate of candidates, other nominations could be made from the floor.

Another proposed revision of the By-Laws would give the Board of Councilors the explicit right to divide a multi-county society into one or more societies if (1) a majority of members from one or more of the involved counties made proper application for the division, or (2) the Board judged that such action would benefit the profession of medicine.

In a detailed report of the Association's financial affairs, the Board of Trustees informs TMA delegates that although the organization presently is operating in the black, the budget for 1964 is fully committed, and that in recent years expenditures have increased more rapidly in relation to gains in income. At the same time that the growing and energetic Association has constantly expanded its programs and services, which require financial support, it also has undergone a substantial loss of revenue from advertising in the *Journal*. The number of committees has increased by 20 since 1954. Then there were 32 committees and now there are 52 whose duties must be underwritten financially. After considering alternatives, the Board requests support of an increase in dues, not to exceed $10, effective in 1965. Of all state medical societies, the average amount charged for dues is $60, and three-fourths of the states have dues higher than $45.

Possibly these and other issues will be debated on the floor of the House of Delegates, and its sessions are open to all TMA members. At the first meeting of the House, reports are accepted and referred to appropriate reference committees, where all sides of an issue are explored, questioned, and debated. These meetings are open, and it is here that any member of TMA who is interested in the outcome of a particular issue has the opportunity to present his views.

Delegates desiring more background information before making up their minds on particular questions are welcome to sit in on reference committee debates.

For the average doctor who may wonder just how the Texas Medical Association operates and how, why, and by whom decisions are made, a visit to a reference committee meeting can be a stimulating way to learn the answers. Times and places of these meetings are listed in the Annual Session Program, page 264 of this *Journal*.

Accreditation Spurred by Dry Run Surveys

IN 1959, Texas had only 130 accredited hospitals out of the 389 which were eligible. Today, 169 are accredited by the Joint Commission on Hospital and Accreditation.

Although Texas has a long way to go toward meeting its ultimate goal, this 30 per cent increase of accredited hospitals is due in large part to the united efforts of the Texas Hospital Association, the Texas Medical Association, the Private Clinics and Hospitals Association and the Texas Medical Record Librarians.

An important part of this united effort has been the Dry Run Accreditation Survey program.

TMA's Committee on Hospital Accreditation, under the sponsorship of Dr. Ray L. Shepperd of Burnet, has been very active in the survey.

As pointed out recently by Mr. J. D. Hall, Jr., chairman, Council on Professional Services, Texas Hospital Association, the survey was organized in 1958 when both physicians and administrators from unaccredited hos-

To be surveyed, a hospital requests a dry run survey through the Texas Hospital Association and agrees to pay only such travel expenses as the survey team may incur. Teams are made up from lists of individuals furnished by the three participating associations.

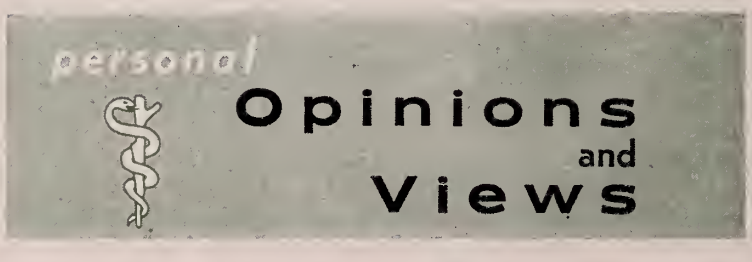
On the dry run surveys, Mr. Hall said, no official report is made and no opinions are given as to whether the hospital will receive accreditation by the Joint Commission, but the preview survey lets the medical staff, board of trustees, and administrators know what to expect from the Joint Commission. An invaluable part of the survey is the suggestions the surveyors make in conferences between team members, medical staff, and hospital personnel.

Since 1959, the program has received 63 requests for dry run surveys. Thirty-five hospitals have been surveyed, and of these

17 have become accredited. However, the sponsoring organizations feel that many who were not accredited the first time will be accredited on their next inspections by the Joint Commission.

The value of the program to individual hospitals, large and small, cannot be overemphasized. Of the 39 newly accredited hospitals, 17 were in the category of very small hospitals.

From the experience of hospital administrators, physicians, and medical record librarians who make up the dry run survey teams, personnel and medical staffs of unaccredited hospitals have the opportunity to gain practical knowledge and insight not only into the hospital's deficiencies, but they learn what to expect from Joint Commission surveys.



The Physician's Part in ADOPTION

ALL THE HELPING professions are interested in adoption. Some are concerned because they work with families who have an adopted child or wish to secure one, others because they directly help to place children for adoption. Some professional people —physicians, social workers, lawyers—have a unique part to play in establishing a happy family for a child who needs a home and new parents through adoption.

Every mother who gives her child for adoption, every child who is adopted, and every couple who adopts a child requires medical, legal, and social services. Any one service without the other exposes them to the tragedies and heartbreak growing out of hasty decisions to give or to adopt a child, placement of children in unhappy homes, unstable adoptions resulting from lack of preparation of all persons involved, or the removal of children from families that have loved and cared for them.

Before judicial action and decree in adoption can occur, a decision must be made as to whether or not permanent separation meets the best interests of the natural parents and the child. While termination of parental rights is a matter requiring court action, medical, legal, and social services should be available in reaching such a decision. Once this decision is made, the focus is on finding a family for a child rather than a child for a family.

In Texas, the State Department of Public Welfare has responsibility for licensing, standard setting, and partial supervision of adoption and foster care services by public and voluntary agencies. Outside of such licensed agencies, it is unlawful for any person (except a natural parent), association, or corporation, whether operating for profit or without profit, to place any child under age 16 for adoption. This is very simple and easy to understand.

There are those who believe that this law is not entirely proper. These people have every right to try to change the law by legislative action, but they should not expect the court to join them in an evasion of the present statute.

The prohibition in the statute does not carry any specific criminal penalty, but it is enforceable nevertheless by injunction proceedings. A person may, upon proper application and after establishment of the fact that the law is being violated, be enjoined by the court from continuing such activities. Upon violation of the injunction the person may be punished for contempt of court.

Several lawyers and some physicians have contended that in helping to place a child (of an unmarried mother, for instance) they are

acting with the authority and under the instructions of the natural mother, and that this is a placement by the natural mother and not by the physician or the attorney involved. Nevertheless, if the physician or the lawyer are acting as agents of the mother in making such a placement, they are acting as a child placement agency. Of course, if the mother herself signs and executes a particular acknowledgment, sworn to and acknowledged by her, placing this child for adoption by a particular named couple, and she knows their names, this is a direct and legal placement by the mother. However, most physicians, lawyers, and social workers agree that the natural mother should not know the persons who adopt her child, simply because it leads to confusion in the future and possible hurt to the child and to the adopting parents, and does not furnish the necessary protection in adoption to all parties.

Since there is no specific penalty of a criminal nature provided for the violation of this placement prohibition, some contend that it is only a matter of conscience on the part of the physician or lawyer as to whether or not he will take part in such a placement. There are two results that can come about from such an attitude or action:

1. The judge of a court may not be expected to join in an evasion of a clear prohibition in the statute of the state.

2. The participant may face charges involving professional misconduct. In the November, 1963, issue of *The Family Lawyer*, a publication of the Section of Family Law of the American Bar Association, the following ruling was reported:

"The Supreme Court, Appellate Division, First Department of New York, 1963, upheld the suspension of an attorney for a period of six months because of the attorney's attempt to place a child in violation of the Social Welfare Law, Article 374, Subdivision 2. The Court unanimously held that the offense did not involve moral turpitude, but constituted professional misconduct warranting suspension for six months. In Re: *Aronson, 238 NYS 2d Page 333.*"

Thus the medical and legal professions, being under self-governing rules and laws in regard to membership and the right to practice, are governed by their own codes of ethics which are enforceable in one way or another under Texas law. Evasions are decidedly risky.

Physicians have both direct and indirect responsibilities in adoptions. They are *directly* responsible for medical service to the natural mother, the child, and the prospective adoptive couple, and for referring them to the appropriate social agency. Social agencies are better equipped and in most states have the legal responsibility for placing children in adoptive homes, but these agencies depend on physicians for medical judgment.

For the natural mother, whose first confidant is often the physician, he provides continuing medical care; he also refers her to a social agency for help with emotional, social, financial, or environmental problems.

For the child whose good start in life depends on the physician, he provides or arranges for periodic diagnostic appraisal and continuous medical supervision. The physician's appraisal of the child is one of the key services in the adoption process, and adequate appraisal of the child before and after adoptive placement is essential.

To the couple who seeks advice and help in adopting a child, the physician provides medical service, determines whether they have reasonably good health and life expectancy, and sees that they have an adequate fertility assessment. He refers them to a legally authorized adoption agency, where social workers skilled in interviewing techniques can help the couple to determine whether or not they *want* or *should adopt* a child. If so, the agency can assist them in making appropriate plans.

Certain other responsibilities of the physician, though indirect, are equally important. They grow out of the physician's relationship with other professions, agencies, or individuals, and out of his duties as a citizen.

As a consultant to a social agency, the physician interprets the physical condition of the child and makes recommendations as to adoptability on the basis of the child's background, or, for the handicapped, his special needs, and the prognosis for healthy growth and development. He also is consulted as to the medical suitability of the adoptive couple. The physician develops criteria on adoptability from a medical standpoint for use by the agency, and he also contributes to the agency's staff development program.

As a hospital staff member, the physician can promote medical staff policies and education of personnel to insure adequate medical and social care for the unmarried mother and her child. Good medical care also in-

cludes maintenance of accurate medical records and proper registration of births.

Sound hospital policies will prevent staff members' participation in the independent placement of a child. Doctors and administrators should work toward policies that not only meet, but go beyond, legal requirements for maximum protection.

As an educator, the physician helps medical and health personnel realize the great impact of adoption on our society, its magnitude and importance, and its problems and hazards. Other professional groups, such as students and faculties in schools of social work, nursing, and law, are within the realm of such counsel. Committees or discussion groups at medical society meetings offer opportunities to expand general knowledge of adoption laws and procedures, as does the inclusion of this material in the curriculum of medical schools, especially in such specialties as pediatrics, obstetrics, psychiatry, and public health.

The physician can build up a file of current literature for his own use and for others, including material on agency resources, policies, procedures, and state laws. The State Departments of Health and Welfare are good sources for this information.

As a citizen, the physician takes part in community planning for adoption services. He may work with people in newspapers, magazines, television, and radio to help educate the public. By serving on citizens' adoption committees, physicians can help to make known the needs of children and of natural and adoptive parents. Also, physicians can seek sound legislative measures on adoption.

The medical competence and judgment of the physician are fundamental to a sound adoption program, but his contribution is of greatest value when it is given, and assessed, in conjunction with others. When all helping professions collaborate, adoption can realize its promise of happy family living for a child who must have new parents.

—CHARLES O. BETTS, Judge,
98th District Court of
Travis County, Austin.

In Bed and "In" School

An apparatus similar to an intercom is enabling a young victim of rheumatoid arthritis and a heart ailment to keep up with her class at school. A 12-year-old who attends San Marcos Baptist Academy in San Marcos, Tex., she follows doctor's orders for extensive rest, yet from her campus bedroom, she hears everything going on in her sixth grade classroom. By pressing a button at the top of the little brown box by her bed, she also asks questions and participates in class discussions.

The local telephone company arranged the two-way communication system which may prove the answer to study problems of the school's infirmary patients.

Medical Assistants

The Texas Medical Assistants Association was organized in 1957 and is a charter member of the American Association of Medical Assistants. The purpose of this fine organization is to educate its members to improve public relations in medical endeavors and to render more efficient services to the doctors whom they serve.

Article II, Section 1, of their constitution states: "It is not nor shall it ever become a trade union or collective bargaining agency. Any member or members attempting to organize as such hereby automatically forfeit their membership."

Receptionists, secretaries, bookkeepers, nurses, and technicians employed by members of the Texas Medical Association are eligible for membership. At present there are 20 local chapters in Texas, each organized in the geographical area of one of our component county medical societies, and new chapters are in the process of formation.

As token of our esteem for the TMAA, the Board of Trustees has appropriated a grant to be used in furthering the educational seminars which are presented to their members in order that they may better serve the physicians of Texas.

The loyalty of this paramedical group is unquestioned; and, more than financial aid, they need the encouragement, cooperation, and interest of every member of the Texas Medical Association.

Robert Mayo Tenery

President
Texas Medical Association

Neuroblastoma and Wilms's Tumor In Infants and Children

THE TWO COMMONEST solid malignant tumors in the pediatric age group are rarely seen in adults. Neuroblastoma and Wilms's tumor have many characteristics which set them apart from other malignant tumors.

Neuroblastoma

Neuroblastoma is a malignant tumor of cells derived from nonchromaffin sympathogonia. These cells are ultimately derived from the embryonic neural crest.

Although the tumor was probably described earlier by Virchow in 1864,[4] Dalton in 1885, and Pepper[10] and Hutchinson[7] in 1901 and 1907, the accepted concept of histogenesis was presented by Wright[4] in 1910. He demonstrated the typical rosette pattern of neuroblastoma cells and compared it with a practically identical arrangement of primitive sympathetic nerve cells from a fetus.

Age Incidence.—The tumor is twice as common in children under age five than in the age group from five to fifteen years. It is very rare in adults.

Pathology.—The tumor most frequently arises in the adrenal medulla. It may be multicentric in origin anywhere neural crest derivatives exist. This tumor may arise along the sympathetic nerve trunks in the abdomen, pelvis, chest, or neck.

The tumor may be smooth or irregular. It is usually yellow or brownish-yellow.

Neuroblastomas vary greatly in their rate of growth and are frequently not predictable. Some may metastasize widely and produce death in a matter of weeks after discovery, whereas others may remain local or may rarely even revert to histologically benign ganglioneuromas.[3] These tumors can again revert to lethal neuroblastomas years later.

It is this lack of predictability that requires caution in deciding on the efficacy of any given form of therapy.

Diagnosis.—The diagnosis is usually made by palpation of a large, intra-abdominal mass which commonly projects deeply into the flank, extends across the midline, and is usually not movable. On intravenous urogram there is a displacement of the kidney downward without intrinsic distortion of the renal pelvis and calyces. Approximately 15 per cent of these tumors show calcification on abdominal roentgenograms.

Retroperitoneal neuroblastoma originating in an adrenal gland frequently will grow by direct extension into the liver and pancreas and metastasize early to both the bone marrow and cortical bone. Nodules on the skull or proptosis from retro-orbital neuroblastoma are not unusual presenting findings.

Pain, anorexia, and weight loss are usually late findings. Metastases to the lungs are not common, even late in the disease.

Recent reports indicate that an occasional neuroblastoma may produce varying amounts of catecholamines which cause symptoms similar to pheochromocytoma.[5, 12]

Greenberg and Gardner[5] have found increased amounts of norepinephrine, 3 methoxy-4-hydroxymandelic acid, dopamine, and homovanillic acid in the urine of some patients with tumors of neural crest origin.

The data indicate that determination of these substances in the urine may aid in the diagnosis and follow-up of neuroblastoma patients.

Treatment.—Physicians with the most experience managing this tumor recommend surgical removal of as much tumor as pos-

A review is presented of the current concepts of diagnosis, treatment, and prognosis of the two most common solid malignant tumors which occur in infants and children.

IRVING A. RATNER, M.D.

sible, followed by x-irradiation (2000-3000 roentgens).[1, 4, 9, 11, 15]

If possible, total excision of the neuroblastoma is desirable. Unfortunately, this is frequently impossible. In such instances, Koop[9] reported that extensive but incomplete removal of a massive tumor is superior to biopsy alone.

When roentgen-ray therapy is added to extensive but incomplete tumor excision, the results are thought to be better.

Recently, there has been considerable interest in a number of oncolytic drugs as adjuvant therapy of neuroblastomas. Nitrogen mustard, Cytoxan, actinomycin D, and Methotrexate have been shown to be of some value in palliation.

Bodian's[2] success with high doses of vitamin B_{12} for infants under one year has not been duplicated in the United States.

Prognosis.—The overall outlook for five-year survival is slightly more than 10 per cent. There have been several long-term survivors who have had incomplete removal of metastatic neuroblastoma of the liver.

Dargeon[4] reported a 13-year survivor with cervical node metastases from a primary mediastinal neuroblastoma.

The importance of the lack of predictability of this tumor is underscored when one tries to apply the overall mortality statistics to any particular child with neuroblastoma. This cannot accurately be done.

Thus far, aggressive surgical and roentgen-ray therapy offer the individual child with neuroblastoma his best chance of survival, even in the presence of obvious metastases.

Wilms's Tumor

This embryonal, malignant renal tumor has been known by many other names, for example, embryoma, nephroblastoma, adenomyosarcoma, myosarcoma, and Birch-Hirschfeld tumor. It was described by a number of others prior to Wilms's[14] classic paper in 1899. The tumor is regarded by many to be congenital.

Incidence.—The exact incidence of this tumor is not known. It is probably about as common as neuroblastoma in the pediatric age group. Gross regards this tumor to be the most common solid tumor in infancy and childhood. In Dargeon's[4] experience, neuroblastoma is the more common.

Age and Sex.—Wilms's tumors occur most commonly between ages three months and five years, although occasionally one may occur in a preadolescent, adolescent, or even an adult. Males and females are about equally represented.

Pathology.—The tumor is believed to arise from the mesenchymal tissue of the renal blastema. The tumor may originate at various times in the development of the renal anlage. This would account for the various types of tissues found in Wilms's tumors periodically (cartilage, muscle, bone, primitive-appearing glomeruli, renal tubules, and fat).

Wilms's tumor is usually unilateral (90 per cent) and may involve any portion of either kidney.

The tumor metastasizes early to local renal and preaortic lymph nodes, and later, through the renal vein to the lungs. Osseous and peritoneal metastases are usually late.

Diagnosis.—The patient usually presents with a painless mass in the abdomen. The mother may find this accidentally while bathing the child. The abdominal mass may be the size of a softball or larger when first seen by a physician. The mass is usually circumscribed and movable, and rarely crosses

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the midline of the abdomen. The child is otherwise asymptomatic.

On intravenous urogram, typical distortion of the renal pelvis and calyces by an intrarenal tumor can be seen.

Pulmonary metastases may be noted on the chest roentgenogram at the first visit to the physician.

Urinary symptoms of frequency, urgency, dysuria, and oliguria are very rare. Hematuria is uncommon and usually a poor prognostic sign because it suggests spread of the tumor into calyces or the renal pelvis. Hypertension has rarely been noted in my experience.

When hematuria exists, cystoscopy should be performed prior to surgical removal of the abdominal mass. Occasionally the implants of Wilms's tumor can be found on the bladder wall.

Treatment.—The currently recommended treatment for Wilms's tumor is urgent but not emergent surgical excision, followed by roentgen-ray therapy of 2000-3500 r. to the tumor bed.[1, 4, 6, 8, 13]

Frequent or rough abdominal palpation prior to surgery is discouraged because of the ease of tumor spread through the renal vein.[13]

Time should be taken to restore body fluids to normal and to assess the child as a whole prior to surgery. This can usually be accomplished in 24 hours.

The surgical excision is best performed transperitoneally through a generous incision. Whether the incision be transverse, vertical rectus splitting, mid-line, thoracoabdominal, or L shaped is a matter largely up to the preference of the surgeon and the location of the bulk of the tumor. The usual retroperitoneal flank approach to the kidney is to be avoided because of the excessive amount of tumor manipulation required for excision and the difficulty in performing adequate removal of the nodes. As soon as the diagnosis is apparent, the renal vein and artery are exposed and ligated in the hope of preventing blood-borne metastases during later tumor manipulation.

It is undesirable to attempt tumor aspiration in an effort to ease the surgical removal. There have been few if any survivors of Wilms's tumor which had broken through the renal capsule.

It is the author's practice to perform en bloc resection of the preaortic lymph nodes from the aortic bifurcation at the pelvic brim to the diaphragm, and of the entire ureter, kidney, and adrenal gland on the affected side. Careful inspection and palpation of the contralateral kidney is made for a second Wilms's tumor. If one is found in either upper or lower pole of the contralateral kidney, that pole is locally resected.

If on prior cystoscopy, performed because of hematuria, implants are seen on one side of the bladder (usually the side of the renal tumor), hemicystectomy will be added to the operation.

The tumor bed is outlined with silver clips which later guide the radiotherapist.

Upon return from the operating room, the first roentgen-ray treatment is given and is continued daily until completion of a 2500-3500 r. tumor dose.

In the past three years there has been considerable interest in certain oncolytic drugs, particularly actinomycin D. This antibiotic is oncolytic in its own right and radiomimetic in addition (that is, it seems to aid roentgen-ray destruction of certain tumors).

In several centers, actinomycin D therapy is routinely given to all Wilms's tumor patients at regular intervals (three to six weeks) after surgical excision, regardless of the presence of metastases. Whether this form of therapy will prove to be the one of choice is at present not known.

The side effects of actinomycin D are leukopenia and alopecia, both of which are reversible upon cessation of the drug. The usual total dosage is 75 gamma per kg. body weight. This is divided into four to six daily doses and administered intravenously.

Preoperative x-irradiation to facilitate later surgical removal or to reduce the possibility of metastases at the time of surgery has not shown as good results as surgery followed by roentgen-ray. It has the added disadvantages of irradiating a possibly nonmalignant renal lesion, softening a Wilms's tumor so that it is more easily ruptured dur-

ing operative manipulation, and distorting the gross limits of the tumor so that the surgeon has a less clear idea of the proper line of resection.

Subsequent solitary pulmonary metastases have been successfully resected locally or treated with roentgen-ray therapy with and without actinomycin D.

Prognosis.—The five-year survival reports have shown considerable variation from 2 per cent to 50 per cent. There is general agreement that the younger the child the better the prognosis. In infants under one year, five-year survivals of 80 per cent have been reported.

The average overall five-year survival is closer to 20 per cent.

Collins has reported that the period of risk for any given child with Wilms's tumor is his age at the time of diagnosis plus nine months. If such a child is treated and then lives beyond this period of time without evidence of recurrence, he is presumably beyond all risk of recurrence. That this period-of-risk principle may be applied in the face of routine, periodic administration of actinomycin D or other oncolytic drugs is doubtful.

Summary

A brief account is presented of the current concepts of the pathology, diagnosis, treatment, and prognosis of the two most common solid malignancies in infants and children, neuroblastoma and Wilms's tumor.

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Smoking and Infant Orality

No relation was found in an investigation of the smoking habits of schoolchildren in Newton, Mass., between breast or bottle feeding and subsequent smoking.

A random stratified sample of senior students and their mothers were interviewed and data on 314 pairs of mothers and students were reported in an article by Dr. Eva J. Salber of Boston in the Feb. 1 issue of *Journal of the American Medical Association.*

No differences were found between thumbsucking, time of weaning, intervals between feedings, and later smoking habits.

It was suggested by Sigmund Freud in his "Three Essays on Sexuality" that such a relationship does exist.

Gastrointestinal Bleeding In Infants and Children

LUKE W. ABLE, M.D.

SYMPOSIUM

BLEEDING ENTITIES, and some nonoperative entities considered as possible surgical lesions, treated in pediatric surgery are discussed herein. Historically, bleeding has been classified by its color and the presence of associated pain (Tables 1-3). The etiology usually is obvious, or clarifies soon with a few selected studies. Occasionally it is impossible to say what caused a bleeding episode.

A history indicates the probable factors precipitating such an episode, the patient's ability to handle previous bleeding, and the associated symptoms. A physical examination reveals pallor, bruising, oral or anal bleeding, the color, character, and amount of bleeding, associated pain, tenderness or obstruction, cerebrovascular status, and urgency of treatment. Petechiae may appear when a tourniquet or blood pressure cuff is applied. The patient's clotting ability or bleeding tendency is noted whenever a finger stick or needle puncture is done. Blood smears are checked for platelets and platelet counts are done as indicated. Prothrombin studies are done on patients with possible liver disease, prolonged bleeding, delayed clotting, or massive bleeding. Massive bleeders are typed and crossmatched, and at least 20 to 50 cc. of blood per lb. of body weight is kept in available reserve until the danger of exsanguination has passed. Oral endoscopy may indicate some of the causes of hematemesis or melena, but when anal bleeding is bright red, proctosigmoidoscopy is done to verify a low lesion or to verify that bleeding is above this area.

Hematemesis and melena occurring on the first day of life can result from maternal blood swallowed in utero, in which case the maternal history may indicate uterine bleeding. These infants will maintain a normal newborn hemoglobin and hematocrit even though massive melena and hematemesis continues. Sodium hydroxide added to a solution of this tarry stool changes this solution's pink color to yellow-brown, indicating adult (maternal) hemoglobin. However, when the tarry stool contains the newborn's hemoglobin (80 per cent fetal and resistant), this pink color is hardly changed. Nurslings have a similar melena when their mother's nipples bleed during breast feeding.

Hemorrhagic disease of the newborn is now recognized as hypoprothrombinemia. Bleeding occurs at the cord, the circumcision, the tongue-clipping, and into the tissues, including the gastrointestinal tract. It became a rare cause of melena neonatorum after vitamin K became available. In later life hypoprothrombinemia and gastrointestinal bleeding occur in association with liver disease, intestinal obstruction, and use of many drugs, especially antibiotics and aspirin.

Unchanged bright blood mixed with stool and mucus has occurred in infants who have recently changed from breast or a partially digested milk to an unrefined cows' milk. Their associated colic and spitting-up is suggestive of intussusception, but no obstruction or mass develops. Their abdomen is nontender and peristaltic sounds are active. The blood loss seldom affects the hemoglobin or hematocrit.

Mucosal ulcerations may occur with the stress of birth, surgery, or anesthesia, as well as from anoxia. Bloody, or coffee-ground, material is obtained when the stomach is aspirated or if the patient vomits. Occasionally in the newborn, a mucosal ulceration continues to bleed (and even perforate) at 30 to 40 hours. Such infants are noticeably pale, and hematocrit is correspondingly decreased. Roentgenograms seldom demonstrate an acceptable ulcer. Transfusions are given as needed. Gastric hypothermia by irrigation with iced Ringer's solution reduces the mucosal blood flow as well as the acidity and enzyme activity. When energetically applied, this therapy has

Gastrointestinal lesions causing bleeding are classified and discussed, as are the various diagnostic tests appropriate in different types of bleeding.

been adequate before perforation.

Peptic ulcerations occur in many patients with hypertrophic pyloric stenosis. These infants have a higher gastric acidity than normal infants of similar age. Violent vomiting may also rip the mucosa, especially at the cardia.

Classical bleeding peptic ulcers are rare in the young. They produce painless bleeding. Mild postprandial pain is either unappreciated or ignored. Mothers agree later that the patient has been irritable for a few days before he was explored for silent but massive melena. Local resection pyloroplasty and vagotomy has been adequate in the pediatric age even when the free acid has been high. So far, neither the non-insulin-secreting pancreatic tumors nor the parathyroid tumors have been recognized as a cause of gastric hypersecretion and peptic ulcers in young children.

The common peptic lesions in children are ulcers that are marginal to diverticuli, duplications, or ectopic pancreatic tissue. Antimesenteric diverticuli (commonly Meckel's) are excised and the associated marginal ulcer heals; similar healing occurs in the marginal ulcer associated with isolated ectopic pancreatic tissue that may occur in the esophagus, stomach, intestines, or colon. Bleeding duplications usually contain ectopic gastric and pancreatic tissue. All duplications have a common muscular wall with some portion of the esophagus or gastrointestinal tract. Esophageal duplications are commonly asso-

TABLE 1.—*Dark Blood.*

1. Swallow Blood
   - Maternal
     - Bleeding in utero
     - Bleeding nipples
   - Oral bleeding
   - Respiratory bleeding
2. Mucosal Ulceration
   - Stress
     - Anoxia
     - Trauma
       - Head injury
       - Burns
   - Endocrine lesion
     - Adrenal
       - Pheochromocytoma
       - Corticoma
         - Cushing tumor
         - Aldosteroma
     - Parathyroidoma
     - Pancreatic
     - Hypothalamic-pituitary
   - Peptic ulcers
     - Esophagitis
     - Gastroduodenal
     - Marginal
   - Obstructive
     - Esophageal
     - Pyloric
     - Intestinal
   - Drugs
     - Aspirin
     - Antibiotics
     - Steroids
     - Nitrogen mustard
     - Numerous poisons
3. Varices
   - Gastric
   - Esophageal
   - Intestinal

TABLE 2.—*Dark and/or Bright Blood.*

1. Hepatobiliary Disease
   - Hematobilia
     - Hepatitis
     - Post-traumatic
     - Cholelithiasis
   - Hypoprothrombinemia
2. Aberrant Gastric and Pancreatic Tissue
   - Duplications
   - Meckel's diverticulum
3. Volvulus
   - Mesenteric thrombosis
4. Intussusception
   - Acute
   - Chronic
   - Postoperative
   - Rectal
5. Systemic Disease
   - Allergy
     - Milk
   - Schönlein-Henoch purpura
   - Glomerulonephritis
   - Salt-losing nephritis
   - Uremia
   - Purpuras
6. Trauma
   - External
     - Blunt
     - Penetrating
   - Internal
     - Gastrointestinal suction
     - Swallowed foreign body
     - Suppositories
     - Foreign body in anus
7. Tumors
   - Hemangioma
   - Telangiectosa

TABLE 3.—*Bright Blood.*

1. Tumors
   - Colonic polyps
     - Inflammatory
     - Adenomatous
     - Familial
   - Intestinal polyps
     - Peutz-Jeghers syndrome (melanin spots)
   - Carcinoma
   - Lymphosarcoma
   - Neuroblastoma
   - Teratoma
     - Sacrococcygeal
     - Ovarian
     - Gastric
   - Rhabdomyosarcoma
2. Enterocolitis
   - Infectious
     - Specific
     - Pseudomembranous
     - Granulomatous
   - Ulcerative
     - Idiopathic
     - Amoebic
3. Anorectal
   - Proctitis
   - Fissures
   - Hemorrhoids
   - Fistula
   - Impactions
   - Stercoral ulcers

ciated with hemivertebrae. Bleeding duplications of the esophagus, stomach, and colon can be excised. Small intestinal duplications lie in the mesentery and in the common blood supply to the adjacent small intestine. When resection of the duplication and its adjacent intestine would be too extensive for nutritional survival, the mucosa of the diverticulum may be stripped out.

Hemorrhage from Peyer's patches was a common complication of typhoid fever. Massive bleeding has occurred following typhoid immunization. Other lympholytic stresses, such as severe illness, nitrogen mustard therapy, and intoxication have caused active bleeding into the ileum at lymphoid patches.

Some infants have reflux regurgitation almost from birth; they may gain slowly and be "gurgly sleepers." When reflux persists peptic esophagitis develops; bleeding may be occult or massive. This may be prevented by early pyloroplasty to improve gastric drainage. Another group, the "slow feeders," have a stenosis either intrinsic (diaphragm or scar) or extrinsic (vascular or tumor). These infants develop a proximal stagnant esophagitis. Likewise, this obstruction should be relieved early. All forms of continued esophagitis cause spasm and esophageal shortening, which leads to hiatus hernia and more reflux-chemical esophagitis causing occult or gross bleeding. A destroyed esophagus requires a colon transplant.

Gastroesophageal varices associated with portal hypertension ultimately produce massive melena and hematemesis. These children usually do not bleed to death from their original hemorrhage, but continued bleeding has been fatal. Infections often precipitate the bleeding episodes. A previous history of omphalitis, umbilical vein transfusions, liver disease, ascites, or recurring anemia has been present in 50 per cent of the patients. Although the spleen contracts during hemorrhage, 100 per cent eventually show splenomegaly. Blood and x-ray studies help to rule out other bleeding lesions. Splenic puncture allows portal pressure measurements and cine-venograms. Cine-venograms outline the portal drainage showing dilatation, obstruction, and the collateral outflow. Preoperative esophagoscopy further indicates the extent of varices. Enthusiastic variceal suture or shunt treatment is indicated at any age except in the rare patient with rapidly progressive liver disease.

A few urgent strangulating lesions originally present as melena or hematemesis. The fretfulness and pain preceding the strangulation may be ignored. Strangulating volvulus usually is associated with a midgut malrotation, however, it may occur with a normal rotation, and about a congenital or postoperative adhesion. Early obstruction is indicated by gastric aspiration of bile and plain roentgenograms; contrast enema studies give diagnosis of malrotation, intussusception, or polyp, and indicate the degree of obstruction. Intussusception occasionally is associated with colic, but presents as painless bleeding; such silent intussusception has occurred in premature and young infants, especially chronically fretful patients. Tenderness indicates impending gangrene. Both strangulating mesenteric and inguinal hernias may cause melena. Diaphragmatic hernia may also strangulate and bleed even before dyspnea becomes apparent. A bleeding that occurs during an early postoperative period requires frequent physical examinations as well as laboratory aids to evaluate possible strangulation. Mesenteric thrombosis can start with any of the above lesions, omphalitis, portal thrombosis, or an arteritis.

Bleeding from polyps of colon is usually painless and bright red unless the polyp is

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the lead-point for an intussusception; massive polyps may produce obstruction in an infant. Polyps within finger reach can be prolapsed out the anus, the bases ligated, and the polyps excised. The majority of polyps are removed from below with present endoscopy aids; even some in the descending colon. When endoscopy is negative and the colon is clean, contrast enema studies may indicate the position of the polyp. The entire colonic mucosa is visualized through two or more operative colotomies. Simple infectious or juvenile polyps, "intestinal warts," are not premalignant lesions; they seldom cause serious bleeding and often subside spontaneously; they are excised for diagnosis and cured by coagulation.[1] Adenomatous, especially sessile, polyps can be premalignant and are widely excised. Diffuse scattered polyps and multiple sessile lesions are treated by segmental resections. Multiple familial polyposis is safely treated by colectomy before carcinoma develops. Small intestinal polyps are usually benign, and are suspected in any patient with pigment spot on the oral mucosa.

A gastrointestinal carcinoma is rare even in the colon. Lymphosarcoma of the ileum is less rare; both of these neoplasms can cause bleeding from the rectum before a mass is palpable, and at a time when complete excision is possible. Examination and studies at three to six weeks may provide better diagnosis than studies immediately after hemorrhage. Hemangioma and telangiectasia are rare benign tumors. Ovarian and retroperitoneal teratoma, neuroblastoma, and rhabdomyosarcomas eventually involve the bowel and cause bleeding. Occasionally, an infarcted cyst or tumor, or an abscess adjacent to the bowel produces hyperemia with bleeding. Adequate palpation is essential even if the frightened child has to be returned or sedated. First menstrual bleeding is sometimes confused with rectal bleeding, especially when this occurs during a postoperative period.

The unligated artery in an inverted appendiceal stump can be a brisk bleeder even 7 to 14 days after appendectomy. Minor mucosal bleeding follows many intestinal operations, intubations, swallowed foreign bodies, and radiation.

Anorectal bleeding is most often from a fissure. When constipation is the cause, treatment is obvious; but fissures also occur with diarrhea. Papillitis, proctitis, perianal abscess, and fistula-in-ano may bleed. The presence of local anal lesions does not exclude a proximal bleeding lesion. Endoscopy can determine if this blood is coming from above. When anal lesions persist in spite of standard therapy, an associated allergy, a specific bacterial or granulomatous (regional) enteritis, or a protozoal or ulcerative enterocolitis may be present and bleeding; fissures and fistulae will persist until the primary lesion is relieved. An ileostomy can be a conservative procedure even for infants.

Summary

Gastrointestinal lesions causing bleeding are classified and discussed. The diagnosis of a gastrointestinal bleeding lesion can be easy, but on rare occasions requires extensive studies and continued observation. Hematemesis indicates swallowed blood, or an upper tract lesion. Selective studies are gastric analysis, radiography, esophagogastroscopy, and liver and splenic studies including biopsy, manometry, and venography. Melena may result from an upper lesion immediately identified by gastrointestinal intubation, or from an in-between midgut lesion. Bright red blood per rectum may indicate a colonic lesion identifiable by anal endoscopy, or a higher lesion associated with a rapid peristalsis.

The possibility of an impending gangrenous lesion is evaluated along with the blood loss. Bleeding early in the course of a malignancy or other serious lesion justifies persistent examinations, studies, and follow-up to establish a positive diagnosis and appropriate treatment.

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The Undescended Testicle In Infants and Children

THE UNDESCENDED testicle is a common surgical lesion of pediatric practice, and misconception and controversy surround its management. The undescended testicle is caused by an arrest of testicular descent at the fifth to sixth month of development. It is a congenital anomaly, and surgical therapy is the primary mode of treatment.

Embryology

A discussion of the undescended testicle rests upon the understanding of testicular descent into the scrotum. The testicle forms in the retroperitoneal tissue near the kidney and descends through the inguinal canal in the fifth to sixth month of development.[2] An outpouching of peritoneum, the vaginal process, precedes testicular descent through the canal. The pathway that the vaginal process and testicle take through the canal is directed by the gubernaculum. When the testicle reaches the base of the scrotum, the vaginal process is obliterated from the testicle level to the internal ring. This simplified explanation is sufficient to allow understanding of the anatomical findings in this discussion.

Arrest of the descent of the testicle from the retroperitoneal region to the upper scrotum results in the clinical findings of the undescended testicle. Persistence of the vaginal process is to be expected, and a hernia will be found in 92 per cent of true undescended testicles upon exploration of the canal. Failure to obliterate the vaginal process after complete testicular descent results in the indirect inguinal hernia of childhood. Incomplete obliteration of the vaginal process with a narrow opening persisting at the internal ring results in either a hydrocele of the cord, or a hydrocele of the tunica vaginalis, determined by the position of the persistent peritoneal sac.

Diagnosis

Problems of the diagnosis of undescended testicle are responsible for much confusion surrounding this subject. Many of the patients referred for evaluation of an undescended testicle have never had the testicle felt by the physician. Inspection reveals an empty scrotum, and a search for the testicle has failed to reveal evidence of the testicle at the internal ring or in the canal. The diagnosis of the position of the testicle is facilitated by placing soap and water on the inguinal area to decrease skin friction. The examiner's fingers are swept across the inguinal canal starting at the anterior iliac spine progressing to the scrotum. The testicle should be felt in the inguinal canal. If the testicle can be brought into the base of the scrotum, the testicle is retracted due to overactive cremasteric muscle action and it is not a true undescended testicle. If the testicle can be found in the canal and cannot be manipulated into the scrotum, it is a true undescended testicle. This examination maneuver will allow palpation of the testicle in 95 per cent of patients. The testicle cannot be grasped to completely evaluate its size, but it can be definitely demonstrated both to the physician and to the parents, and an opinion can be obtained as to the difficulty to be anticipated in scrotal placement. If the testicle cannot be demonstrated by this means, the testicle may lie within the retroperitoneal structures, may be atrophic, ectopic in position, or lie within the inguinal canal obscured by the overlying excess fat. Exploration is warranted

Management is described with emphasis on diagnosis, the proper age for surgery, and surgical technique.

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if the testicle cannot be felt by the previously described maneuver to definitely establish the presence or absence of the testicle.

Retracted Testicle

Much of the controversy concerning the therapy of the undescended testicle is caused by the condition known as retracted testicle. As described previously, this is a testicle in the canal because of an overactive cremasteric muscle action. The testicle can be manipulated into the base of the scrotum although it will subsequently retract back into the canal. An inguinal hernia cannot be demonstrated in association with a true retracted testicle. When the child goes through puberty with testicular enlargement and increase in weight, the cremasteric muscle effect is overcome and the testicle descends into the scrotum. Advice to observe a patient with a nonscrotal testicle until puberty for the possibility of spontaneous descent rests upon the diagnosis of a true retracted testicle. Gonadotrophic hormone will increase testicular weight and allow descent of a retracted testicle. Hormone therapy is not desirable or necessary.

Age of Election For Orchiopexy

Three viewpoints are commonly expressed by physicians caring for patients requiring orchiopexy. The age of election for surgery is either after puberty if descent is not noted during puberty, at eight to ten years of age, or prior to five years of age. Physicians advising observation through puberty for testicular descent usually are not aware of the clinical condition of retracted testicle as described. Observation to puberty allows irreversible changes in the testicle with little hope of normal spermatogenesis. Robinson and Engle[3] demonstrated the earliest degenerative tubular changes in undescended testicles of children at five years of age when compared with normally descended testicles. This finding has dictated advice to have orchiopexy performed prior to five years of age. Those who still advise observation until eight years of age make the assumption that the changes noted are not significant or permanent if orchiopexy is carried out by this age.

The proper age for orchiopexy is between two and five years of age. The associated hernia sac with the undescended testicle is extremely thin and the operation is technically difficult prior to two years of age. If complications such as incarceration of the hernia are noted, the operation can be performed in infancy by an experienced surgeon.

Indications for Orchiopexy

In addition to the previously stated indication for surgery for possible preservation of spermatogenesis, parents should be told of the presence of an indirect inguinal hernia in association with a true undescended testicle in 92 per cent of patients. Performance of orchiopexy for the cosmetic effect of placement of the testicle in the base of the scrotum is also important. It is known that tumor formation in an undescended testicle is more common than in a normal descended testicle. However this may well be related to an inherent defect of the testicle and not to its position.[1] Therefore orchiopexy may have no influence on the tumor problem except to allow easier diagnosis by palpation. Parents should not be advised that surgery is indicated to prevent tumor formation.

Patients with bilateral undescended testicles are considered to be sterile without proper operation. Patients with a unilateral undescended testicle will not be sterile as long as the normal testicle is preserved.

Surgical Technique

The method of orchiopexy utilized is the method described by Koop.[2] A transverse skin crease incision is made with inguinal canal dissection to expose the testicle and hernia sac. The hernia sac is dissected off the cord structures and ligated at the internal ring. Vascular length for placement of the testicle in the base of the scrotum is obtained by the dissection of the inguinal hernia to the inguinal ring, retroperitoneal dissection posterior and superior to the inguinal ring, and separation of the vascular structures and cremasteric muscle fibers of the cord. In the majority of cases, this dissection will allow placement of the testicle at the base of the scrotum without significant interference of blood supply to the testicle. The inferior epigastric vessels are not severed. Wide retroperitoneal dissection is seldom utilized. The testicle is placed in a dartos muscle pouch formed in the anterior wall of the scrotum. This pouch is necessary to fully dissect the scrotal cavity to the base of the scrotum. The testicle is maintained in this position by a silk suture placed through the base of the scrotum. The suture is taped to the thigh by an attached rubber band. This is not a traction suture and it is often discarded if vascular length sufficient to place the testicle in the base of the scrotum is easily obtained. The total hospitalization time required is seldom over 48 hours.

Complications

Operative complications include the complications of inguinal herniorrhaphy. In addition, a basic problem is interference with the blood supply of the testicle. Atrophy of the testicle can occur with overzealous vascular dissection or excessive traction on the testicle. Due to the excessive traction often placed on the vessels by the Torek operation, this procedure of implantation of the testicle into the thigh is not advocated. Parents should be warned of the possibility of atrophy after any technique to bring the testicle into the scrotum.

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Follow-up Study

Parents considering orchiopexy for their son should be warned that perhaps 20 per cent of undescended testicles are abnormal testicles and probably will not produce normal sperm regardless of position.[1] The identification of testicles that will not be protected by orchiopexy prior to five years of age is impossible by inspection and probably impossible by biopsy at the time of orchiopexy. Conclusive follow-up studies by testicular biopsy have not been done to give statistical proof of the effect on spermatogenesis of the undescended testicle brought into the scrotum by proper technique before five years of age. Operations properly performed by this age have been available for approximately 15 years and information as to the true effect on spermatogenesis should be forthcoming soon. Any conclusive arguments as to the effect of orchiopexy must await these biopsy studies.

Summary

Management of the undescended testicle in infants and children is described. Emphasized are diagnosis, the proper age for surgery, and surgical technique. The common retracted testicle should be differentiated from the true undescended testicle to determine which testicle requires scrotal placement by surgery. The optimum age for orchiopexy is from two to five years of age.

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Management of Imperforate or Ectopic Anus

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SYMPOSIUM

THE TERM *imperforate anus* in itself is misleading when used collectively to describe all anatomic variations occurring with the absence of a normal anal opening. The more accurately descriptive terms, *ectopic rectal opening* or *ectopic anus,* could better be used in discussing these malformations of the recto-genito-urinary complex,[1, 2] and at the same time discourage the mistaken aura of simplicity associated with the former term.

As is the case with most serious anomalies requiring surgical intervention during the first few days of life, the occurrence of ectopic anus is relatively rare. Consequently, the thought given to this specific problem by the average physician and surgeon, teachers, and textbooks is pitifully little. Knowledge of all aspects of the problems encountered during management of every variation of "imperforate anus" would easily fill a sizeable booklet, yet most adequately trained surgeons still harbor the concept that the average age occurrence of an absent anus is a simple problem easily and satisfactorily corrected with a simple perineal incision to open a thin anal membrane which simply failed to disintegrate during embryonic life. Though this actual situation has been known to occur it has been infrequently observed and is absent in many large series. When the truth of the situation is realized too late, the operating surgeon may have wrought irreparable damage and the best opportunity to obtain the possible desirable result is forfeited.

The future well-being and happiness of such a child may in a large measure be determined by the adequacy of the first operative procedure. Any oversight, failure, or complication stemming from this first attempt at correction will almost certainly compromise any subsequent necessary revision should the first procedure prove unsuccessful. Prerequisite to consistent successful correction is an accurate knowledge of the anatomic varieties known to occur, together with an understanding of the embryonic development of the hindgut, anus, and lower genitourinary organs in both male and female.

The absence of a normally developed anorectal outlet has been graphically described as the result of "failure of migration of the rectal opening" in early embryonic life after the hindgut and urogenital sinus have formed a common cloaca.[1]

Classification

Bill and Johnson[1] provide understanding of the simple as well as complex varieties of recto-genito-urinary anomalies heretofore referred to as "imperforate anus." An excellent and simple classification of the many known anal anomalies has been suggested by Gans and Friedman.[2] Defining *ectopic anus* as any abnormally located opening of the terminal bowel, the anatomic site of this opening defines the type of anomaly. Thus, the terms *vesical ectopic anus, urethral ectopic anus, vaginal ectopic anus,* and *perineal ectopic anus* classify the known varieties of ectopic anus (Figs. 1 and 2). The anatomy in the extremely rare *membranous imperforate anus* (Fig. 3a) is in normal position with no developmental anomaly other than simple persistence of the cloacal membrane. Excepting this latter specific situation, the ectopic rectal opening is almost always found at some point along the midline route of "rectal migration" in every subject lacking a normally developed anus. Such assumption has important bearing upon the proper method of therapy in each type of ectopic anus.

Vesical and urethral ectopic anus occurs most frequently and almost exclusively in the male, whereas high vaginal ectopic anus

A suggested simple classification for anatomic variations which occur when a normal anal opening is not present is given. In addition, the author lists his preferences for surgical correction of each type.

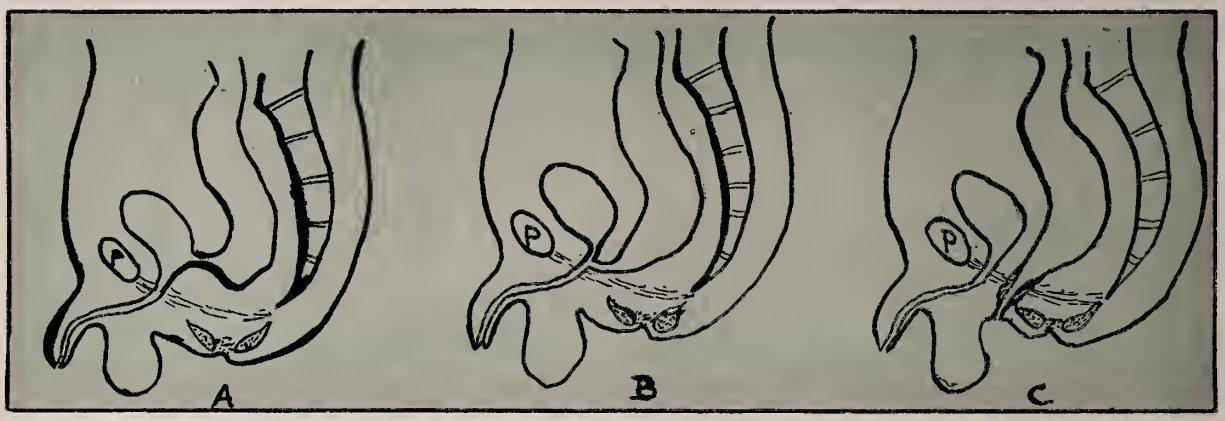


Fig. 1. Anal ectopia in the male: a. vesical ectopic anus, b. prostatic urethral ectopic anus, and c. perineal ectopic anus.

is relatively rare (Fig. 2c). Perineal ectopic anus may occur in either sex and is considerably more common in the female.

Numerous technical methods have been described for correction of these various anomalies, and experience has proved no method of management is universally applicable. Each type requires individual consideration and application of a technique most suitable to the circumstances involved. No attempt will be made here to describe in detail the technical procedures most preferred, but some emphasis will be placed upon the importance of definition of each anomaly complex so that the optimum procedure might be selected for each case. Technical details of the recommended surgical procedures may be found in specific references.[1, 2, 4-6]

The ultimate solution to every known type of ectopic anus has yet to be revealed, but current knowledge and experienced skill can offer a satisfactory prognosis in most cases. The chance of obtaining completely normal anorectal function is possible only when the lower rectum with its internal sphincter and pelvic innervation are well developed and can be carefully preserved (Figs. 1c, 2a, 2b, 3a). In this most favorable circumstance, the rectum extends caudally below the sling-like puborectalis fibers of the levator ani muscle, and its external opening will be seen in the perineum or vulva somewhere anterior to the normal anal site. Meconium or gas may pass from this ectopic opening, which may be large or small, and this opening will invariably be in continuity with the main rectal canal within 1 to 1.5 cm. of the perineal skin surface (except as in Fig. 3a).

In cases of ectopic vesical, ectopic urethral, and high vaginal ectopic anus, the urorectal septum failed to develop and completely separate the rectum from the urogenital sinus, leaving a persistent communication between rectum and urinary or genital tract. The level of this communication usually, but not always, lies above the levator ani muscle, and the distal-most end of the rectum seldom is found extending below the levator ani muscle. The specialized circular muscle fibers forming the internal sphincter in a normally developed rectum do not form at this high level and the infant born with this less favorable situation will never have the functional advantage of internal sphincter action. Selective control of flatus and liquid stool will likely be poor under these circumstances, though surprisingly good continence of solid stool can be obtained. Bowel habit training carefully encouraged after the child reaches a cooperative age (four to five years of age) can result in satisfactory bowel control far superior to that obtained with a permanent colostomy. There are, fortunately, only rare occurrences of such extensive maldevelopment of the perineum, coccyx, lower sacrum, and rectum for which satisfactory anoplasty by any presently known procedure is not feasible. In these few instances a permanent abdominal colostomy is the most satisfactory answer yet devised.

Corrective Approaches

The basic technical approaches to the correction of anal ectopia are through the perineum alone or through a combined abdomino-perineal attack. Selection of the proper approach should be governed by the specific critical factors existing in each given case. Careful inspection of the perineum of

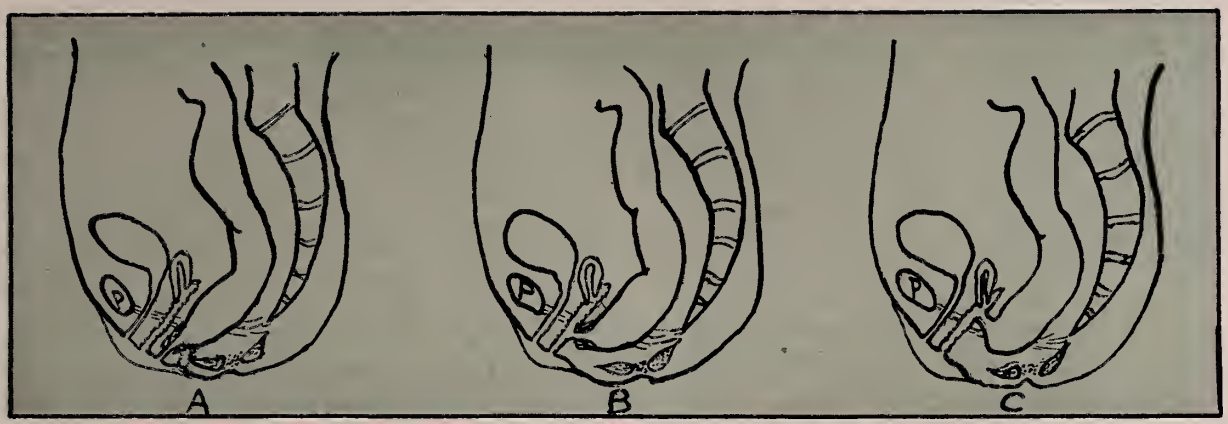


Fig. 2. Anal ectopia in the female: a. vulval ectopic anus or perineal ectopic anus, b. low vaginal ectopic anus, and c. high vaginal ectopic anus.

the unanesthetized infant should reveal one or many such factors:

1. The visible presence or absence of a rectal outlet.

2. The position of the center of the superficial sphincter muscle, if present at all, can be seen when the perineum is sharply thumped with the finger to stimulate contraction of the muscle. This point is important in properly placing an opaque marker for reference in determining by roentgenography the position of the distalmost rectal pouch in relation to the anal skin.

3. The presence of a midline dimple or tissue mound in the perineum, as a rule, does not mark the position where the anus should be or where the center of the superficial sphincter muscle exists.

4. If no perineal ectopic opening is seen and no meconium is seen in the vagina or male urethra, moderate pressure might be exerted upon the lower abdomen to compress the sigmoid colon and urinary bladder, which may cause passage of gas or meconium from the vagina or urethra and perhaps make visible a tiny opening in the perineum previously undetected. Meconium stain might be seen in expressed urine.

5. A fluctuant bulge at the normal anal site may indicate that the rectal pouch is very close to the skin (Fig. 3a).

The need for surgical intervention for correction of an ectopic anus *per se* is never urgent immediately following birth, though there may be other serious anomalies existing which require immediate relief. The frequency of coexisting anomalies is approximately 30 per cent, and they account for the major share of mortality associated with surgery performed for correction of anal ectopia. Much useful information may be gained by procrastinating 12 to 24 hours and observing the child while swallowed air traverses the intestine to fill the large bowel. Inversion of the infant and gentle kneading of the lower abdomen should encourage gas displacement of meconium in the distal rectal pouch. With an opaque

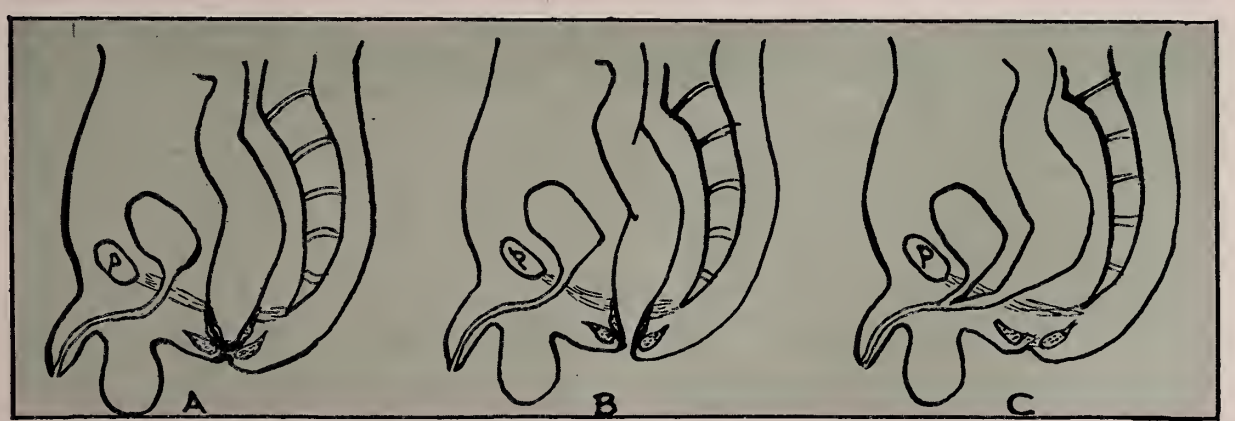


Fig. 3. a. True membranous imperforate anus (rare) with no ectopia of anus, b. normally developed rectum and anus, and c. low membranous urethral ectopic anus (male).

marker placed at the superficial anal sphincter, a seven-foot lateral roentgen film exposure centered across the intertrochanteric line should provide an accurate measurement of the distance between the anal marker and distal end of the gas-filled rectal pouch.[8]

The perineal approach as specifically described by Potts, Riker, and DeBoer[4, 5] is recommended for correction of the anomalies of perineal ectopic anus in both male and female subjects. Whenever the rectal pouch is demonstrated to lie within 1.5 cm. or less of the anal skin, this technique is most successfully utilized. Great care must be exercised to avoid injury to the male urethra, and every effort should be made to avoid traumatization and unnecessary cutting of the anal sphincter muscles. The perineal approach is advised only when there is reasonable assurance that the rectum can be adequately mobilized to permit anastomosis of its distal end to the anal skin without any tension whatsoever at the suture line.

Anal ectopia in the female is most frequently seen as a small opening communicating directly from the distal end of the rectum to the fossa navicularis or perineum immediately behind the posterior fourchet (Fig. 2a); less frequently the opening will be found at the hymenal ring and even less often in the lower posterior vaginal wall (Fig. 2b). In any of these situations the tract is short and elastic and can easily be gently dilated with a fusiform instrument to attain the size of an average wooden pencil. Through this adequate opening the child may easily pass gas and feces with no complications or threat to health for an indefinite time, so long as the tract is kept adequately open and is not torn. Whenever the child reaches the size and age preferable to the surgeon, the anomaly may be corrected as recommended by Potts.[5] It is quite important, as is emphasized by Potts, that females with these specific forms of the anomaly have a definitive corrective anoplasty performed well before the age of six months, if all is otherwise well, in order that the necessary three months of painful postoperative anal dilatations may be past before the child retains permanent memories of these unpleasant daily experiences.

The situation is not the same in the male with perineal anal ectopia, for the length of the tract from rectum to skin is usually longer in the male and is usually surrounded by a tough cuff of fibromuscular tissue which does not yield readily to stretching dilatation. Instead of stretching it will frequently tear, and will not remain satisfactorily dilated as does the shorter tract in females. For this reason the author prefers to proceed with perineal anoplasty as primary treatment of this specific situation in the newborn male.

If the rectal pouch lies at a distance greater than 1.5 cm. from the anal skin, adequate mobilization of the rectum is not feasible through total perineal approach without unwarranted mutilation of the perineal tissues and destruction of the superficial sphincter and puborectalis sling. In all such circumstances, including *high* vaginal ectopic ani (Fig. 2c) and all vesical and urethral ectopic ani (Figs. 1a, 1b), excepting rare low membranous urethral communication (Fig. 3c), a refinement of the combined abdominoperineal approach originally described by Rhoades, Pipes, and Randall[6] has yielded gratifying results. By this technique the rectum can be adequately mobilized, the anomalous communication to the bladder or prostatic urethra or upper vagina can be seen, divided, and closed through the abdominal approach, and with such exposure care can be exercised to avoid destruction of the pelvic autonomic innervation to the lower bowel and seminal apparatus when mobilizing the rectum and sigmoid colon.[7] The only perineal manipulation necessary is accomplished through a small 1 to 1.5 cm. sagittal skin incision placed over the center of the superficial sphincter muscle. The mobilized rectum is then grasped with a large curved instrument previously passed retrograde through the superficial sphincter muscle fibers to create a canal anterior to the puborectalis muscle sling and pulled down along this canal to be sutured to the anal skin without tension. This procedure can be well tolerated by the average full-term infant, but is ill-advised for the premature child or one afflicted with other serious anomalies in whom the problem of bowel obstruction would best be alleviated by creation of a temporary transverse colostomy.

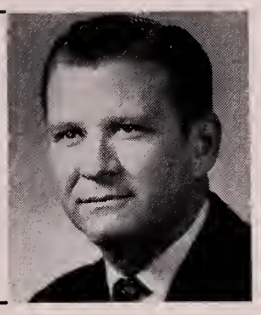
In any circumstance where demonstrable tension exists at the anal suture line, the sutures will soon cut through the tissue and permit retraction of the rectum towards the pelvis, leaving raw tissue exposed to granulate and become infected. The resulting permanent cicatrization and fibrosis of the peri-

rectal and perianal tissues with entrapment of muscle fibers in scar is irreversible and incorrectable even if anal stricture is prevented by diligent daily dilatations of the anal canal.

All infants who have undergone anoplasty by any of the mentioned techniques should receive daily anal dilatations beginning 10 to 14 days after surgery and continuing throughout a period of no less than three months to avoid progressive stenosis of the anus and rectal canal. Dilatations should be started with a relatively small fusiform dilator gradually progressing in size until the mother's smallest finger (covered with a rubber finger cot and well-lubricated with water soluble lubricant) can be inserted past the second knuckle. As the baby grows a larger finger may be used, but care should be exercised never to tear, only gently stretch, the anorectal tissues. Surgical creation of an anus large enough so that dilatations should not be necessary usually results in undesirable division of some of the superficial sphincter muscle fibers and leaves a large, patulous, ectropic anus undesirably increasing in size as the child's body grows.

Complicating Anomalies

One should be well prepared to deal with other complicating anomalies of the urinary and enteric organs frequently found in combination with anorectal anomalies. The myenteric plexus in the distal rectum or colon is improperly developed in a significant number of infants born with anal ectopia, and these children will ultimately develop clinical manifestations of Hirschsprung's disease. This additional anomaly has been found in 21 histologically proved cases reported by Sulamaa and co-workers in Helsinki[3] and corroborated with fewer but similar experiences by several independent workers in Sweden and the United States. Congenital bladder neck obstruction, hydroureter and hydronephrosis, uterine and vaginal atresia are some of the more frequent associated anomalies in the immediate anatomic area, while esophageal atresia and other points of bowel obstruction occur not infrequently in the same individual. The mere coexistence of more than one serious correctable lesion does not render the situation hopeless, although it is precarious. However, with experienced judgment, skilled technical surgery, and knowledgeable postoperative care, most such babies can survive to grow into healthy, normal citizens.



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A newborn child with anal ectopia can usually be moved safely to facilities especially equipped and having personnel experienced in the care of the newborn critically ill. Any resulting inconvenience or added expense to the parents or community will likely be more than compensated if the first surgery attempted is done properly and successfully. Should it become mandatory that a physician inexperienced in the surgical procedures discussed be forced to afford some means of relief for the colonic obstruction, the wisest procedure would be creation of a simple loop colostomy in the left transverse colon with no attempt at correction of the anal anomaly at that time.

Summary

The embryologic origin of the known variations of imperforate and ectopic anus is discussed together with a suggested simple anatomic classification of each type. In the author's opinion, the best known methods for surgical correction of each type variation is cited. Reference to the original descriptions of these techniques is urged for technical details of surgery.

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Surgical Lesions of Lung and Thorax In Infants and Children

SYMPOSIUM

PERHAPS IN no other area of the body are lesions so amenable to prompt, proper surgical therapy as those of the thorax and parietes. This is particularly true in infants and children, in whom malignancy of the lungs or pleura is rare.

For discussion these lesions will be grouped according to their location in the thoracic wall, the pleura, or the lung.

Thoracic Wall Lesions

Visible lesions of the chest are usually of soft tissue origin. These lesions and their treatment are briefly outlined.

1. *Gynecomastia* without endocrine cause in the young male, a cause of embarrassment and psychological trauma, is curative by simple excision of the redundant tissue.

2. In the breasts of the prepubertal and pubertal female, *painless lumps* of various sizes are found frequently. Treatment is reassurance and not surgery.

3. *Mastitis with abscess* is seen frequently in the infant. Two parallel incisions, wide apart, with a drain through and through is needed; culture and sensitivity studies will decide the antibiotic choice.

4. *Lipomas* are readily diagnosed as sharply delineated, nontender, mobile, subcutaneous masses. They are removed only if location subjects them to trauma or if they are large.

5. *Malignant melanomas* are extremely rare in children. A pigmented (junctional cell) nevus is common. Should the nevus show any change in color or size or be subjected to repeated trauma, excision is mandatory. In the prepubertal and pubertal age close scrutiny is necessary because there is increasing tendency toward malignancy as adulthood is entered. The large hairy nevus is removed only for cosmetic reasons. A skin graft is sometimes needed to achieve a good result.

6. *Hemangiomas* rarely require removal. Giant cavernous hemangiomas with thrombocytopenia pose a difficult problem. Infants with these die from generalized bleeding. When located on the chest, excision is difficult if not impossible. Treatment by heparinization, to release thrombocytes sequestered in the hemangioma, combined with roentgen-irradiation has been reported. Fortunately these lesions occur more often in extremities where removal is possible. Hemangiomas of the rib produce irregular erosion. Surgical excision of the segment of rib involved with ligation of all tributaries is sometimes needed.

7. *Dermoids or sebaceous cysts* are excised as in other cutaneous sites.

8. *Osteomyelitis* of the rib may appear as a solitary lesion of bone destruction difficult to be distinguished from eosinophilic granuloma or a single metastatic lesion. The solitary lesion may be found fortuitously on a roentgenogram taken for another reason, although swelling and tenderness over the involved rib are more commonly associated. Diagnosis is made by excisional biopsy. Appropriate antibiotic therapy follows.

9. *Eosinophilic granuloma* is a nonlipid reticuloendotheliosis. It occurs in the skull, long bones, and occasionally in a rib as a punched-out bone lesion with little or no evidence of systemic disease. Excisional biopsy or curettage is curative.

10. A *metastatic lesion* in the rib from neuroblastoma or osteogenic sarcoma is usually associated with evidence of the primary tumor. The roentgenographic characteristics aid in identifying these metastases, but a rare single metastatic lesion may require removal for diagnosis.

Lesions of the thoracic wall, the pleura, and the lung are reviewed together with an outline of diagnostic features, surgical indications, and operative management.

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11. *Bone cysts* of the rib are managed by curettage and filling the cavity with bone chips.

12. *Pectus excavatum*, or funnel breast, is a deformity of the sternum and cartilaginous ribs producing depression of the sternum of various degrees. The heart is displaced to the left and respiratory function impaired. In the more severe cases, the xiphoid touches lower thoracic vertebrae. Unequal growth of ribs, sternum, and cartilages, abnormal bands between the sternum and diaphragm, and heredity may all have a causative role.

13. *Abdominal protrusion and kyphoscoliosis* give children a unique appearance. Disturbances of respiratory and cardiac functions are indications for surgical correction. Repair for psychological reasons is warranted. (The number of men in the armed services during World War II who requested surgical correction of mild degrees of funnel breast attests to the fact that this deformity is psychologically traumatic.) The optimum age for correction is between four and nine years. There is about a 50 per cent recurrence rate with a less extensive procedure such as division of sternal attachments of the diaphragm.

14. *Deformities of one or two ribs*, if severe enough, can be corrected by local subperiosteal excision of the deformed segment.

15. *Congenital absence of one or more ribs* is associated with a hemivertebra. Ballooning of the soft tissue in the area of the defect attracts attention to it. No symptoms are associated. Surgery is contraindicated.

Pleural Lesions

These lesions and their treatment are outlined.

1. *Hemangio-endothelioma* of the pleura is rare. It can be associated with extreme pulmonary distress, particularly when bleeding occurs. Extensive lesions of other viscera often are associated. Melena, hematochezia, bloody pleural effusion, dyspnea, pallor, anemia, and associated symptoms occur in various combinations.

The smaller lesions of the pleura can be removed surgically. The prognosis is cautious since undetected lesions of the viscera can cause death later. A case of extensive involvement of both visceral and parietal pleura in a ten-year-old girl has been reported by the author.* Decortication of both lungs in two stages seemed to have solved the problem. The child died suddenly from cardiac tamponade from a bleeding varix within the pericardium.

2. *Pleurisy* is not a surgical lesion except as it enters into the differential diagnosis of other lesions. Tumor, bacteria, viruses, trauma, and irritation are causes. A massive pleural effusion may have to be drained by closed thoracotomy if it reaccumulates after thoracentesis and is the cause of respiratory embarrassment. Diagnosis is aided by smear, culture, and cell block of fluid.

3. *Chylothorax* due to damage to the thoracic duct following open heart and vascular procedures has been seen several times by the author. All responded completely to closed thoracotomy and drainage for several days.

4. *Empyema* requires closed thoracotomy and drainage. The chest tube is inserted at the time roentgenographic evidence of empyema is shown. One should not temporize with repeated aspiration. Suction is applied to the chest tube with periodic irrigations with saline and proteolytic enzymes to keep

*Children's Quarterly Report, Children's Memorial Hospital, Chicago (Summer) 1963.

the tube open. Culture and sensitivity studies are done. Pleural thickening following pleurisy or empyema can produce atelectasis with chronic infection in the contiguous lung tissue. Decortication is indicated in long-standing cases which are unresponsive to nonoperative therapy.

5. In this age of high speed vehicles, *trauma* to the chests of infants and children is becoming more common.

Sucking chest wounds are treated immediately with bulky airtight dressing to prevent air exchange while shock, head injuries, bleeding, and other more life-endangering injuries are treated. Debridement of the wound, hemostasis, and closure of the chest wound in an airtight manner followed by continuous negative pressure of 18 cm. of water to the chest tube to aid expansion of the lung summarizes the treatment.

Crushing chest injuries require attention to underlying lung damage. A chest tube is inserted for tension pneumothorax or hemothorax. Sternal traction may be needed for flail chest, and tracheotomy may be required to remove pulmonary secretions since coughing is limited by pain.

Intercostal nerve blocks at four to six hour intervals for 24 to 48 hours will control pain and aid in respiratory function. After this period the pain is not great.

Tension pneumothorax is treated by insertion of a catheter into the second interspace on the affected side. The catheter is connected to an underwater seal.

Pneumomediastinum in the newborn and pneumothorax may be life endangering unless quick action is taken. Reaccumulation of pneumothorax after catheter insertion suggests pneumomediastinum with dissection of air along the bronchial tree. A tube should be placed in the mediastinum. Occasionally, opening the superior mediastinum through a suprasternal transverse incision will suffice.

6. Surgery of *fungus infections* of the lungs and pleura, such as actinomycosis and candidiasis, is limited to drainage of empyema or abscess and occasional resection of pulmonary tissue.

Lung Lesions

Lesions of the lung and treatment are listed.

1. Children put anything and everything in the mouth. Aspiration of *foreign bodies* such as nuts, vegetable seeds, food, pins, plastic objects, small particles of wood, marbles, beads, and many other objects is common. There is usually a severe tussive episode followed by a typical wheeze, often noted without a stethoscope. Radiopaque articles are seen on roentgenogram. Bronchoscopic removal is usually successful. The nonradiopaque articles can be detected on fluoroscopy by delay in filling and emptying of a lung segment. Small particles in a secondary or tertiary bronchus may require thoracotomy for removal. A foreign body left in place will eventually lead to lobectomy or pneumonectomy because of obstructive emphysema and infection.

2. *Reinfection cavitary pulmonary tuberculosis* in children is best treated with chemotherapy and no surgery. The occasional case of a single cavity not responding or a single lobe or segment involved may be treated by lobectomy, segmental resection, wedge resection, pneumothorax, pneumoperitoneum, or combinations along with chemotherapy.

3. *Bronchiectasis* occurs less frequently than in the days prior to antibiotics. There are several causes with no single mechanism common to all cases. Congenital defects in the bronchial wall, chronic infection with fibrosis limiting the elasticity of the bronchial wall, sinusitis, foreign body such as a seed or grass particle, cystic fibrosis of the pancreas, and agammaglobulinemia are noted. Diagnosis is suggested by repeated attacks of pulmonary infection with progressive severity, a chronic cough producing a purulent, fetid sputum, persistent rales in the basilar areas of the lung, and slight morning fever. Diagnosis is proved by bronchogram.

Antibiotic treatment with postural drainage and inhalation therapy should be tried first. If not curative, this regimen prepares the patient well for surgery. Lobectomy or segmental resection, or both, accomplishes excellent results when postoperative medical therapy is carefully applied.

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4. *Postpneumonic cysts* of the lung and *congenital cysts* can be differentiated by a history of pneumonia, a prepneumonic roentgenogram showing absence of the cyst, and rapid decrease in size and disappearance of the postpneumonic cyst. Congenital lung cysts are less frequent and usually occur in the first six months of life, although they are occasionally seen in children or teenagers. Occurrence in the infant may produce enough respiratory embarrassment to endanger life. Immediate aspiration is followed by surgery. The infant should never be left unattended until the cyst is removed. A lobectomy or rarely a pneumonectomy is needed when cysts are generalized in one lung. Replacement by new alveoli occurs in the infant in contrast to replacement by emphysema of adjacent alveoli in adults.

Recurrent accumulations of pus in the same area of lung in children and teenagers can be caused by a congenital lung cyst. Differentiation from postpneumonic lung cyst is difficult in this age group. Microscopic evidence of respiratory epithelium lining the cyst and a wall with smooth muscle and/or cartilage is diagnostic of congenital lung cyst. Postpneumonic lung cysts are watched expectantly for progressive respiratory distress. Surgical removal usually is not necessary.

5. *Bronchial cysts* arise from the trachea or a major bronchus and produce symptoms by compression of a bronchus. Removal is tedious since the cyst is intimately associated with the bronchial wall. The lesion is approached through an anterolateral thoracotomy. Removal of the cyst is followed by immediate respiratory improvement.

6. *Congenital lobar emphysema* causes slowly progressive respiratory distress in infants of six months or less at time of onset. The cause is a congenital malformation of the cartilage of the bronchial wall permitting ingress, but not normal egress of air. Roentgenogram shows an emphysematous lobe resembling congenital lung cyst, but a faint outline of the structure of the lung can be seen throughout the distended lobe. Treatment is lobectomy.

7. *Congenital bronchial stricture* manifests itself by recurrent infection limited to one lobe or one lung. Bronchogram reveals the stricture. Lobectomy or pneumonectomy may be required.

8. A *sequestrated lobe* results from a developmental anomaly in which there is no connection between the bronchus of the lobe and the main bronchus. The lobe is never expanded. Thoracotomy with removal may be necessary for diagnosis.

9. The *accessory lobe of Wrisberg*, or azygos lobe, revealed in about 1 per cent of chest roentgenograms of patients of all ages is important only in that it may be confused with other conditions.

10. The *middle lobe syndrome* caused by compression of the middle lobe bronchus by lymph nodes is rarely encountered in young children. In older children and teenagers it manifests itself by recurrent infection, fever, hemoptysis, and chronic cough. Bronchography or bronchoscopy establishes the diagnosis. As in congenital stricture, lobectomy is the treatment.

The fascinating and challenging tumors of the mediastinum, vascular rings, tracheoesophageal fistula, esophageal atresia, congenital heart lesions amenable to open heart and vascular surgery, diaphragmatic hernias, and other lesions directly or indirectly affecting the lungs are beyond the scope of this paper.

Summary

Surgical lesions of the lungs and thorax in infants and children are reviewed. Diagnostic features, surgical indications, and operative management are discussed briefly.

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There's a new disease going around that might be termed "TV medicitis," according to Dr. A. M. Aibinder, New York City.

"Certain television viewers watching medical dramas, by identifying with the actors, tend to 'catch' the disease being portrayed," Dr. Aibinder wrote in a letter published in the Jan. 25 *Journal of the American Medical Association*. "Convinced they have the disease, they visit their doctor and even request the treatment they saw administered on TV."

Juvenile Diabetes:
Early Observations of 181 Cases

CASE REPORTS

CLINICALLY, THERE are two types of primary diabetes mellitus. Though there are a number of recognized classifications, the following are convenient and applicable to this discussion:

I. Juvenile type diabetes, insulin dependent.
   A. Onset under age 19. Onset during growth.
   B. Onset 19-40 years.
   C. Onset 40 years and thereafter.
II. Adult type diabetes, insulin production impaired.
   A. Onset over age 40. Frequent.
   B. Onset under age 40. Occasional.

It is the purpose of this paper to analyze the characteristics of those individuals whose diabetes was discovered before age 19, who qualify as true juvenile, or "growth onset," diabetics. Carefully conducted surveys indicate there are approximately 2,000,000 people in the United States who have known diabetes, and another 1,000,000 whose diabetes is undiscovered. It has been difficult to assess the number of true juvenile diabetics, but it is thought they represent roughly 5 per cent of the known diabetics; another source indicates that one out of every 2,500 children has diabetes.

The conclusions drawn from this series of patients are not new; they have perhaps been documented before and, unfortunately, in some respects are incomplete. These patients have been seen personally in the office of a private practitioner, chiefly during the last 16 years, though one has been seen for 26 years. Sixty-nine of these patients have been treated by the author since the discovery of their diabetes, followed through the years, and are still his patients. One hundred of these patients have been seen within the past year. Some of these patients were seen on consultation only, and others have moved from the community. In the latter instance an effort has been made, with considerable success, to follow the destinies of those patients now living elsewhere. Unfortunately, some data which is now desirable has not been recorded. In many instances dependable conclusions cannot be drawn; only impressions may be presented.

Diabetes is unique in that there are no known cases of this disorder being present at birth, but this peculiar metabolic syndrome may appear any time thereafter, from infancy to old age. For thousands of years prior to the discovery and utilization of insulin in 1921-1922, the child who developed diabetes had a dismal and lethal future with wasting of the body and an inevitable death from keto-acidosis, usually within six months. However, with modern treatment a child may now be restored to good health, grow and develop normally, and live a long time—possibly four-fifths of his normal life expectancy. It is not known how many diabetics are surviving and in good health after having taken insulin for the 42 years of its availability, but the number is probably in the hundreds. The incidence of childhood diabetes is greater at the present and will undoubtedly continue to increase for self-evident reasons.

General Findings in This Series

In this series of 181 cases, the age of discovery of diabetes is shown in Table 1.

The average age of clinical onset of diabetes in this series is 9.39 years. The highest incidence of onset was in the age group from 10 to 12: age 10, 12 girls and 2 boys; age 11, 12 girls and 5 boys; and age 12, 9 girls and 7 boys.

Early observations in 181 cases of juvenile diabetes are presented, with brief data on the 11 deaths which occurred. Average age of onset in the series is 9.39 years; the patients have been observed an average of 12.5 years. Typical onset, diagnosis, and treatment are summarized.

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In this series there were 79 boys and 102 girls. At clinical onset of diabetes, the histories revealed there were 75 cases in which there was known diabetes in the family history; 83 cases in which there was no known diabetes; and 23 cases in which there was some question. These figures are unimportant inasmuch as many of the ancestors involved perhaps had undiscovered diabetes. When there was a known history of diabetes, it most often involved remote relatives, such as aunts, uncles, or grandparents. Only in 12 cases was diabetes in the family known to precede or follow the diabetes of the child. One child had a diabetic mother and 11 children had diabetic fathers.

TABLE 1.—*Age of Discovery of Diabetes.*

Age at Discovery	Number	Age at Discovery	Number
1	1	10	14
2	8	11	17
3	13	12	16
4	12	13	11
5	7	14	9
6	13	15	11
7	11	16	3
8	12	17	7
9	11	18	5

Eight parents developed diabetes prior to onset of diabetes in the child; one parent developed diabetes one year after the child; two parents developed diabetes five years after the child; and one parent developed diabetes 16 years after the child.

The average present age of the patients of this series is 22.06 years and the average duration of diabetes is 12.5 years. One patient has been taking insulin for 42 years and continues in good health, having had a myocardial infarction four years ago from which he recovered.

Onset and Diagnosis

There is a relatively consistent composite picture of the child who develops diabetes. The child most often has been in good health, of normal weight, and his parents do not have known diabetes. Usually, there is a rather abrupt and explosive onset of the classic symptoms of thirst and resultant excessive urination, accompanied by a normal to increased appetite. As a rule, within the following two or three weeks the child develops paradoxical weight loss and soon thereafter will manifest apathy, irritability, and a disinclination to follow the normal activities of a healthy child.

Prior to the last 20 years, before parents and physicians were alerted to the early symptoms, the child with new diabetes would often be diagnosed in a state of clinical ketoacidosis. Much credit is due the American Diabetes Association and its affiliates for an educational program which has alerted all to the symptoms of childhood diabetes, and early diagnosis results from this knowledge. In one instance a mother, having read of diabetes, secured a simple sugar-testing device from her pharmacist and made a diagnosis on her own child.

When childhood diabetes actually begins is not known. It is thought the disorder is predestined from conception; that somewhere in the genes which produced the child there is a component which inevitably will produce diabetes. As stated before, the onset of symptoms represents the "explosion" and then clinical diabetes is recognized.

The diagnosis of juvenile diabetes is usually rather simple. The symptoms are present, and glycosuria and ketonuria supported by elevated blood sugars, taken at almost any time of the day, are diagnostic. Rarely is it necessary to do a glucose tolerance test.

For example, in case 33, the glucose tolerance test was entirely unnecessary and could have been harmful (Table 2).

TABLE 2.—*Case 33, Glucose Tolerance Test.*

Time	Blood Sugar Per 100 cc.	Urine Sugar
Fasting	336 mg.	2 +
30 min.	373 mg.	2 +
1 hour	474 mg.	2 +
1½ hours	551 mg.	2 +
2 hours	703 mg.	4 +
3 hours	415 mg.	4 +

On the other hand, occasionally there is a relatively slow onset of clinical symptoms in early diabetes and a glucose tolerance test is of importance. It is stated that this occurs in approximately 4 per cent of juvenile diabetics, but in this series only 3 per cent are so classified. Case 174 presented a variable glycosuria and uncertain blood sugars, and the glucose tolerance test was extremely helpful in establishing the diagnosis of diabetes (Table 3).

TABLE 3.—*Case 174, Glucose Tolerance Test.*

Time	Blood Sugar Per 100 cc.	Urine Sugar
Fasting	126 mg.	Neg.
30 min.	243 mg.	1 +
1 hour	290 mg.	4 +
2 hours	214 mg.	4 +
3 hours	Not done	4 +

Treatment

Certain principles in the treatment of the newly discovered juvenile diabetic appear valid. At onset, the diabetic child not in clinical ketosis and able to take food by mouth should be placed on a liberal and adequate diet based on his requirements as dictated by age, and designed to contribute to normal growth and development. The child usually has been in a temporary state of catabolism and starvation, and his requirement for food appears relatively enormous. Though he may be "de-sugarized" with regular insulin for a few days, on the other hand, either lente or NPH insulin may be used initially and, over a period of two or three weeks, either will tend to produce both clinical and physiological improvement. It is important that the diet be distributed to fit the absorption "timing" of these insulins. After the diagnosis has been established, blood sugars are rarely helpful except in emergencies, and the insulin dose can be adjusted by routine testing of the urine for sugar before each meal and at bedtime, the goal being two or three negative specimens daily. A test for acetone should be done at least once a day, and more often if the diabetes appears to be unstable or if stressful situations are present.

It should be recognized that the dramatic state of clinical keto-acidosis, with all its implications regarding health, hospitalization, and finances, is an entirely preventable catastrophe; therefore, the daily acetone test is particularly important inasmuch as the dramatic end result of vomiting and coma can be avoided by appropriate treatment.

The family, particularly the mother of the younger child and later the child himself, should be taught at the onset all of the fundamental principles regarding diabetes and its control to the end that they will not remain dependent upon the doctor, but rather consult him at periodic intervals for advice. They should be taught to alter insulin dosage depending upon certain specific criteria; they should be told of the ill effects of poor control on the immediate health of the patient, and of the complications of diabetes which may be put off into the distant future, or even perhaps avoided, by good control. The attitude of the physician should, in general, be optimistic in this regard.

Certain observations appear to be justified in connection with the various problems to be encountered in the lifetime of the diabetic child. Rarely have insulin-induced hypoglycemic reactions produced damage to the patient, and the patient should be encouraged not to fear insulin, but rather to use the greatest amount he can "get away with." The child should be taught to give his own insulin at the earliest date possible and to assume full responsibility of the routine testing of urine specimens and dietary control. By the arbitrary age of 14, he should be in a position to take full responsibility for his diabetic state. Attendance at summer camps for diabetic children has proved to be very beneficial in the development of independence and self-reliance, and should be encouraged.

Occasionally, fat atrophy will occur at the site of injection of insulin. It has been reported that this will happen in approximately 4 per cent of patients; in this series it was found to be present in 4.9 per cent of the patients. Atrophy of fat may occur at

Dr. Edwin L. Rippy, a Dallas internist, presented this paper to the Texas Diabetes Association at its meeting in April, 1963.

the onset of insulin therapy or at any time thereafter; it may be permanent or, as in most cases, transient. In most instances, as the child grows in musculature and weight, the areas of atrophy will disappear. The injection of insulin into the thighs and arms of young patients, particularly girls and those showing some allergic reaction to insulin, should be discouraged. An excellent site for injection is the upper abdominal wall.

Female diabetics are extremely prone to urinary tract infections and urinalyses in the office should always include the microscopic. When infection is evident, it should be treated vigorously and not allowed to become chronic. It is also to be assumed that most of the girls are constantly infected with *Candida albicans* and trichomonads, and often this may flare into an acute problem which will require long-term treatment. Young girls who have diabetes may have a late onset of menstruation and thereafter may have irregular cycles. When maturity is complete, however, they appear to be quite fertile and advice regarding pregnancy should be given early in marriage.

Rarely are the diabetic neuropathies encountered in this group, although if diabetes has been present for ten or more years, neuritis of the lower extremities or the "bladder syndrome" is occasionally encountered. Peripheral vascular disease is rarely seen, though in case 28, profound damage to the nerves of the lower extremities produced such trophic changes that infected calluses developed, and over a four-year period this patient required bilateral midcalf amputation—the only such case in the series.

Review of Deaths

In this series there have been ten known deaths, eight as a direct result of diabetes and two related to diabetes. These patients have appeared to lead charmed lives inasmuch as it is not known that any one has died as a consequence of other illness or by accident. A brief analysis of these deaths follows:

Case 3, female, developed diabetes at age three and died at age 31. She had evidence of Kimmelstiel-Wilson nephritis at age 23, and myocardial infarction at age 29. She was emotionally unstable and addicted to alcohol and tranquilizers; cause of death was thought to be excesses of alcohol and tranquilizers. She died a convulsive death.

Case 38, female, developed diabetes at age nine and died at age 23 of Kimmelstiel-Wilson nephritis and uremia. She had advanced retinitis, numerous episodes of ketosis, and very poor control of diabetes.

Case 57, male, developed diabetes at age three; died at age 28. Uremia with generalized edema resulting from nephritis was the cause of death. He had been totally blind for seven years.

Case 58, female, a diabetes patient since age three and blind for 20 years, was found dead in bed, presumably from a myocardial infarction at age 31.

Case 69, female, developed diabetes at age 18, died at age 35. She had Kimmelstiel-Wilson nephritis and uremia, with excessive blood pressures as high as 220/130 mm. of mercury. She had very poor control of diabetes.

Case 93, female, developed diabetes at age three, died at age 26. She had Kimmelstiel-Wilson nephritis with advanced retinopathy, and died of kidney failure and uremia. She had poor control of diabetes.

Case 96, female, developed diabetes at age nine, died at age 31. She had coma at onset of diabetes; ketosis on two or more subsequent occasions; and died of Kimmelstiel-Wilson nephritis with retinopathy and wet uremia, and heart failure. There was poor control of diabetes.

Case 106, female, developed diabetes at age ten and died at age 27 of Kimmelstiel-Wilson nephritis and hypertension. She had retinopathy with blindness.

Case 113, male, developed diabetes at age three, died at age 30. He had Kimmelstiel-Wilson nephritis with advanced retinopathy and died of uremia.

Case 171, female, developed diabetes at age 12, died at age 22. She had an unstable personality and numerous episodes of ketosis, but no demonstrable complications. She was married and pregnant six months, and became rebellious, stopped insulin, and started eating sweets. She developed profound ketosis, had spontaneous miscarriage, and died.

Case 173, male, developed diabetes at age three, and died at age 24 of Kimmelstiel-Wilson nephritis and wet uremia.

In analyzing these deaths, it is noted that most often death occurred as a result of kidney failure with, as a general rule, retinitis and impaired vision. When kidney failure is

the terminal cause of death, there usually is associated congestive heart failure.

It is interesting to note that in this series there have been only three clinically recognized myocardial infarctions. Case 3, female, after 23 years of diabetes, had a heart attack at age 29 from which she survived, but eventually developed congestive heart failure. Case 53, female, after 27 years of diabetes, had a myocardial infarction at age 38 from which, apparently, she recovered completely. Case 23, male, age 51, had an infarction at age 48, after 37 years of diabetes, with asymptomatic survival. Case 180, female, with diabetes 21 years, was the only patient in the series who was the victim of a cerebral vascular accident which produced right hemiplegia with only partial recovery. Her vision was good and there was no evidence of kidney disease. It would appear that coronary heart disease and cerebral sclerosis with vascular accidents occur primarily in the diabetic patient who is also aging.

Situations Encountered

There is a story worth telling in the life history of every juvenile diabetic. It is rather surprising how well the child accepts the disciplines of diabetic care, and how often it is the parents who must be conditioned. Following are certain cases in this series that present typical situations worth recording.

Case 7, male. With the classic onset of diabetes he was placed on Orinase tablets, and as a result developed diabetic ketosis. He recovered with the use of insulin.

Case 8, female. Orinase tablets at onset of diabetes resulted in weight loss of 16 pounds. Eventually, the patient required 200 units of insulin daily.

Case 20, male. He developed diabetes at age three; his older sister developed diabetes at age five. The patient was under fair control, went on a fishing weekend, failed to make sugar and acetone tests, and went into profound ketosis and coma. He recovered with treatment.

Case 45, female. She developed nephrosis at age nine, at which time she received large doses of steroids. She developed permanent diabetes; her father had known diabetes.

Case 52, female. She has had diabetes 32 years, and now is 44 years old. She developed extensive retinitis proliferans with partial blindness without clinical evidence of kidney disease. She had myocardial infarction six years ago, and now is in good general health.

Cases 57 and 58. Both 28 years of age with diabetes for many years; both developed retinopathy with blindness. They met in the Texas School for the Blind, Austin, and married. She has no evidence of kidney disease. He, with evidence of Kimmelstiel-Wilson nephritis, died of uremia.

Cases 107 and 108, females. Identical twins, developed diabetes within a year at age five.

Case 172, female. At age nine, the patient developed diabetes without symptoms and was somewhat overweight, which is unusual. Within the first 12 months, she required only six units of protamine zinc insulin every other day; now, after a period of 18 months, she receives 40 units of lente per day.

Case 180, female. This previously mentioned patient was the only juvenile diabetic to develop apparent cerebral vascular accident with right hemiplegia.

Case 28, male. The patient had diabetes since age 15, and is now 38. When first seen, there was such a severe neuropathy of lower extremities that there was no sensation, and no recognition that there were three fractures of foot bones. Infected calluses of both feet were treated conservatively, but after four years, bilateral midcalf amputations were necessary. This is the only case in this series involving amputation in a juvenile diabetic.

Case 51, female. She has had diabetes of classic juvenile type since age three and was first seen at age eight. She had been hospitalized repeatedly for appearance of acetone in urine or insulin reactions. The family became panic-stricken by all emergencies, but when properly instructed as to the principles of diabetic care, the child has required no further hospitalizations or special laboratory tests, except daily urine tests done at home. She is now 16.

Outlook for the Future

Philosophically, morally, and medically, the problems and destiny of the diabetic child could be the subject for many discussions. Certainly, the child developing diabetes before insulin was discovered had a dismal outlook, but now he may be assured a favorable youth and early adult life. After this, his health and longevity depend upon the degree of his control of the disease and other factors not fully understood.

Many problems are now being studied by dedicated researchers; they may or may not be resolved. Of course it would be desirable to find ways of preventing diabetes, or, next in order, to prevent the total destruction of the islands of Langerhans in early diabetes. Insulin eventually may be synthesized, and it is to be hoped that it may be taken by mouth or that a longer-acting parenteral insulin may be developed.

Present treatment appears to handle carbohydrate metabolism rather well, but it is

to be hoped that the "x" factor in diabetes which produces the various complications might be isolated, and that its counteragent might be found.

Summary

Early observations are presented on a series of 181 cases of juvenile diabetes with onset under age 19; of these, 79 are male and 102 are female. The average age of onset is 9.39 years. The significant statistical value of these observations is limited inasmuch as the duration of diabetes in these patients averages only 12.5 years.

Comments are made on the "typical" onset of juvenile diabetes, methods of diagnosis, and treatment.

Brief data is given on 11 deaths in this series. There is a story in the life of every diabetic child, and several such representative situational problems are presented.

Addendum

1. Since compiling this series 13 additional patients have been seen. Their inclusion would not alter the impressions presented.

2. Case 7, female, age 31. The patient, who had had diabetes 18 years, had cerebral thrombosis with right hemiplegia. She had partial recovery and died in six months from another "stroke."

Dr. Rippy, 3622 Fairmount, Dallas 19.

World of Tomorrow

Thirty years from now we will be living in the intersolar age, beyond the nuclear age and the interplanetary age. Travel to the moon will be rather routine with a permanent base established there. Astronauts will have gone to Mars.

These are the predictions of University of Texas Engineering Dean John J. McKetta, Ph.D., who spoke recently at a meeting of about 600 Houston area high school students at Clear Creek High School.

His vision of the future also included these predictions: The average life span will rise to 85. Men will develop electronic thinking caps, and a synthetic myelin will be used to coat brain cells to catalyze thinking power.

A preventive and cure for cancer will be found. Artificial hearts, lungs, and other parts of the body will be used. New cells or parts of bodies will be grown.

Automobiles will be driven automatically by electric eyes. Gasoline in automobiles will be replaced by fuel cells.

New sources of energy will supplement petroleum. Pint-size reactors powered by uranium will be shipped around the world, bringing electricity to remote and underdeveloped areas. Thermonuclear reactions will be harnessed, providing inexhaustible power. Huge parabolic mirrors will be dotted with solar cells to catch the sun's powerful rays; the cells will convert the rays to energy.

Such words as magnetohydrodynamics, thermoionic electricity, plasma jet, stellarator energy, and polyoxyglycols will be as common as today's plastics, television, synthetic fibers, antibiotics, and penicillin.

Perinatal Study Techniques
With Case Report

CASE REPORTS

IN THE REVIEW of charts in our hospital, it was found that during the latter part of the second stage of labor while preparations for delivery were being made, during the process of delivery itself, and from the time of delivery until the infant was handed to the pediatrician, a period of time existed in which data was sparse or totally absent with regard to the clinical information on the fetus and the newborn infant.[3] To rectify this situation, Desmond and others devised a plan whereby the infant could be followed from birth through the first six hours after delivery by a team of observers, clinically monitoring the vital signs of the newborn infant.[3] More recently, obstetricians and pediatricians at Baylor University College of Medicine, through the financial support of The John A. Hartford Foundation, Inc., have formed a perinatal study team, hoping to combine intrapartum fetal studies with neonatal studies. It is the purpose of this paper to describe the perinatal study techniques and to report an illustrative case.

Subjects; Methods of Study

The infant described in this study was delivered and cared for on the clinic service of Jefferson Davis Hospital, a city-county charity hospital in Houston. The study team included obstetricians, pediatricians, and nurses.

Each study morning, a patient in early labor is selected from the labor ward. She preferably has had an incidental and/or coincidental, antepartum and/or intrapartum complication of pregnancy. With her consent, she is transferred to the perinatal study room which is equipped with a delivery set-up, an Isolette,* and the necessary equipment for continuous electronic monitoring of the fetal electrocardiogram and heart rate, amniotic fluid pressure, and neonatal electrocardiogram, heart rate, respiratory rate, and temperature. A sterile vaginal examination is performed with notations being made of cervical effacement and dilatation, fetal presentation, position and station, and the status of the membranes. If the membranes are still intact, they are artificially ruptured if it is safe to do so at that time. A no. PE 320 polyethylene tube is then inserted inside the uterine cavity through a Drew-Smythe, hard metal, curved catheter which has been inserted alongside the presenting fetal part. The polyethylene catheter is then connected to the multi-channeled recording instrument† by way of a Statham pressure transducer, thereby allowing continuous electronic monitoring of the intra-amniotic fluid pressure throughout labor. Dr. Edward H. Hon's scalp clip electrode is then affixed to the presenting part of the fetus and connected to the recording instrument, thereby allowing continuous monitoring of the fetal electrocardiogram and heart rate throughout labor.

Clinical, non-electronic, intermittent monitoring of maternal blood pressure, pulse rate, respiratory rate, cervical effacement and dilatation, and position and station of the presenting fetal part is performed and recorded throughout labor. Other important observations such as maternal position, breath-holding, pushing or straining, and

*Air Shields Company, Hatboro, Pennsylvania.

†The Physiograph, manufactured by the E. & M. Instrument Co., Houston.

Lack of data between clinical intrapartum studies of the fetus and clinical studies of the neonate prompted obstetricians and pediatricians at this institution to initiate a joint effort to obtain more meaningful data from perinatal studies.

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type, amount, and time of medications are all recorded.

At the time of delivery, other important observations are noted and recorded such as type, amount, and time of anesthesia; time and duration of fundal pressure; time and duration of forceps application, traction and removal; time of episiotomy; time of delivery of the presenting part; time and duration of suctioning; time of total delivery; and time of cord clamping. As soon as the chest is delivered, an explorer electrode is placed over the precordial area of the infant so that continuous electronic monitoring of the neonatal electrocardiogram and heart rate can continue almost uninterrupted from the fetal into the neonatal period. Immediately following total delivery, the neonate is placed on the mother's abdomen where monitoring is continued during cord clamping and cutting and during the first ten minutes of life. During these first ten minutes, in addition to the continuous electronic monitoring of the neonatal electrocardiogram and heart rate, the respiratory rate is clinically counted and recorded at one-minute intervals. The Apgar score[1] is determined and recorded at one-minute intervals. Other important neonatal observations are made and recorded such as position, activity, micturition, defecation, flaring of alae nasi, grunting, and retraction. At ten minutes of age, the neonate is transferred to the Isolette. A small rectal probe‡ is inserted and the temperature is recorded. Thereafter, the neonate's electrocardiogram, heart rate, respiratory rate, and temperature are electronically monitored at 15-minute intervals along with clinical observations of color, cry, activity, micturition, defecation, and so on for periods varying from 6 to 24 hours.

The recordings and observations are then analyzed in great detail and graphs are made of the fetal and neonatal heart rates, the neonatal respiratory rates, and the neonatal temperatures, with important clinical observations such as activity, micturition, defecation, and cry, written in at their times of occurrence (Fig. 1).

Case Report

A 33-year-old gravida 10, para 9 patient was admitted to Jefferson Davis Hospital at 40-weeks' stage of gestation in active labor following an uneventful antepartum course. Her past medical and obstetrical histories were negative for complications. Physical examination on admission revealed no abnormal findings. The single fetus was in a cephalic presentation at station 0, the cervix was 4 cm. dilated with 80 per cent effacement and the membranes were intact. The fetal heart rate was 144 beats per minute by fetoscope. Admission was five hours prior to delivery and two hours following the onset of labor. She was given 100 mg. of Demerol HCl and 0.4 mg. of atropine SO_4 intramuscularly shortly after admission. One hour and 15 minutes prior to delivery, because of secondary uterine inertia, an amniotomy was performed and considerable meconium staining of the amniotic fluid was noted. Immediate auscultation of the fetal heart tones revealed a fetal bradycardia of 80 beats per minute between contractions. With the diagnosis of fetal distress, the patient was transferred to the perinatal study room where the previously described study methods were begun. The fetal heart rate was found to be 165 beats per minute, the fetal bradycardia having disappeared. There were no further episodes of bradycardia throughout the remainder of her labor. There was a gap of only 20 seconds between the end of the fetal heart rate tracing and the beginning of the neonatal heart rate tracing (Fig. 2). Delivery was spontaneous under caudal anesthesia, the anesthetic having been started 22 minutes prior to delivery.

‡Tele-Thermometer Probe, #402, Yellow Springs Co., Yellow Springs, Ohio.

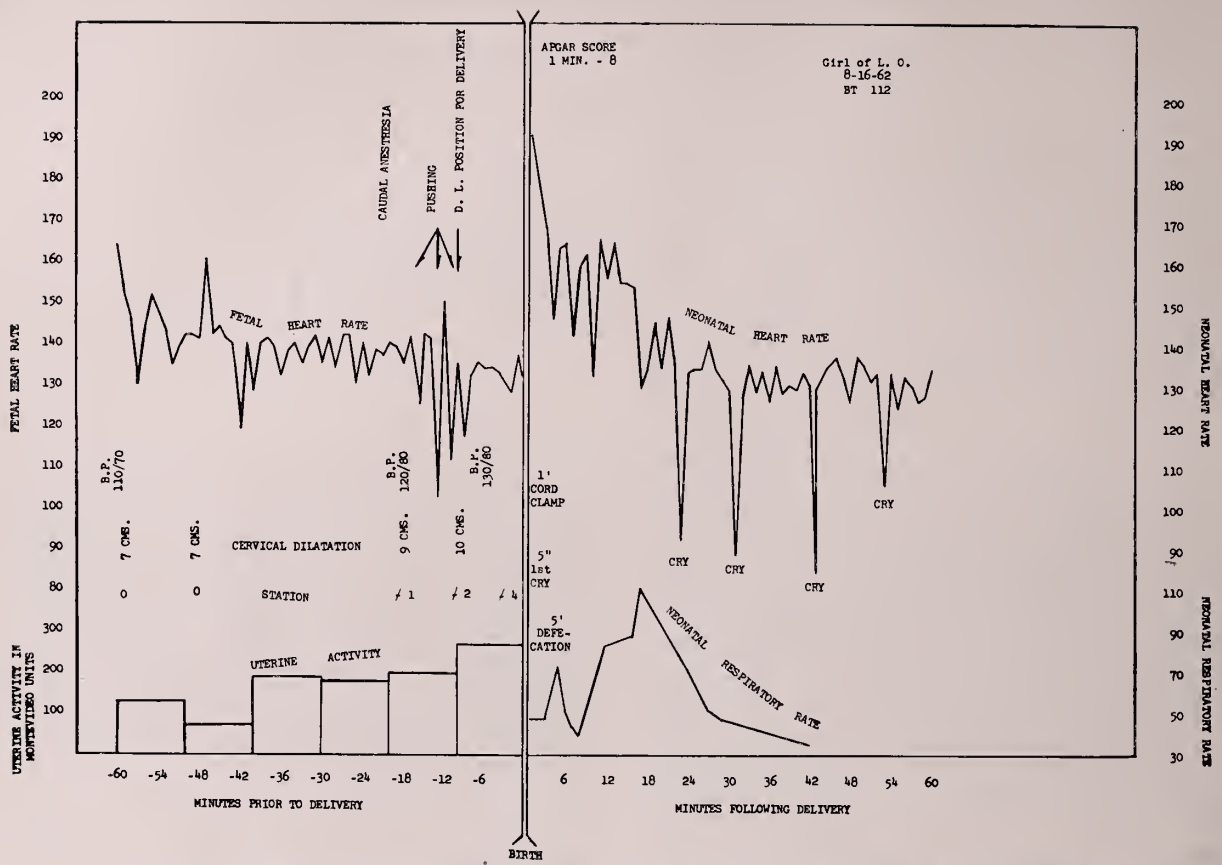

Fig. 1. Graph of fetal and neonatal heart rates, neonatal respiratory rates, and uterine activity. Uterine activity has been measured in Montevideo Units,[2] that is, intensity of contractions (mm. of mercury) times frequency of contractions (number of contractions per ten minutes).

Five seconds following delivery of a 6 lb., 10 oz. female infant, the first cry was noted. The cord was clamped at one minute of age. The one-minute Apgar score was 7, consisting of 2 points for heart rate, 2 points for respiratory rate, 2 points for responsiveness, 1 point for tone, and none for color.

At two minutes of age, the muscle tone was good, giving the infant an Apgar score of 8. The color had not yet improved.

At four minutes of age, there was an outburst of crying followed by a rapid increase in respiratory rate from 46 to 74 respirations per minute. This was shortly followed by the passage of a meconium stool. At this time, the Apgar score remained at 8 as the color was still not pink, although the infant had normal respirations. Thereafter, the color improved. The heart rate was fluctuating (varying between 130 and 170) but not at any time approaching the peak rate of 194 achieved at one minute of age.

Between seven and eight minutes of age, the infant was noted to have increased tone and this per-

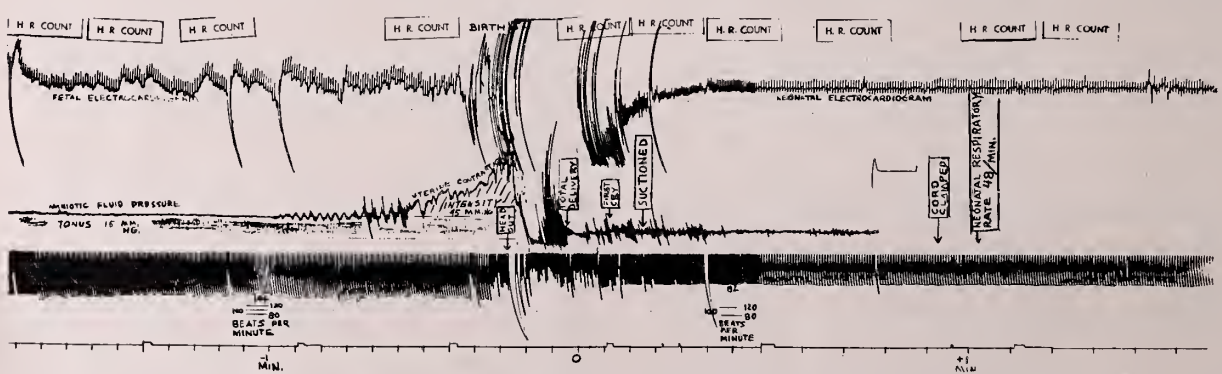

Fig. 2. Tracing of continuous fetal electrocardiogram, fetal heart rate, neonatal heart rate, and amniotic fluid pressure. A "gap" of only 20 seconds between the end of the fetal and the start of the neonatal tracings is illustrated.

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sisted during the first ten minutes of life. Apart from the episodes of bradycardia (86-104) with crying, the heart rate steadied between 130-140 beats per minute. The respiratory rate increased gradually in the first 17 minutes after birth to reach a peak of 112 breaths per minute and then the rate slowed over the next 30 minutes.

Discussion

The foregoing case illustrates how pediatricians and obstetricians at Baylor University College of Medicine have combined intrapartum fetal studies with neonatal studies in hope of obtaining more meaningful data during the perinatal period. Our next aim is to record electronically more of the maternal variables which affect the well-being of the fetus and to obtain biochemical studies upon the mother and the neonate to allow a more scientific appraisal of the well-being of the fetus and the neonate. It is also our aim to obtain long-term neurological follow-ups on the infants studied by the perinatal study team to determine if there may be some cause and effect relationship between events occurring during labor, delivery, and the immediate neonatal period and the future neurological and psychological development of the child. In this fashion pediatricians and obstetricians working together may make fetal and neonatal studies much more meaningful.

In closing, it may be noted that the authors have chosen their best tracing for the foregoing case report. The interval between the end of the fetal tracing and the beginning of the neonatal tracing has varied between 20 to 45 seconds during spontaneous deliveries with cephalic presentations. During forceps application, traction, and removal, there was much problematical electrical interference which disturbed the quality of the tracings. The same holds true during the periods of necessary handling and manipulation of the infant during assisted breech delivery. The tracings came through intermittently, however, allowing some idea of the fetal heart rate intermittently rather than continuously. This problematical interference will probably be overcome in the future by better methodology and instrumentation.

Summary

1. The lack of data in the interval between clinical intrapartum studies of the fetus and the clinical studies of the neonate has been discussed.

2. An attempt to obtain meaningful data during this interval through the combined efforts of pediatricians and obstetricians working as a perinatal study team has been discussed.

3. A case report illustrating perinatal study techniques at Baylor University College of Medicine has been presented.

Dr. Edward H. Hon, Dr. Stanley T. Lee, and Dr. Warwick Newman assisted in the guidance of the authors during the beginning stages of the intrapartum studies. The pediatricians assisting were Dr. Murdina M. Desmond and Dr. A. J. Rudolph.

*The paper was originally presented to the Texas Association of Obstetricians and Gynecologists on March 2, 1963, in San Antonio.

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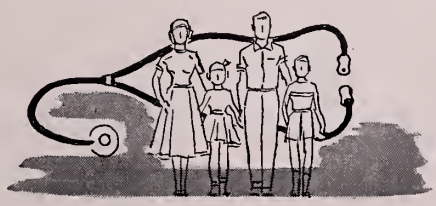

Near Fatal Reaction To Tetanus Antitoxin Skin Test

CASE REPORTS

FATAL ANAPHYLACTIC shock has been reported following the administration of horse serum both intradermally and intramuscularly.[1, 5, 10] Opinions as to the unreliability of tetanus antitoxin skin tests, especially after injury, have been discussed.[3] Brochures accompanying biologicals differ over the type of test, dilution of testing antigen, and the quantity of material to be used. Excellent plans[7] have been outlined providing safe and effective tetanus prophylaxis, but in spite of these, severe allergic reactions continue to occur.

Electrocardiographic studies[4, 6, 8, 9] have been made on numerous occasions during anaphylactic shock resulting from the administration of tetanus antitoxin horse serum, but none have been reported associated with a serious reaction due to an intradermal skin test. Since this information might be of value in future understanding of this problem and in the specific treatment and recovery of such a patient, this case report is presented.

Case Report

L. M., a well developed and well nourished 23-year-old male was seen on Dec. 27, 1962. One day prior to hospitalization, he had received a slight contusion and abrasion of the left side of the forehead, following an injury from the handle of an automobile jack. For three days prior to being seen, he had been taking an over-the-counter vermifuge which had not caused unusual reactions. He had previously received tetanus antitoxin without trouble. His family physician gave him a skin test dose of approximately 0.10 cc. of a diluted tetanus antitoxin horse serum. Approximately 45 seconds later, the patient collapsed. He received immediate treatment for shock, including several doses of epinephrine hydrochloride 1:1000 subcutaneously, 2 cc. of Benadryl intramuscularly, and an undetermined but sufficient injection of a steroid parenterally. There was only temporary improvement and the patient was transferred by ambulance to the authors' offices. When first seen by them, he appeared restless, apprehensive, and transiently semi-comatose. Breathing was rapid and labored with obvious wheezing. Temperature was 99.2 F., respirations 40, pulse approximately 150 per minute with the volume varying in intensity and fullness. Blood pressure was 80/60 mm. of mercury. His skin was warm to the touch and had a purplish erythematous color. Massive areas of elevated urticaria over the trunk and legs were seen. The lips and mucous membranes were dry; the tongue and lips were slightly swollen. His face was flushed and his eyelids were also swollen. Lungs revealed wheezing throughout the entire fields with sonorous and sibilant rales heard diffusely. No murmurs could be heard and there was no apparent evidence of cardiac enlargement by physical examination. The abdomen was soft but slightly distended; the liver and spleen were not palpable. Extremities showed some edema of the ankles and feet which may have been part of the generalized angio-edema. A large urticarial wheal was seen on the left forearm where the skin test had been performed. The patient was immediately given additional small doses of epinephrine hydrochloride 1:1000, varying from 0.10 to 0.30 cc. subcutaneously. An infusion was started immediately, consisting of 500 cc. of 5 per cent glucose to which was added 40 units of ACTH and 7½ grains of aminophylline. The fluid was given at the rate of 50 drops per minute. Since the patient was able to take oral medication when first seen, he was given 8 mg. of Medrol and 50 mg. of Pyribenzamine. Oxygen was given for a brief period but was discontinued when the patient began to vomit. Vomiting soon became uncontrollable and his general condition continued to deteriorate in spite of the emergency medications. He was then promptly admitted to the hospital, two and one-half hours after the initial skin test was performed. There he was placed in an oxygen tent with Alevaire mist, and intravenous medication was continued as

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The authors report a near fatal case of cardiac arrhythmia associated with anaphylactic shock following an intradermal skin test for sensitivity to horse serum. The patient had a history of extreme sensitivity to horses and deer.

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well as the administration of epinephrine hydrochloride 1:1000 at intervals of 15 to 30 minutes. In addition, he was given 25 mg. of Wyamine sulfate and 100 mg. of Solu-Cortef into the intravenous tubing. In spite of these measures, his blood pressure remained between 70/40 and 90/60; respirations were 40; his pulse remained rapid, between 160 and 200 per minute; and he showed little signs of improvement. Four hours after the initial shock reaction, a roentgenogram of the chest was taken revealing no abnormalities and an electrocardiogram was done. The latter (Fig. 1) revealed a marked regular tachycardia, appearing to be supraventricular in origin. It was felt that the increased rate was prolonging the hypotension. He was then given rapid digitalization along with intravenous Lanoxin. Levophed was added to the intravenous fluid according to the usual methods of administering this drug. By this time, six hours after the onset of his reaction, the patient had not voided and it was necessary to catheterize him. Within four hours after the reception of digitalization and Levophed, his blood pressure had risen to 100/64, the pulse slowed to 110 per minute, and respirations were 30. The peculiar violescent color of the skin and much of the urticaria and angio-edema had subsided. Twelve hours later, another electrocardiogram (Fig. 2) was taken. The rate was 100, the T waves were inverted in T_2 and T_3 and DVF, with the presence of S_1, S_2, and S_3, indicating some degree of right heart strain.

Twenty-four hours later the patient felt well; his pulse was 80 and all of his symptoms had abated with the exception of a slight urticaria. The initial skin test was about the size of a silver dollar. He was maintained for a while on oral steroids, ephedrine sulfate, and antihistamines. A follow-up complete examination, including an electrocardiogram (Fig. 3) done on Feb. 4, 1963, was found to be normal.

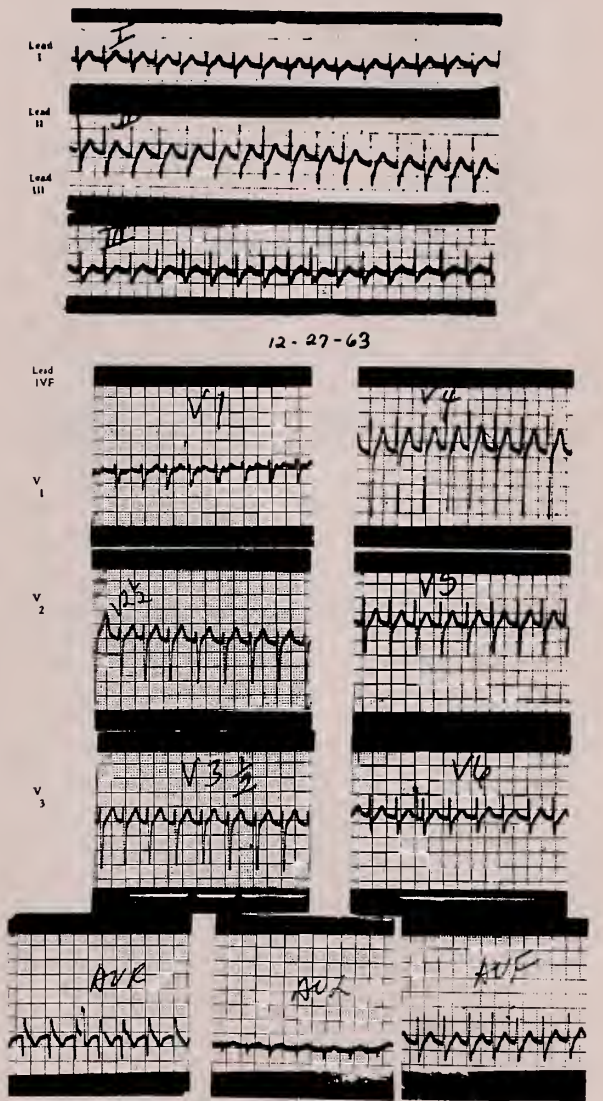

Fig. 1. Electrocardiogram taken four hours after initial skin test showing tachycardia.

Discussion

This is a case of a young male who had received an intramuscular injection of tetanus antitoxin serum in the distant past without any untoward reaction. However, he did

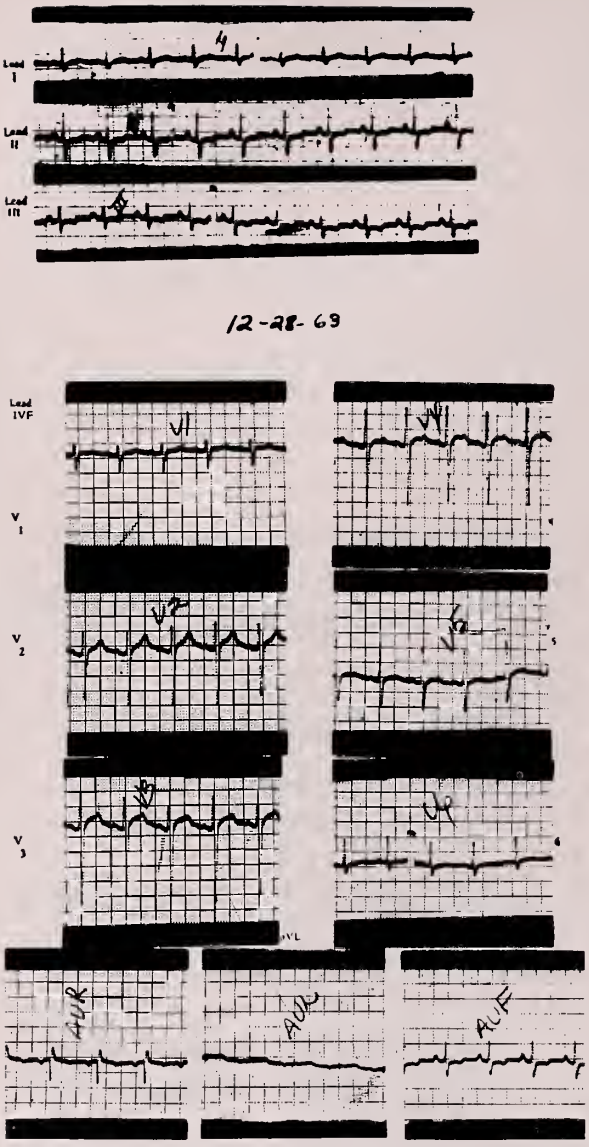

Fig. 2. Electrocardiogram taken 12 hours after initial skin test and digitalization.

present an unusual history of sensitivity to horses and deer. Contact with these animals or their danders caused violent reactions including vomiting and acute urticarial swelling. No symptoms resulted from contact with dogs, cats, or cattle. He had no history of hay fever, asthma, or drug allergy. The information concerning his sensitivity to horses and deer was not given to the physician who performed the initial skin test which caused the reaction reported here. Since it is well known, as Kaufman[7] warns, that individuals who are allergic to horses may react to horse serum, it is doubtful that this patient would have been skin-tested with horse serum had the physician

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known these facts. Because of the foregoing illness, the patient was clearly advised to avoid this exposure again, and was given 0.5 cc. of tetanus toxoid as his initial immunization procedure and instructed to complete this active immunization. He received a warning card indicating his hypersensitivity and was advised to carry this card in his billfold at all times. Criep and Riley[2] pointed out that anaphylactic shock in animals and man is known to be associated with severe and pronounced cardiac changes. These consist of variation in the frequency of the heart beat, disturbances in conduction, and signs of myocardial damage. Such findings have been reported following the administration of intramuscular antitoxin and this is a report of a case with marked arrhythmia following a simple skin test with horse serum.

Summary

A near fatal case of cardiac arrhythmia associated with anaphylactic shock following an intradermal skin test for sensitivity to horse serum is reported in a patient having a history of extreme sensitivity to horses and deer. Evidence of myocardial complications were substantiated by electrocardiographic findings. The abrupt termination of the tachycardia and hypotension was finally accomplished by the use of digitalis and Levophed intravenously.

A warning is hereby presented to avoid administration of horse serum in any form to patients with a history of sensitivity to horses.

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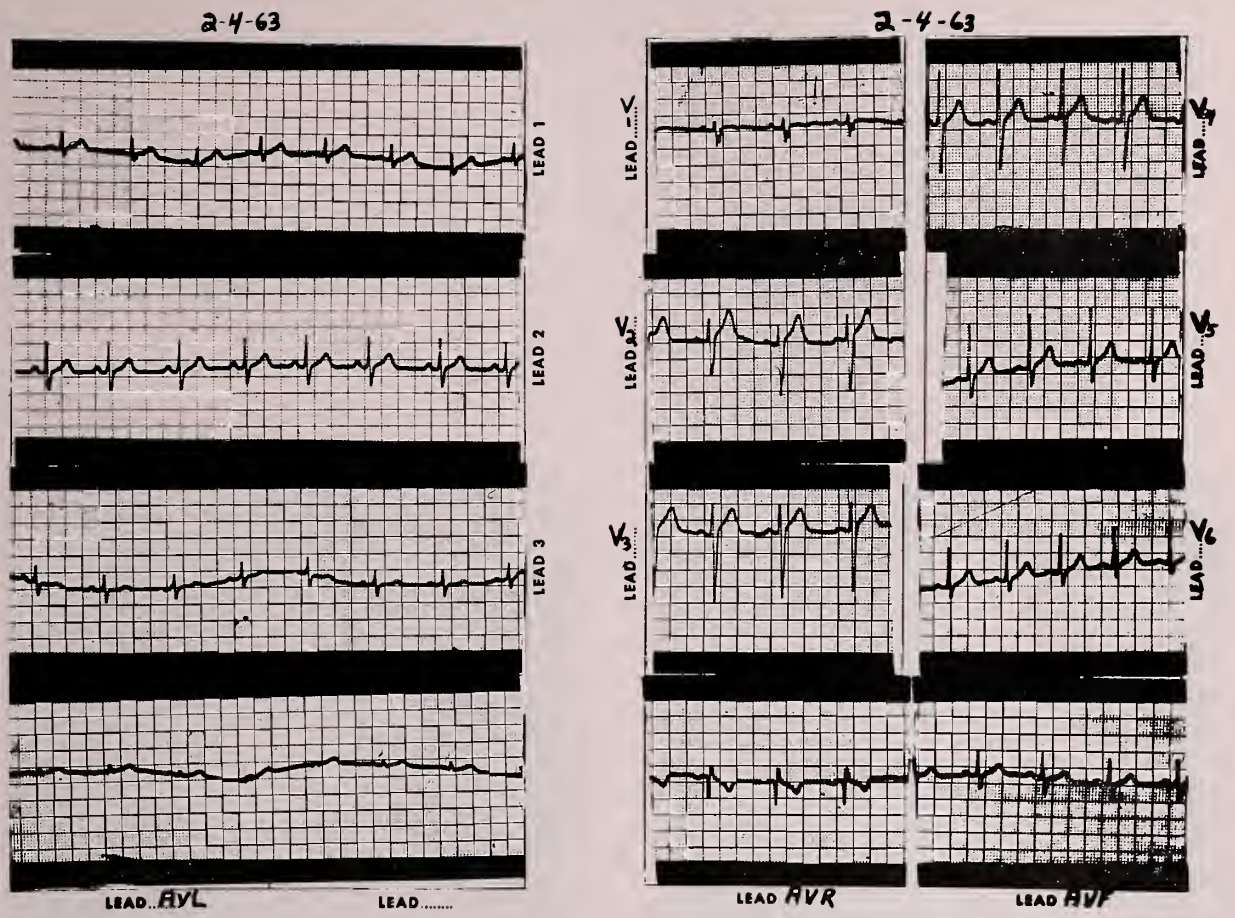

Fig. 3. Electrocardiogram one month after acute anaphylactic shock showing a normal rhythm.

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Dr. Meyer, 1702 Nix Professional Building, San Antonio.

COMMENT

This article is timely because of the current emphasis on tetanus toxoid immunization. It again emphasizes the dangers of horse serum administration, and it deals with the treatment of anaphylactic shock, an uncommon but catastrophic event requiring prompt and correct treatment.

Electrocardiographic changes occur in anaphylaxis. In this case, the administration of large doses of epinephrine prior to the first tracing, and digitalis and Levophed before the second, may possibly have contributed to some of the changes in the electrocardiogram.

Restoration of the blood pressure was the most difficult therapeutic problem. Epinephrine, which was not effective in this case, is still the drug of first choice. Other methods to restore the blood pressure, Levophed and digitalis (perhaps by correcting the tachycardia), resulted in success. Advertisements and enthusiastic detail men have made physicians conscious of the use of cortisone and related drugs in treatment of allergies. As inferred by the authors in this case, the adrenal steroids do have a place in the management of anaphylaxis, but epinephrine and vasopressor drugs are immediate acting and life saving.

—T. S. Painter, Jr., M. D.,
Austin.

Development of Solitary Gastric Polyp In Postgastrectomy Stomach Remnant

Report of Two Cases

CASE REPORTS

PROBLEMS FOLLOWING subtotal gastrectomy have become of increasing interest. The mechanism of the dumping syndrome, postgastrectomy anemia, and nutritional problems have been studied extensively. The following case reports describe the finding of a benign polyp in the gastric remnant of two patients, 8 and 12 years after partial gastric resection. Polyps had not been present before the gastric resection in either patient. The association of benign gastric polyps and adenocarcinoma in the stomach has been reported.[2, 7, 12] However, the relationship of gastric polyps to the pathogenesis of malignancy is difficult to determine.[1] These case reports indicate a third variable, the development of benign polyps in the postoperative gastric remnant following surgery for both benign and malignant disease.

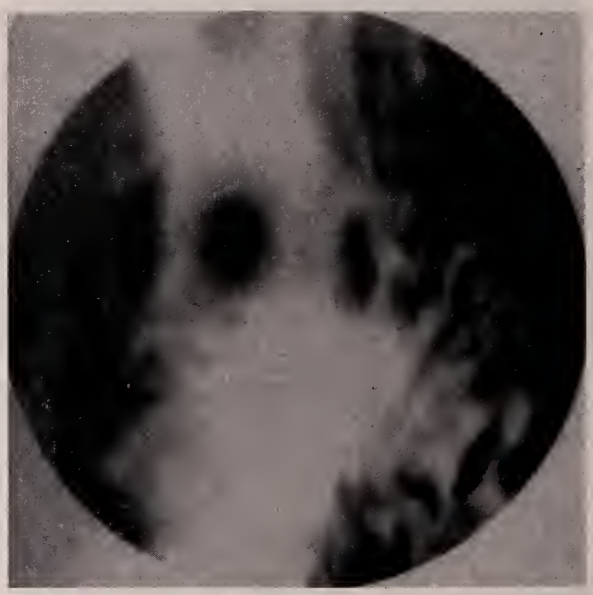

Fig. 1. Radiolucency shows a benign polyp in gastric remnant above the gastrojejunal stoma eight years after subtotal gastrectomy for adenocarcinoma.

Case Reports

Case 1.—A 47-year-old man, seen in 1946, complained of anorexia, a full feeling in the epigastrium, and constant, dull epigastric pain. He was unwilling to eat because of early satiety. There had been no weight loss. Physical examination was normal. The hemogram, urinalysis, and chest roentgenogram were normal. Tests for occult blood in stools were positive. Narrowing and irregularity at the pyloric antrum were seen in roentgen studies of the stomach. The duodenum was deformed in its first portion. There was 40 per cent retention of barium in the stomach after six hours. The fasting free hydrochloric acid was 6 mEq. per liter with a peak of 22 mEq. per liter at 30 minutes after histamine. An erosion measuring 0.5 cm. was seen through the gastroscope with a rugal fold in the middle third of the stomach. The clinical impression was duodenal ulcer with obstruction. After six weeks of medical therapy, the symptoms of anorexia and epigastric distress persisted. Weight loss became evident. Abdominal laparotomy was done. A grade 2 adenocarcinoma of the antral portion of the stomach was resected. No apparent spread to lymph nodes was found.

Five years later the patient was seen because of an eight pound weight loss. Roentgenograms of the intestinal tract were normal. Three years later the patient again presented himself because of malaise and anorexia for the preceding three months. A radiolucency measuring 2 cm. in diameter was seen in the roentgen examination of the stomach (Fig. 1). A 2 cm. polyp on a thick stalk was seen through the gastroscope in the stomach remnant. The mucosa was intact and the polyp had a benign appearance. Local excision of this polyp was accomplished and it was benign histologically (Fig. 2).

Case 2.—A 40-year-old housewife was seen first in February, 1942. She presented a ten-year history of upper abdominal pain occurring shortly after meals and persisting for one to two hours. There were 52 mEq. per liter of hydrochloric acid in the gastric analysis. Roentgen examination of the stomach was normal. A superficial gastritis was seen at gastroscopy. She was treated with diet,

After partial gastric resection, two patients developed solitary benign polyps in stomach remnants. One patient had survived gastric carcinoma for eight years, the second had had resection for peptic ulcer disease. Continued observation of patients with partial gastrectomy may be indicated.

TRAVIS BRIDWELL, M.D.
ROBERT J. PRIEST, M.D.

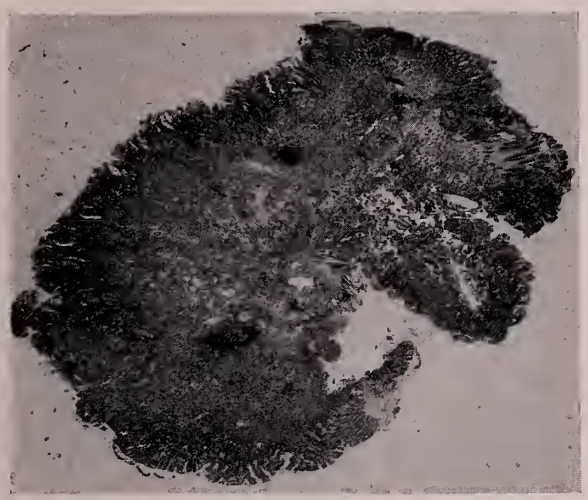

Fig. 2. Low power view of gastric polyp, case 1. Scattered nodular accumulations of lymphocytes are present. Hematoxylin and eosin stain, X 11.

antacids, sedatives, and vitamins. Two years later, the gastroscopic appearance of an area 3 cm. in diameter on the lesser curvature toward the posterior wall was that of an obliteration of the rugae with an irregular mucosa. An early malignancy was suspected and a partial gastrectomy was done. The pathologic examination revealed acute gastritis.

A fasting free hydrochloric acid of 8 mEq. per liter was found in the gastric analysis following the operation. The patient continued to have epigastric pain. Superficial gastritis was seen in a progress gastroscopy 18 months after the previous examination. Roentgen studies of the stomach were normal on three occasions during the following two years. In November, 1947, the patient was operated on for an intestinal obstruction. Multiple adhesions were released and a vagotomy was done. No free hydrochloric acid was found after histamine stimulation two years later. Between 1947 and 1956, roentgenographic studies of the stomach were done at two-year intervals. In the latter year, a radiolucent defect, 2 cm. in diameter, was seen in the stomach remnant (Fig. 3). The patient was hospitalized and a large villous polyp removed by local excision of the gastric wall. The polyp was benign histologically (Fig. 4). Postoperative roentgen studies

Fig. 3. This 2 cm. polypoid defect was seen in the stomach remnant 12 years after partial gastrectomy.

of the stomach have been normal and there has been no return of the polyp.

The appearance of new polyps in post-gastrectomy stomach remnants adds another

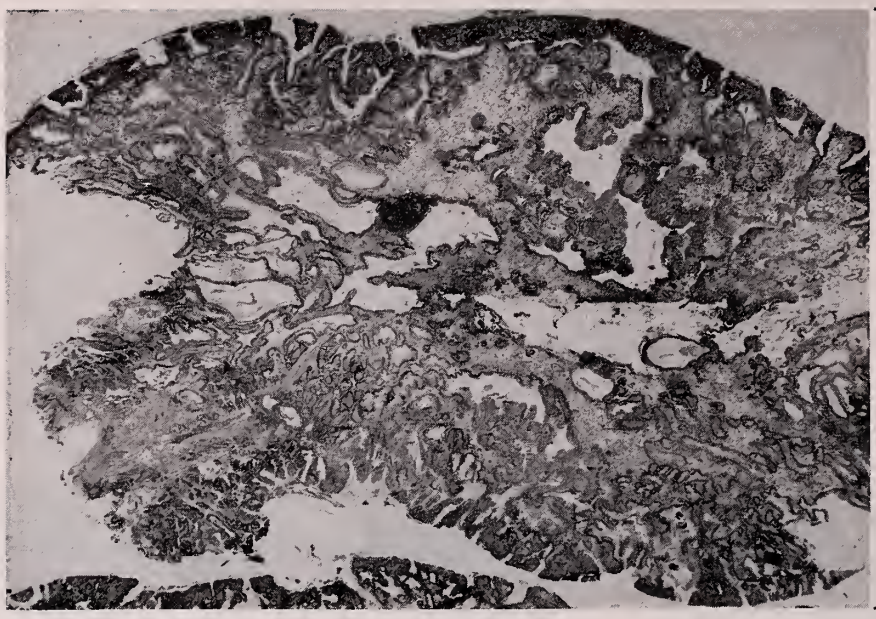

Fig. 4. Law pawer view af gastric palyp, case. 2. Resected stalk is visible in lower left carner. Nate large vascular channels. Hematoxylin and eosin stain, X 13.

aspect to the follow-up of these patients. To the physiologic "misbehavior of an abbreviated stomach remnant following partial gastrectomy"[4] is added the dilemma of the management of gastric polyps. The management of gastric polyps remains an unsolved problem because of the diversity of opinion regarding their premalignant potential. Benign gastric polyps have been removed preceding the development of malignancy in the stomach.[2,3,10] The size and number of polyps, presence or absence of gastric acidity, and gastroscopic appearance of stomach mucosa have not been reliable criteria to differentiate benign from malignant polypoid lesions.[1] Many gastric polyps have been observed for years and have not become malignant.[2] Huppler and associates[6] considered roentgen evaluation the most important technique and found that 20 per cent of 300 postoperative patients had malignant disease.

The dilemma of the gastric polyp could be resolved by excision of all such tumors diagnosed.[3,11] However, a conservative management with careful follow-up by roentgen and gastroscopic examinations of solitary polyps has been satisfactory to other observers.[1,5,12] Certainly, one would hesitate to permit a polyp to remain in a stomach remnant when carcinoma had been resected previously from the same stomach, as in case 1. The question remains whether the polyp developing in the stomach remnant of the second patient could have been observed safely over a longer period of time. The gastrectomy had been performed for benign ulcer disease. Yet, the subsequent vagotomy had resulted in a histamine-refractory achlorhydria.

Partial gastrectomy has been widely employed in this country a relatively few years. There is now a large group of such patients available who have had partial gastrectomy for both benign and malignant disease. Fifteen-year survivals following surgery of gastric carcinoma are being reported.[8,9] Periodic roentgen evaluation of postgastrectomy stomach remnants may be indicated following resection for both benign and malignant disease.

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John Hunter
The Father of Modern Surgery

HOWARD R. DUDGEON, JR., M.D.

HISTORICAL ARTICLES

RIGHTLY CONSIDERED, the father of modern surgery is John Hunter, who in 1785 performed a successful operation on a ruptured popliteal aneurysm. He had noticed that most varieties of stag each year shed their antlers and grew a new pair. He ligated one of the common carotid arteries of a stag and a few days later found that the antler on the side of the ligated artery had stopped growing and was cold. On reexamination a week later, the antler again had resumed its normal growth and was warm. He killed the stag, reopened the wound in the neck, and found that some of the smaller arterial branches above and below the ligature were greatly enlarged. From this simple observation of the altered arteries, he deduced the entire principle of collateral capillary circulation.

About four months after this discovery, a 45-year-old coachman, identified in Dr. Hunter's records as "A. B.," was admitted to St. George's Hospital in the late stages of a popliteal aneurysm. This condition was one to which coachmen were prone because their knees were continually flexed. Most cases were treated by amputation or by ligating the popliteal artery proximal to and distal to the aneurysm and then by opening the aneurysm and removing the clot; amputation was usually necessary later anyway.

Hunter decided to ligate above and below the aneurysm and not disturb the aneurysm itself. The coachman recovered and returned to his duties in six weeks. The patient later died of an unrelated cause and Dr. Hunter had to retrieve from the grave the leg for his museum. In later operations, only the popliteal artery proximal to the aneurysm was ligated. All four of his operations were successful. One patient, also a coachman, lived 50 years following surgery.

Mysteries of Nature Fascinated Him

John Hunter, of a Norman-Scotch line, was born in February, 1728, at Long Calderwood, Lanarkshire, near Glasgow. His parents were of the minor gentry and owned a rocky 79-acre estate which produced little, but required much hard work. John was the youngest of ten children, seven of whom died before their parents. Such afflictions spared few houses. Of all the children born between Land's End and John o'Groat's, not even two thirds survived their first year and not even three-fourths lived to be six. Smallpox was the great killer of the young. The "spitting sickness" or consumption killed many adults.

John attended school from the age of 6 to 13. He was not a very good student and was more interested in the mysteries of nature than in the study of Latin, which he never mastered. His mother took him out of school when he was 13. He could barely write coherent English and had acquired a permanent distaste for academic halls.

His brother, William Hunter, ten years older than John, had gone to London after studying medicine as a medical apprentice to William Cullen. In London he studied under William Smellie, "the master of British midwifery," and had succeeded in establishing a flourishing obstetrical practice.

The "Father of Modern Surgery" had little formal education, was prone to temperamental outbursts, but was an inspiring teacher and a man of many interests and infinite curiosity.

Dr. Howard R. Dudgeon, Jr., a Waco surgeon, originally presented this paper to the Texas Surgical Society.

After the death of his father from bladder stone and of an older brother from consumption, John decided to join his brother William, a handsome, cultivated gentleman, in London. John was his brother's opposite; he had no wig to cover his coarse, carroty hair and no conversational niceties to soften the harshness of his Lowland speech. Unlettered, naive, and boisterous, he reeked of the stable and the field, and his natural pugnacity was intensified by the anti-Scotticism around him in London.

William Hunter had started an anatomy school at his home in the Great Piazza. He installed John as a dissector and thus started him on his road to fame. John was a frequent visitor to Covent Garden, an area noted for its coffee houses, taverns, Drury Lane Theater, brothels, and criminals. Here he often met the "sack-'em-up men."

Although private dissection by an anatomical school had been legal since the dissolution of the Barber-Surgeons Company in 1745, there was still no legal way of obtaining enough bodies. One way to obtain bodies was by claiming the body of an executed criminal, but there were not enough of these. To supply the demand for bodies, there arose the group called the "sack-'em-up men" or "resurrectionists." They were expert at robbing a grave and would do this the night after burial before the body could putrefy. English law at that time ruled that a body belonged to no one, so the grave robbers would steal only the body—but not the grave clothes, for which they could be hung. The clothes were considered private property. Finally, after some of the grave robbers began to murder to obtain bodies, Parliament passed the Anatomy Act in 1832 which insured an adequate supply of bodies.

John studied under William Cheselden, the most famous lithotomist of his time. Cheselden, a surgeon who operated very rapidly, could remove most bladder stones in two to three minutes. He removed one in 30 seconds. All of this was done without an anesthetic. He also studied under Percival Pott of St. Bartholomew's Hospital, noted for the description of Pott's fracture and Pott's disease of the vertebra.

William and John Hunter did much original and valuable work in anatomy. They demonstrated the connection between the testicle, epididymis, and vas deferens by injecting mercury into the vas deferens and then dissecting the structures. The Hunters also charted the anatomy and most of the functions of the lymphatic system. Pott and the Hunters did much to clarify the surgical anatomy and the origin of inguinal hernia. One of their greatest works was "The Anatomy of the Gravid Uterus" which required 23 years' preparation and the dissection of about 400 bodies. John successfully skin-grafted an ulcer on a child's ankle. After serving in the army, he urged that most gunshot wounds be treated without attempting to remove the bullet.

John Hunter's four masterpieces included "The Natural History of the Human Teeth," 1771; "A Treatise on the Venereal Disease," 1786; "Observations on Certain Parts of the Animal Oeconomy," 1786; and his "Treatise on the Blood Inflammation and Gun Shot Wounds," 1794.[1]

St. George's Admits Him

In the summer of 1754, John Hunter was admitted as a surgeon's pupil at St. George's Hospital with which his name would be later associated. Here he spent the rest of his professional life and made this hospital famous. He worked here as a student, intern, and staff surgeon, and he died here following a stroke at a staff meeting.

The Hunter star was on the rise. William became the man-midwife to the queen of England and John later became surgeon to the king.

John was more than a surgeon. He taught anatomy and helped his dental friends with their dental surgery. (Dentists at that time were even lower in the social scale than surgeons.) The Hunters also had learned something about embalming. John Sheldon, one of their dissectors, decided to embalm the body of his mistress, an Oxford Street prostitute who had died of consumption. Sheldon liked the result so much that he kept the body in his bedroom until he married and

his wife objected. Martin Van Butchell, a dentist friend of John's, had his wife embalmed and kept her in a glass case in his waiting room for many years. Both Sheldon's mistress and Mrs. Van Butchell now stand side by side in the Royal College of Surgeons' Museum in London. The bodies of course have greatly deteriorated.

John Hunter's most famous experiment was when he infected himself with pus from a body he thought to be suffering from gonorrhea. Unfortunately, the man had both lues and gonorrhea. John described the resulting chancre—since known as the hunterian chancre. This error led to the confusion and belief that all venereal diseases were really only one disease. John put off his marriage until he had observed all the symptoms of his disease and then cured himself with mercury, he thought. After this he married Ann Home, daughter of a doctor friend.

An Inspiring Teacher

John Hunter was now at the height of his professional success. He became a Fellow of the Royal Society. He published many original papers on anatomy, physiology, and surgery. He made a great deal of money, but spent it on his home and his famous museum. As a member of the surgical staff of St. George's Hospital, he lectured and trained future surgeons. He was a poor speaker but an inspiring teacher. He changed the theory and the base of English surgery and thus laid the foundation for modern surgery. He was appointed surgeon-extraordinary to King George III. His pupils became the leading English surgeons for the next 50 to 60 years. The Hunter brothers also influenced American medicine; many American doctors, beginning with William Shippen, Jr., studied at their school.

As John grew older he had more attacks of severe chest pain, usually brought on by anger or argument. This may have been due to a luetic disease of the heart. He often said, "My life is at the mercy of any rogue who chooses to provoke me."[2] On Oct. 16, 1793, at a board meeting of St. George's Hospital, following a disagreement with another doctor, he suffered an attack of chest pain, became unconscious, and died several hours later.

In 1859, 66 years after his death, his bones were transferred to Westminster Abbey where they lie in the north nave beside the tomb of Ben Jonson.

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The Stethoscope: A Relatively New Invention

The stethoscope, a tool which has become almost a part of the physician, is less than 150 years old. Invented by a French physician, René Laennec, in 1816, the first model of the instrument was merely a rolled-up piece of paper.

Dr. Laennec experimented with several materials and finally used a wooden tube of a given length and bore for his final model. Since then the stethoscope has changed so much that only the idea remains, says Crawford F. Barnett, Jr., in an article in the January issue of *The New Physician.*

It is remarkable that while the stethoscope is a comparatively new invention, the instrument is one of the first to be used to aid the physician in diagnosis.

NEWS

Record Crowd of 382 Attends Austin Hospital Accreditation Institute

MOST FREQUENT criticisms of hospitals noted in accreditation surveys concern poor attendance at meetings, poorly organized medical staff, not having the recommended 20 per cent autopsies performed, poor medical records and lack of documented clinical review, inadequate tissue reports, and incomplete staff by-laws.

A record crowd of 382 persons attending a hospital accreditation institute in Austin on Feb. 15 heard these criticisms presented by Dr. Kenneth B. Babcock, Chicago, director of the Joint Commission on Accreditation of Hospitals. The institute was conducted by Texas Hospital Association and sponsored by Texas Medical Association and Private Clinics and Hospitals Association.

Dr. Babcock added that hospitals are almost never rejected for accreditation for only one reason; there are usually 30.

He said that delineation of medical staff privileges was probably the most unpalatable problem hospitals deal with, but hospitals must decide who can do surgery and "how much can Dr. X or Y do?"

He added, "The Joint Commission feels it cannot set up definite rules in Chicago as to whether Dr. X can take out a gallbladder. There are no quantitative measures as to when a man is qualified to do surgery. We think a man should be judged by his credentials by his own staff which is cognizant of his ability."

Dr. Babcock said that the Joint Commission criticized hospitals that had categories preventing general practitioners from doing general surgery. "You must not take away privileges by the label to which a man belongs. There are excellent general practitioners capable of doing some operations. They must be assessed as individuals."

Dr. F. Leigh Crozier, Houston, president of the Texas Hospital Association, told hospital officials what to expect on an accreditation survey. Although Dr. Crozier emphasized that the hospital accreditation surveyor is interested in all phases of the hospital, some of his questions may concern trustee meetings, disaster plans, regulations concerning attendance of medical staff meetings, surgical privileges and how they are granted, consultations, medical staff organization and documented evidence of periodic reviews, emergency room care and necessary facilities for this department (including books), anesthesiology and operating room records, the nursery, kitchen, and even the store room areas.

Any hospital that is eligible may apply for accreditation, Dr. Crozier said, and eligibility means that the hospital must have been in operation one year, have 25 beds, be listed by the American Hospital Association, and present evidence of a sincere desire by all groups concerned to maintain the standards.

Another conference participant, Dr. A. W. Bronwell of Lubbock, said, "The most important part of accreditation is medical records. It is the medical record which mirrors the quality of patient care."

Dr. Bronwell said in his experience in the Dry Run Survey program most of the deficiencies, varying from a nurse's note to a

physician's signature, were found in medical records.

He disapproved of check lists that some hospitals use in recording patient history and said, "Positive statements regarding a physical examination give superior records to a check list."

Philip R. Overton, General Counsel for the Texas Medical Association, pointed out some of the legal pitfalls in medical records. All medical records should be preserved for two years, he said, and in cases involving minors, the records should be kept until two years after the minor attains 20 years of age.

"Consent is a vital part of medical records," he noted. In regard to blanket consents, a number of recent decisions say the consent must be "informed." This means that the physician should advise the patient of the hazards he might be facing, he said.

Both Mr. Overton and Mr. W. D. Norwood, who presented the trustee viewpoint, noted that the board of trustees assumes complete legal and moral responsibility for a hospital's policies and conduct, and this legal responsibility cannot be limited by delegation of authority to the medical staff.

The board of trustees "is responsible for gearing the hospital to community needs, securing funds, and bringing about high professional standards," Mr. Norwood observed, and although authority must be delegated, there are several methods of evaluating the hospital's effectiveness: periodic reports from the chief of staff, joint conference committee meetings, or hospital record department reports.

Mr. C. Jack Price of Dallas listed the safety measures required by the hospital accreditation association: The basic requirement is providing a safe and clean place for patient care. This means that the physical plant must have facilities for the segregation of patients, facilities for isolation, adequate space for each bed, and adequate emergency lights, gas, and water.

However, "any hospital operated on minimum requirements either of the Joint Commission or of the State has a precarious plan," he cautioned. "These should be looked upon as foundations on which to build."

Dr. Hugh F. Rives of Jacksonville said the underlying problem in getting the small hospital accredited is getting specialized personnel. At present there are 267 hospitals in Texas that have 25 to 75 beds, he said. Twenty-two per cent of these hospitals are accredited.

In small towns, pathologists, radiologists, dietitians, medical records librarians, and registered technologists are hard to find, but some small hospitals have a plan of sharing these specialized personnel. In others, they are called in on a periodic consultant basis.

Dr. G. V. Brindley, Jr., of Temple analyzed important committees in accreditation. "You should be certain of the definite, demonstrable need for each committee. Members should be carefully selected, and their objects, responsibilities, and authorities carefully delineated. Alterations in scope, program, even personnel should be periodically reviewed, altered, and even changed."

Dr. Brindley noted that in small hospitals, many committees can be combined under two or more general committees.

Texas TB Association To Meet March 20-21

The annual meeting of the Texas Tuberculosis Association will be held March 20-21 in Austin at the Commodore Perry Hotel.

Theme of the meeting is "The TB Problem Cluster." Simultaneous programs have been announced for the group's medical community health sections.

Both sections will convene together to open the meeting Friday morning and hear Mrs. Marty Mann of New York speak on "Alcoholism and the TB Patient."

Speakers and papers to be presented Friday before the medical section include Drs. Robert E. Carr, Fort Worth, and Richard W. Ernst, Dallas, "Changing Concepts of Bronchiectasis"; Dighton Rowan, Ph.D., Dallas, "Some Observations on Respiratory Viruses in a Virology Laboratory"; Dr. R. G. Loudon, Dallas, "Droplet Production in the Respiratory Tract"; Drs. W. Cone Johnson, John R. Calderly, and John G. Weg, San Antonio, "Concomitant Hypothyroidism and Alveolar Hypoventilation"; Drs. J. B. Des Rochers and Walter A. Carlson, San Antonio, "Psychotic Reactions Due to Antituberculosis Drugs"; and Dr. J. E. Miller, Dallas, "Professor Miller's X-Ray Quiz."

Saturday program items for the medical section include Drs. D. L. Paulson and Harold C. Urschel, Jr., Dallas, "Pulmonary Complications of Esophageal Reflux"; Dr. Monte B. Miller, Dr. Johnson, and Dr. Lionel L. Harkleroad, Lackland Air Force Base, "Pul-

monary Diffusion in Eosinophilic Granuloma of the Lung"; Drs. Katharine H. K. Hsu, Nita I. Carreon, and Daniel E. Jenkins, and Fongee Jeu, R.N., Houston, "Epidemiologic Significance of Large and Small Tuberculin Reactions"; Drs. Dionisio S. J. Salvador, Harlingen, Ellison F. White, Enrique Trapp, and James M. Schless, Austin, "Evaluation of Ethionamide Hepatotoxicity with Serial Liver Biopsies"; and Dr. James B. Seaman, Tyler, "Operative Pneumoperitoneum with Pulmonary Resection."

At 12:30 p. m. Friday the Texas Thoracic Society will meet for luncheon and hold its annual business meeting. Friday evening a banquet has been planned for all those attending the Texas Tuberculosis Association meeting. Dr. Robert R. Shaw of Dallas will speak on "Medicine in Afghanistan." At noon Saturday, the board of directors will meet for luncheon and a business session.

Pamphlets

Drug Addiction.—A history of narcotic drug use and control and the legal, medical, and social complexities created by drug abuse in the United States is contained in a new book, "Narcotic Drug Addiction Problems," record of a National Mental Health Institute symposium on the subject.

The book, Public Health Service Publication No. 1050, is for sale by the Superintendent of Documents ($1.75), U. S. Government Printing Office, Washington, D. C., 20402.

Heart Failure.—A new Public Health Service publication, "Congestive Heart Failure: A Guide for the Patient," describes in nontechnical terms the causes, process, treatment, and management of congestive heart failure. A completely revised edition of "Understanding the Management of Congestive Heart Failure" presents similar material for nurses and other health agency personnel.

Single copies of both booklets are available without charge, from the U. S. Public Health Service, Washington 25, D.C.

Early Immunization.—In spite of dramatic reductions in the number of cases of diphtheria, whooping cough, smallpox, poliomyelitis, and tetanus, these diseases still take their toll. A comparatively simple step, however, offers protection against these communicable diseases—vaccination of children "right from the start."

"Right from the Start," by Judy Graves, is the title of a new Public Affairs Pamphlet and a related film produced by the Public Affairs Committee on a grant from the U. S. Children's Bureau and made in cooperation with the Conference of State and Territorial Directors of Public Health Education. Both the pamphlet and the film stress the importance of early immunization of children. The pamphlet is available for 25 cents a copy from the Public Affairs Committee, 22 East 38th Street, New York, N. Y. 10016; information about purchase or rental of the film, which is in color and runs 22½ minutes, is also available from the Committee.

Heart Disease.—"The Food You Eat and Heart Disease" is the subject of Public Health Service Publication No. 537 (Revised 1963). It is available at 10 cents per copy or $7.50 per 100 copies from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402.

Cancer.—"Cancer of the Skin," "Cancer of the Uterus" and "Cancer of the Breast" are informative folders available at 5 cents per copy or $3.25 per 100 from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. They are prepared by the Research Information Branch, National Cancer Institute, National Institutes of Health, Bethesda, Md.

Southwest Allergy Forum Scheduled for April 5-7

The Southwest Allergy Forum will be held in Hot Springs, Ark., on April 5-7.

Texans who will participate in the program and their addresses are Dr. William Sellars, Dallas, "Dust and Mold Emulsion Therapy in Severely Asthmatic Children: A Double Blind Controlled Study"; Dr. Harris Hosen, Port Arthur, "Provocative Skin Testing"; Dr. John P. McGovern, Houston, and Dr. Paul Petit, Beaumont, panelists, "Use of Alginate in Allergy Extract Injections"; Dr. Bernard Fein, San Antonio, "Side Effects and Complications Associated with Corticosteroid Administration in Allergy"; Dr. William Grater, Dallas, and Dr. Ben Fisch, Tyler, panelists," Clinical Evaluation of Drugs Used in Allergy"; Dr. Homer Prince, Waco, and Dr. Warren Raymer, Houston, panelists, "Repository and Allpyral Therapy."

Additional information about the meeting may be obtained by writing to Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark.

April 5-7 Date Set By Texas Surgical Society

The Texas Surgical Society will meet April 5-7 at the Holiday Inn in Waco.

The scientific program will take place in the Westview Playland auditorium next to Holiday Inn. Program items include the following:

On Monday: "Multiple Cancers of the Upper Respiratory Passage and Lung," Drs. A. J. Ballantyne, Howard T. Barkley, and Harry Preble, Houston; "Bronchogenic Carcinoma in Young People," Col. Daniel C. Campbell, Jr., Lackland Air Force Base; "Tracheostomy, A Major Consideration," Dr. R. Maurice Hood, Austin; "Subarachnoid Hemorrhage: Significance and Treatment," Dr. James L. Poppen, Boston; "Hyperparathyroidism; an Ubiquitous Diagnosis," Col. Oakley K. Park, Lackland Air Force Base; "Hyperfunction of the Parathyroids," Drs. Albert O. Singleton, Jr., and O. T. Kirksey, Galveston.

"Bone Tumors of the Femur," Dr. Frank F. Parrish, Houston; "Combined Surgery and Chemotherapy for Malignant Melanomas of the Extremities: Six Year Report," Dr. John S. Stehlin, Jr., Houston; "Alvar Nunez Cabeza de Vaca—Soldier, Trader, Medical Man and Explorer—First European Visitor to Texas," Dr. Samuel R. Snodgrass, Galveston; "Current Status of Transplantation of Tissue in Man," Drs. Manning B. Shannon and Don Patman, Dallas; "Use of Polypropylene Mesh in the Repair of Direct Inguinal Hernias," Dr. F. Cowgill Usher, Houston.

On Tuesday: "Zenker's Diverticula Associated with Duodenal Ulcer," Dr. Thomas A. McDowell, Marlin; "Granuloma of the Ileocolic Area of the Bowel," Dr. Porter M. Bailes, Tyler; "Surgical Management of Splenic Injuries," Drs. William B. Gordon, Jr., A. L. Shirkey, and Arthur C. Beall, Jr., Houston; "Progressive Exophthalmos: Surgical Treatment and Results," Dr. Poppen; "Antipolar Efferent Jejunal Segment—A Surgical Approach to the Dumping Syndrome," Dr. B. V. Williams, Houston; "Abnormal Renal Mobility as a Cause of Renal Ischemia—Technique of Diagnosis," Dr. John R. Derrick, Galveston.

"I'll *give* you the diet pills; I don't trust you near the soda fountain in the drugstore."

Americans Invest $8 Billion In Health Insurance

Nearly $8,000,000,000 was invested by 140,000,000 Americans in health insurance premiums in 1962, according to the annual survey by the Social Security Administration. This represented more than one-third of America's total medical care budget of $22,000,000,000 in 1962.

While this budget total was up 5.8 per cent over 1961, health insurance payments increased 13.4 per cent, and direct payments for medical care rose only 2.2 per cent. The proportion of the total medical care dollar covered by health insurance benefits has increased steadily from a level of 8.2 per cent in 1948 to 31.1 per cent in 1962.

Per capita payments for health insurance amounted to $41.17 in 1962, up $4.33 from the 1961 level of $36.84 per capita. Total medical care expenditures per capita rose $4.93, from $114.51 in 1961 to $119.44 in 1962.

Blue Cross and Blue Shield Plans accounted for 41.2 per cent of the nearly $8,000,000,000 total premium income, while 52.4 per cent was paid to insurance companies and 6.4 per cent to "independent" plans. The Blue Cross-Blue Shield share of total health insurance income has remained relatively stable at about 41 per cent ever since 1950.

Nursing Homes and Dangerous Drugs

THE VAST rapid growth of rest and nursing homes has emphasized the question of how, or if, these institutions are to handle drugs.

The Texas Dangerous Drug Law became effective Aug. 9, 1959. Up until that time, Texas law enforcement officers found it almost impossible to curb the illicit possession of drugs which, if not properly dispensed and taken under the direction of a physician, could cause serious injury and even death. Many youngsters were becoming addicted to drugs such as barbiturates and amphetamines. Hypnotic drugs were being sold to youngsters through illicit channels and were creating serious problems. Thus, the Texas Dangerous Drug Law was enacted to restrict severely those persons or firms entitled to possession of such drugs so that the illicit channels of drug distribution could be better controlled.

The law lists those drugs considered to be "dangerous." The following are among the more significant: barbiturates, amphetamines, hypnotics, and any drugs bearing the legend, "Caution: Federal law prohibits dispensing without prescription."

Section 4 of the statute lists those persons who are entitled to possess these dangerous drugs in Texas:

1. A pharmacy, drug store, dispensary, apothecary shop, or prescription laboratory, duly registered with the Texas Board of Pharmacy;
2. Medical practitioners;
3. Persons who procure dangerous drugs for disposition by or under the supervision of pharmacists, or for the purpose of lawful research, teaching, or testing, and not for resale;
4. Hospitals which procure dangerous drugs for lawful administration by practitioners;
5. Officers or employees of federal, state or local law enforcement agencies;
6. Manufacturers and wholesalers;
7. Carriers and warehousemen.

The Texas State Board of Pharmacy has interpreted Section 4 with respect to hospitals to mean those institutions that are licensed by the State Department of Health as hospitals in the State of Texas. *Of course, this does not include nursing or rest homes.*

All persons or organizations not listed may not possess a drug considered a "dangerous drug" in Texas without becoming subject to the penalties of the statute. These include rest homes and nursing homes as well as the individuals responsible for these institutions. Thus, there is no statutory authorization for rest homes to possess dangerous drugs, and any possession subjects the owners or operators to the statutory penalties.

Even if the statute permitted rest homes or nursing homes to possess dangerous drugs, which of course it does not, the Texas Pharmacy Law would prohibit those in charge of these establishments from dispensing such dangerous drugs unless they are registered pharmacists. While hospitals are specifically exempt from the operation of the Pharmacy Law with respect to dispensing to inpatients, nursing and rest homes are not accorded such an exemption. Thus, Section 8 of the Texas Pharmacy Law would prohibit distributing or dispensing and reads in pertinent part as follows:

> It shall be unlawful for any person who is not a registered pharmacist under the provisions of this Act [Pharmacy Law] to compound, mix, manufacture, combine, prepare, label, sell or distribute at retail or wholesale any drugs or medicines. . . .

While nursing and rest homes are not entitled to possess dangerous drugs, many homes have found this to be no particular problem because of their close liaison with pharmacists in their communities. A physician's prescription for a particular patient or guest is taken to the local pharmacist where it is properly filled and returned to the home. In this way, it is unnecessary for the home to have drugs on its premises, and the request of the attending physician is scrupulously adhered to by virtue of the pharmacist dispensing, compounding or in otherwise filling the prescription for the patient or guest. This is by far the most expeditious and, to my way of thinking, practical solution to the problems of obtaining drugs for patients or guests of nursing and rest homes.

Another method by which the larger homes have complied with both the Dangerous Drug and Pharmacy Laws is to apply to the Texas State Board of Pharmacy for a store permit allowing the rest home itself to operate a

pharmacy. This is not practical for smaller homes because of the requirements of the Pharmacy Law as to operation of such a retail pharmaceutical outlet. This, of course, would require the hiring of a registered pharmacist.

Finally, there is some confusion as to what should be done with dangerous drugs left by patients in a rest or nursing home. It is inadvisable and in fact is illegal to allow relatives of deceased patients or guests to have pharmaceuticals prescribed for the deceased person. It is said that a prescription dies with the patient, thus disposal of such drugs becomes a problem. The Texas Dangerous Drug Law provides for disposal of dangerous drugs by employees of the Texas State Board of Pharmacy. The Board's investigators will come to the homes pursuant to a request by the home for disposal of drugs. It is suggested that these drugs be accumulated and kept in a safe, locked container, or space until such disposal may be effected. Request for a disposal of dangerous drugs may be made to Mr. J. H. Arnette, executive secretary, Texas State Board of Pharmacy, Littlefield Building, Austin. Disposal of narcotic drugs must be by the Bureau of Narcotics, and requests directed to Mr. Ernest Gentry, Bureau of Narcotics, Dallas, will facilitate disposal of such narcotic preparations.

—PHILIP R. OVERTON, LL.B., Austin.

Mental Health Planners Meet

The General Planning Committee for Comprehensive Statewide and Community Mental Health Planning met for the second time Feb. 7 in Austin. Eighty-two out of the 113 members on the Committee attended the meeting, and for the first time research data on state community resources, psychological data on patients, and sociological data on communities were available to the members.

In explaining the computer data presented at this meeting, Dr. Fred Crawford, Research Chief of the Office of Mental Health Planning, outlined the objectives the Committee would be concerned with: (1) treatment facilities available, (2) communities and needs, (3) state governmental and sociological structure, and (4) ratio of diagnostic categories existing now.

The next meeting of the General Planning Committee will be April 3-4 at the Driskill Hotel in Austin.

Personals

Dr. J. T. O'Banion who has practiced in Luling since 1938 retired on Jan. 1.

– o –

Dr. A. J. Gill, dean of Southwestern Medical School, received the Caldwell Award at the recent meeting of the Texas Society of Pathologists, Inc.

– o –

New officers of the Texas Society of Pathologists, Inc., are *Drs. Mervin H. Grossman,* Dallas, president; *John R. Rainey, Jr.,* Austin, president-elect; *Wilson Brown,* Houston, vice president; and *Vernie A. Stembridge,* Dallas, secretary-treasurer.

– o –

Drs. John P. McGraw of Houston and *Dean B. Jones* of San Antonio are new fellows of the American College of Radiology.

– o –

Dr. Charles LeMaistre of Dallas has been named to a five-member committee to direct the American Medical Association Education and Research Foundation's long range program of research on tobacco and health.

– o –

Dr. May Owen, Past President of Texas Medical Association, is reported recovering nicely from a recent heart attack. She is in All Saints Episcopal Hospital of Fort Worth.

Physicians, Surgeons To Meet April 3-4

The Association of American Physicians and Surgeons, Inc., will hold its interim meeting in Chicago April 3-4.

The meeting is to be devoted to business and will be held at the LaSalle Hotel.

Meet Set on Diseases In Nature Transmissible to Man

The 14th annual Southwestern Conference on Diseases in Nature Transmissible to Man will be held in Austin at the auditorium of the Texas State Department of Health, on April 2-3.

This meeting is open to all interested physicians, veterinarians, epidemiologists, public health officers, nurses, microbiologists, biologists, public health engineers, sanitarians, laboratory workers, and individuals in related fields.

Information may be obtained by writing J. V. Irons, Sc.D., director of laboratories, Texas State Department of Health, Austin.

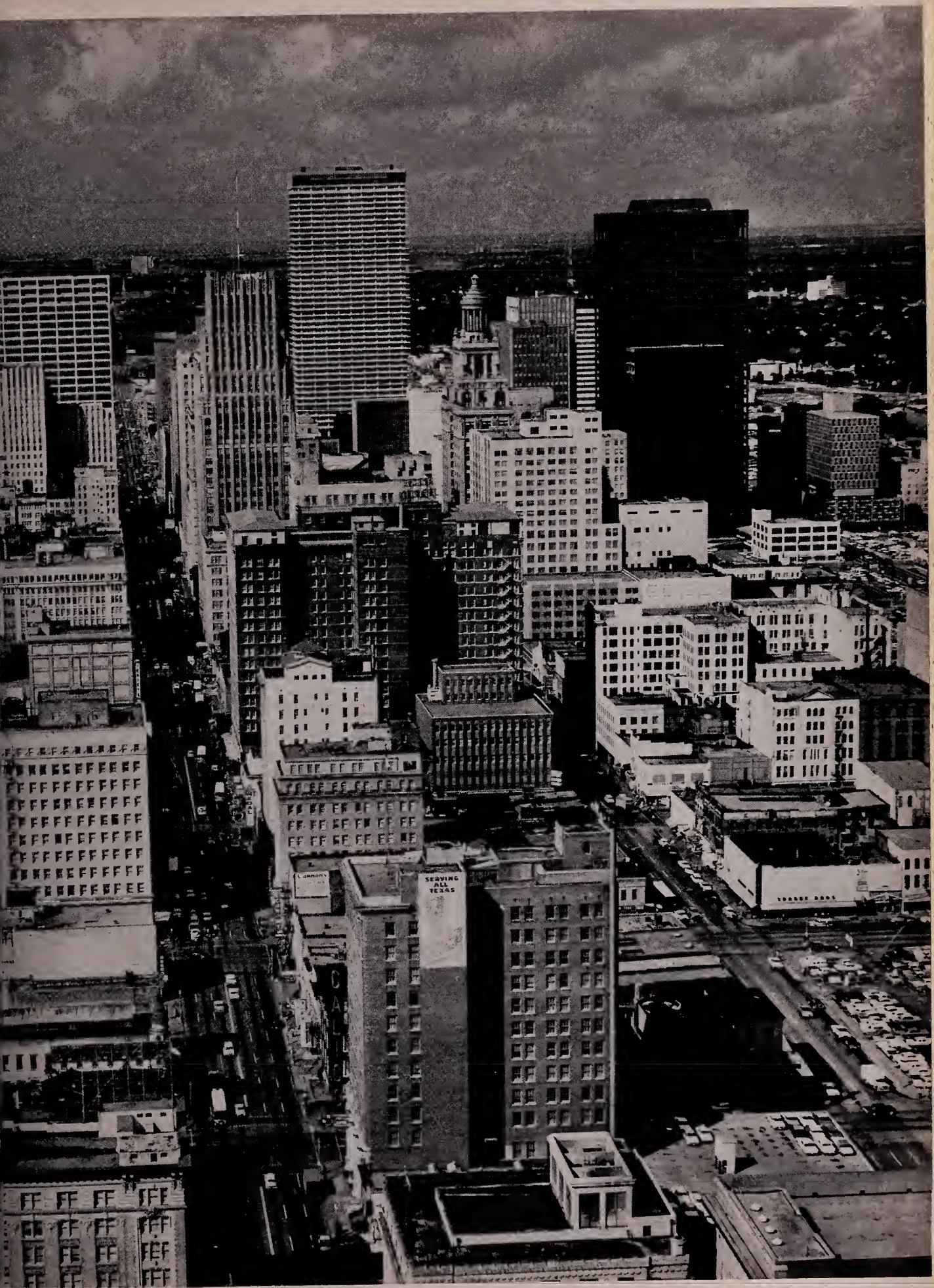
Space City Awaits TMA

HUGE, and still growing, describes Houston, site of the 97th Annual Session of Texas Medical Association April 23-26.

Any description of Houston tends to run to superlatives. The 4,000 physicians and guests expected for the 1964 meeting will find themselves in the largest city in the Southwest and the seventh largest in the United States. The port of Houston is the third largest in the nation in point of tonnage. The domed stadium, now under construction in Houston, will be the world's first air-conditioned stadium for baseball, football, and other sports events. The new 44-story Humble Oil and Refining Company building in Houston is the tallest building west of Chicago. Houston is the leading manufacturing center of the Southwest. The Houston Symphony is one of the country's finest orchestras. Houston is the nation's top-ranking distributing center for oilfield equipment and supplies. Houston is located in the largest cattle-raising county in the state. Houston's Texas Medical Center is destined to be one of the nation's greatest concentrations of medical institutions for research, training, and treatment.

Houston is now called Space City, U. S. A. Although it is only one of 16 National Aeronautics and Space Administration (NASA) centers in nine states, Houston is the center of research for manned space flight. As headquarters for the Manned Spacecraft Center, Houston is the home of the astronauts. A huge undertaking involving countless people (if taxpayers are included) is behind these world-famous space travelers. Their mission is to put a man on the moon. The Manned Spacecraft Center has had a fantastic effect on Houston. The estimated cost of the Center construction is now $150,000,000, including a $50,000,000 center for control of manned space-exploration probes. Part of the TMA Annual Session program will be a closed-circuit television presentation beamed from NASA headquarters.

Those attending the Annual Session will hear about Houston's port, phenomenal because it is man-made. A ship channel was dredged down Buffalo Bayou in the early 1900's, and on Nov. 10, 1914, President


SERVING
ALL
TEXAS

Woodrow Wilson opened it by pressing a button at the White House. Now, ships from all over the world dock here, and as many as 48 vessels carrying as much as 500,000 tons of cargo have squeezed into the channel in a single day.

A must for every visitor to Houston is a tour of the U. S. Battleship Texas. This famed warship of two world wars is permanently berthed at San Jacinto State Park, a few minutes' drive from Houston. A few steps away from the Texas is the tallest monument in the United States, the San Jacinto Monument, 570 feet high and commemorating the battle in which Sam Houston routed the Mexican forces, captured Santa Anna, and won freedom for Texas.

Houston was founded Aug. 30, 1836, four months after the battle of San Jacinto, and as all Texans know, was named after the hero of the battle, Gen. Sam Houston. Many Texans may have forgotten that Houston was one of the early capitals of the Republic of Texas.

Physicians and their families may enjoy visiting Houston's many recreational areas. The city has 150 municipal parks and playgrounds, four municipal golf courses, 37 municipally-operated swimming pools, and 86 tennis courts. A top-flight zoo is located in Hermann Park. At Lake Houston, about an hour's drive from downtown Houston, the Alexander Deussen Park attracts picnickers and fishermen.

For those who wish a 30-mile view of Houston, surrounding countryside, and the Gulf of Mexico, a trip to the observation deck on the 45th floor of the new Humble Building will suffice. For those who wish closer-range sightseeing, Gray Line Tours include visits to a wide variety of interesting attractions.

Houston is proud of its cultural offerings. The Houston Symphony is conducted by world-renowned Sir John Barbirolli. The Houston Museum of Fine Arts is directed by the eminent James Johnson Sweeney. Three theaters make Houston one of the nation's most active centers of the performing arts. Houston maintains its own ballet and opera companies.

Every visitor to Houston hears about the

A short drive from Houston is the San Jacinto Battlefield and two symbols Texans take pride in—the U. S. Battleship Texas and the San Jacinto Monument.

new domed stadium now under construction. It is scheduled for completion in time for the baseball season in 1965, and it will be the world's first air-conditioned stadium for baseball, football, and other sports events. It will be approximately 200 feet high, cost well over $20,000,000, and its dome is to be covered with a translucent plastic to permit sunlight to enter and grass to grow. The stadium is expected to seat 46,217 for baseball, 52,913 for football, and up to 66,000 for boxing. The seating arrangement can be changed from football to baseball in five minutes. Encompassing some 230 acres, the stadium grounds provide space for parking 25,000 cars.

During the Annual Session, physicians and their families will want to explore Houston's wide variety of eating places. Here in the heart of cattle-raising country, beef steak is a prime menu item. Seafood from the Gulf and salt water bays is another culinary specialty. And then there are a number of places that feature exotic dishes.

Among the eating places featuring steak are Bud Bigelow's Charcoal House at 7939 Westheimer; Dobbs House, 5138 Westheimer; Cellar Door with three locations, 6732 Stella Link Road, 3598 Old Spanish Trail, and 5930 Westheimer; Gaido's, 9200 S. Main; L-C Cafeteria, Main or Travis at Walker; and Look's "Sir-Loin" House, 6112 Westheimer.

Seafood is a specialty at the following: San Jacinto Inn, adjacent to the San Jacinto Battleground; Dobbs House, 5138 Westheimer; Gaido's, 9200 S. Main; and Massa's Oyster House, Capitol and Louisiana.

Exotic restaurants include Albert Gee's Poly-Asian, 9530 S. Main; Alfred's Restaurant and Delicatessen with two locations, Rice Blvd. near Rice Stadium and 9123 Stella Link Road; Del Monico's, 3925 Westheimer; Dobbs House "Luau," 5138 Westheimer; Maxim's Restaurant Francais, corner of Milam and Lamar; and Mediterranean Room, located in the Houston Airport Inn directly opposite the airport terminal.

Work of the medical profession in Houston is concentrated in the Texas Medical Center, a multiple-institution enterprise which co-

The Port of Houston is one of the nation's three greatest. It is connected with the sea lanes of the world by means of a 50-mile-long ship channel to the Gulf.

An aerial view of Texas Medical Center shows many new and impressive buildings, facilities for some of the nation's finest patient care, health education, and medical research.

ordinates health education, health research, and patient care. Located on a tract of land south of Hermann Park, the Center was organized in 1945. Today, it includes 16 institutions as well as numerous affiliated organizations.

In 1962, 72,000 patients were admitted to the various hospitals in the Medical Center. These patients spent a total of 563,636 days in the hospital. Outpatient clinic visits for the same period amounted to 327,000. Capital investment in all units of the Center exceeds $83,000,000. It is the policy of the Center to maintain a quality of teaching, research, and patient care that is competitive with the best in America.

Prior to the formal organization of the Texas Medical Center, Inc., in 1945, the original Hermann Hospital had been in operation for about 20 years. Arrangements had been completed with the University of Texas to locate a cancer and tumor research hospital in the Center and with Baylor University to move Baylor University College of Medicine there. In 1948, the College of Medicine moved into a new building in the Center and in 1949 the new Hermann Hospital was completed.

Between 1951 and 1955 facilities for the following institutions were completed and opened: Methodist Hospital, Arabia Temple Crippled Children's Clinic, Jesse H. Jones Library Building, Texas Children's Hospital, St. Luke's Episcopal Hospital, the University of Texas M. D. Anderson Hospital and Tumor Institute, and the University of Texas Dental Branch.

In 1959 and 1960, the Texas Institute for Rehabilitation and Research, the Houston Speech and Hearing Center, Houston State Psychiatric Institute, Texas Woman's University College of Nursing, and the Institute of Religion were completed.

In May of 1963 the Ben Taub General Hospital was officially opened, and on June 6, a large addition to Methodist Hospital was dedicated and opened.

In August, five additional operating rooms were opened at Hermann Hospital, and in

October a special care unit to accommodate 12 acutely ill patients was also opened. Several new buildings were added to Baylor University College of Medicine. These include the Anderson Basic Science Research Building, the Jesse Jones Clinical Research Building, and the Jewish Institute for Medical Research. The Rayzor Student Center and March Culmore Hall have been constructed. A garage has been acquired by the Center as well as additional parking area.

Plans are now under way for further development of physical plants, improved and expanded educational programs, and more comprehensive and intensive research undertakings.

Plans are being developed for the construction of dormitories for male students. Expansions are planned for M. D. Anderson Hospital and Tumor Institute, St. Luke's Episcopal, Texas Children's, and Methodist hospitals. The Houston Speech and Hearing Center is discussing expansion. Construction on a new City Department of Health Building will be begun in 1964.

Provision has been made for the establishment of the University of Texas Graduate School of Biomedical Sciences at the Texas Medical Center. This school will grant master's and doctor's degrees in several areas of the life sciences including biology, biochemistry, nuclear medicine, and bio-mathematics. The Texas Commission on Higher Education has recently authorized Texas Woman's University to offer a master's degree in nursing with the emphasis of the program being placed at the Texas Medical Center.

Baylor University College of Medicine plans to establish an M.D.-Ph.D. program which would enable highly qualified medical students to carry on a graduate program which would result in the ultimate awarding of both the M. D. and the Ph.D. degrees to those who complete the course of study.

During 1963, the principal research and educational institutions in the Medical Center carried on research and training grants and training programs totaling approximately $10,000,000. Some of the important research projects include the cardiovascular center at Methodist Hospital which is soon to be enlarged, the Lipid Research Center located in the new Research Buildings at Baylor University College of Medicine, the clinical cancer chemotherapy program at M. D. Anderson Hospital, the Chronic Illness Research Center at the Texas Institute for Rehabilitation and Research, the leukemia virus research program at Baylor University College of Medicine, and the radiation therapy and radiation biology program at M. D. Anderson Hospital.

The Texas Medical Center, like the City of Houston, has had a rapid, dynamic growth in the last few years. Physicians at Annual Session who come from outside the Houston area will not want to miss seeing this great Center.

Houston Clinical Pathologists Announce Seminar

The Houston Society of Clinical Pathologists has announced its 1964 seminar, to be conducted by Dr. Lewis B. Woolner, pathologist, Mayo Clinic, and professor of pathology, Mayo Foundation Graduate School of Medicine, and Dr. Harold G. Jacobson, chief of Division of Diagnostic Radiology, Montefiore Hospital, Bronx, N. Y. The subject will be a general seminar: "Respiratory System, Gastrointestinal System, Genitourinary System and Bone."

The meeting will be from 9 a.m. until mid-afternoon Saturday, April 25, at the Jesse Jones Medical Library Auditorium, Texas Medical Center, and will be followed by a reception. A limited number of slide sets, protocols, and prints of x-rays are available at $10 per set from Dr. William T. Hill, St. Luke's Episcopal Hospital, Texas Medical Center, Houston.

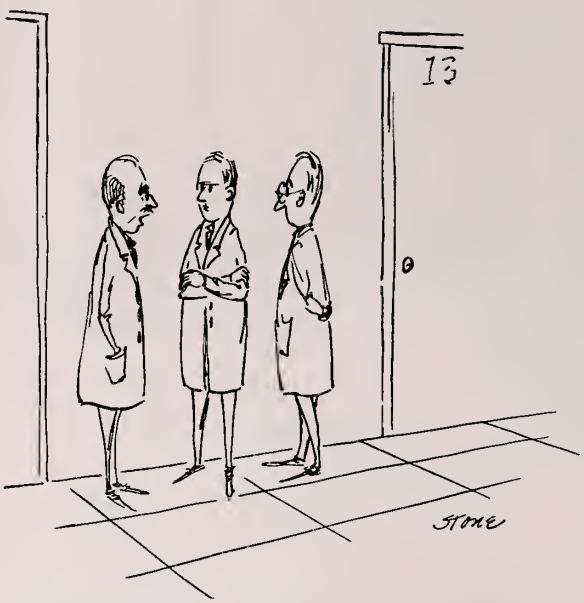
Medical Board Exams Set for June 22-24

The next meeting of the Texas State Board of Medical Examiners when examinations will be given and reciprocity applications considered is scheduled for June 22-24 at the Hotel Texas, Fort Worth.

Completed examination applications for graduates from United States medical schools must be filed with the Board, 1714 Medical Arts Building, Fort Worth, 30 days prior to the meeting date.

Completed examination applications for graduates of foreign medical schools must be filed 60 days prior to the meeting date.

Completed reciprocity applications must be filed 60 days prior to the meeting date to be given consideration, according to Dr. M. H. Crabb, secretary to the Board.



"We appointed him as a figurehead, but he turned out to be a hardhead."

Basic Science Exams Set for April 10-11

The next examination of the Texas State Board of Examiners in the Basic Sciences has been set for Friday and Saturday, April 10-11, 1964, in Austin, Dallas, Galveston, and Houston.

Details as to time and place may be obtained by writing to the executive secretary at 1012 Sam Houston State Office Bldg., 201 East 14th Street, Austin.

Physical Therapists Set April Meet in Brownsville

The 24th annual meeting of the Texas Chapter of the American Physical Therapy Association will be held April 24-26 in Brownsville at the Holiday Inn.

Persons interested may write to Geraldine Midgley, president Texas Chapter, APTA, 5115 Hialeah Drive, Houston, for additional information.

American College of Physicians To Meet in Atlantic City

The American College of Physicians will hold its 45th annual session in Atlantic City, N. J., April 6-10.

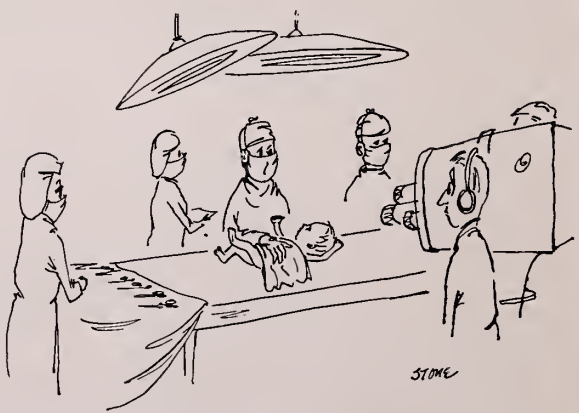
The five days of meetings will include rounds of scientific education aimed at keeping internists abreast of medical development.

Additional information may be obtained by writing Dr. Edward C. Rosenow, Jr., 4200 Pine Street, Philadelphia.

Peterson High on Medical Exam

Dr. Carl Christian Peterson, Jr., of Dallas made the highest grade on the December examinations given by the Texas State Board of Medical Examiners. Dr. Peterson had a general average of 92.4. He is from Rockford, Ill., and is a 1962 graduate of Northwestern University Medical School.

Examinations were held at the Blackstone Hotel in Fort Worth.



"Sorry, but the rabbi refuses to perform for television."

HOUSTON 64

97th Annual Session Program

April 23-26, 1964 (Thursday-Sunday)

Houston, Texas

Highlights

How To Use This Program

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SERVICES

Registration, Information, and Messages

Registration area will be open daily Thursday through Sunday, April 23-26, 7:30 a.m. to 5:30 p.m. in the Lobby of the Shamrock Hilton Hotel.

Information may be obtained from the registration desk or from the Message Center (booth located off Lobby on north promenade). The Message Center (telephone MO 6-2476) will accept emergency messages for physicians and will post lists of those receiving such messages in the meeting areas of the headquarters hotels. Physicians are asked to encourage their office staffs, families, and patients to keep their calls to the Message Center to a minimum and to check with their offices and homes at intervals to receive any messages.

The Harris County Medical Assistants Society will assist with ticket sales and in information and messenger service to physicians throughout the session.

Business, social, and educational sessions of the Woman's Auxiliary to the Texas Medical Association will be held at the Sheraton-Lincoln Hotel.

The Texas Medical Association will maintain an office in Room 351 of the Shamrock Hilton Hotel (Stenographers' Room). Mail and telegrams may be addressed in care of the Texas Medical Association, Shamrock Hilton Hotel, Houston, during the period of the annual session.

First Aid Station

A first aid station will be maintained throughout the annual session in Room 337, Shamrock Hilton Hotel. Dr. Thomas S. Behr, Houston, is in charge. The station will be staffed by nurses of the Houston-Harris County Chapter, American Red Cross.

News Room and TMA Daily Bulletin

A News Room will be maintained in Room 444 of the Shamrock Hilton Hotel (telephone MO 6-4456). Headquarters for *TMA Daily Bulletin* will be in Room 448 of the Shamrock Hilton Hotel.

Stenographers

A Stenographers' Room will be set up in Room 351 of the Shamrock Hilton Hotel (telephone MO 6-4711). Stenographic service will be furnished for Association business upon request at the Message Center in the Shamrock Hilton Hotel.

Equipment

Requests and problems involving equipment for meeting rooms may be directed to Room 335 of the Shamrock Hilton Hotel (telephone MO 6-4522).

HOTELS AND MOTELS

The Shamrock Hilton Hotel (Main at Holcombe Boulevard) and the Towers Hotel (2130 West Holcombe Boulevard) have been designated co-headquarters hotels for the Association. The Sheraton-Lincoln Hotel (777 Polk Avenue) will serve as Auxiliary headquarters.

In addition to the headquarters hotels, a partial

Presidents and Presidents-Elect

Dr. Robert Mayo Tenery
Waxahachie, President,
Texas Medical Association

Dr. Max E. Johnson
San Antonio,
President-Elect,
Texas Medical Association

Mrs. Hamilton F. Ford
LaMarque, President,
Woman's Auxiliary to the
Texas Medical Association

Mrs. W. D. Nicholson
Lake Jackson,
President-Elect,
Woman's Auxiliary to the
Texas Medical Association

list of hotels and motels housing Association and Auxiliary members is given below:

Grant Hotel, 8200 South Main
Hiway House, 9300 South Main
Holiday Inn, 10319 South Main
Las Vegas Motor Hotel, 9604 South Main
The Hotel Plaza, 5020 Montrose
Sheriton Oaks Motel, 7021 South Main
Sun Valley Motor Hotel, 1310 Old Spanish Trail
The Surrey House, 8300 South Main
Tidelands Motor Inn, 6500 South Main
Twenty-Nine Palms Motor Hotel, 9430 South Main
The Warwick, Main and Hermann

LOCATION OF EXHIBIT AREAS AND MEETING ROOMS OF HEADQUARTERS HOTELS

Scientific and Technical Exhibits Areas
Hall of Exhibits, First Floor, Shamrock Hilton Hotel

Shamrock Hilton Hotel

Azalea Room, Hall of Exhibits
Bluebonnet Room, Hall of Exhibits
Camelia Room, Hall of Exhibits
Castilian A, B, and C, Third Floor
Continental Room, First Floor
El Charro Room, Second Floor (enter through International Club)
Emerald Room, First Floor
Garden Room, First Floor
Grecian Room, First Floor
Hide-away, First Floor
International Club, First Floor
Nile Room, Third Floor
Normandy A and B, Third Floor
Pavilion Dining Room, First Floor
Pool Bar, First Floor (enter through Pavilion Dining Room)
Rooms 335, 337, 351, 353, 355, Third Floor
Rooms 442, 444, 446, 448, Fourth Floor
Room 1558, 15th Floor
Rooms 1620, 1658, 16th Floor
Storage Room, First Floor Check Room
Venetian Room, Third Floor
Walnut Room, Third Floor

Towers Hotel

Bayou Room, Second Floor
Minuet Room, Second Floor
Room 225, Second Floor Conference Room
Tower Room, Second Floor

Sheraton-Lincoln Hotel

Ballroom, East, Second Floor
Ballroom, Grand, Second Floor
French Salon, Third Floor
Massanet, Third Floor
Mezzanine, Second Floor
Mirador, 28th Floor

TAGP Accreditation

Hour-for-hour accreditation will be granted registrants by the Texas Academy of General Practice for attendance at scientific lectures of the Texas Medical Association Annual Session.

SPECIAL PROGRAMS

In conjunction with the programs of the scientific sections and related organizations, seven special programs will be offered:

Curbstone Consultations (informal round table discussions on topics in ten medical specialties), Friday, 8-9:30 p.m., Garden and Grecian Rooms, Shamrock Hilton Hotel.

Seminar of Texas Academy of General Practice, "Physiology and Pathology of the Lung," Thursday, 12:30-6 p.m., Azalea Room, Shamrock Hilton Hotel.

Symposium on Alcoholism, "A Three-Phase Treatment Program for Alcohol Addiction," Thursday, 8 p.m., Grecian Room, Shamrock Hilton Hotel.

Symposium on Cerebral Palsy, Saturday, 8:30 a.m.-4:30 p.m., Garden Room, Shamrock Hilton Hotel.

Symposium on Disaster Medical Care, Friday, 9 a.m.-12 Noon, Garden Room, Shamrock Hilton Hotel.

Symposium on Nutrition, "Basic Sciences in Relation to Nutrition," Friday, 2:30-5:30 p.m., Venetian Room, Shamrock Hilton Hotel.

The Teller Lecture (luncheon), "Operation Plowshare and Peacetime Uses of Atomic Energy," Edward Teller, Ph.D., Sunday, 12:45 p.m., Continental Room, Shamrock Hilton Hotel.

CLOSED-CIRCUIT TELEVISION PRESENTATIONS

Two annual session topics will be presented via closed-circuit television to registrants. Eidophor, the large-screen, color system developed by Ciba Pharmaceutical Company, Summit, N. J., will be utilized to transmit the papers of Dr. Mark B. Coventry, Head, Section of Orthopaedic Surgery, Mayo Clinic, Rochester, Minn., and Dr. Charles A. Berry, Chief, Center Medical Operations Office, National Aeronautics and Space Administration, Manned Spacecraft Center, Houston.

Friday at 11 a.m., in a general meeting session, Dr. Coventry will present and discuss patients with "Low Back Pain." Dr. Berry will discuss the preparation of the astronaut for flight and his monitoring while in flight in his topic, "Medical Support of Man in Space," Saturday, 11 a.m., before a general meeting session. Both television presentations may be seen in the Emerald Room of the Shamrock Hilton Hotel. By a two-way audio system, the audience will be given an opportunity to direct questions to the speakers.

ENTERTAINMENT

Luncheons

The Association will sponsor two luncheons during the annual session. The General Meeting Luncheon will be held on Friday and the Annual Luncheon and Installation of Officers will be held on Saturday. Both luncheons will be held at 12:30 p.m. in the Continental Room of the Shamrock Hilton Hotel. Members of the Association and Auxiliary and guests are invited. Tickets will be required for admittance, and they will be on sale at $3.25 each at the registration desk in the Lobby of the Shamrock Hilton Hotel.

Past Presidents Association and Fifty Year Club

The Past Presidents Association will have a reserved table at the General Meeting Luncheon on Friday and the Fifty Year Club a reserved table at the Annual Luncheon and Installation of Officers on Saturday. Both luncheons will be held at 12:30 p.m. in the Continental Room of the Shamrock Hilton Hotel. Tickets at $3.25 each will be on sale at the registration desk in the Lobby of the Shamrock Hilton Hotel.

Alumni Events

Alumni receptions and banquets, scheduled for Saturday, are being arranged under the general direction of Dr. Carl J. Lind, Jr., of Houston. Tickets will be on sale in the registration area. Events planned, together with location, time, and person in charge, are as follows:

Tulane University School of Medicine, New Orleans, Reception and Banquet, Castilian A, B, and C, Shamrock Hilton Hotel, 6:30 p.m. Dr. Morris W. Kilgore, Houston, in charge.

University of Texas Medical Branch, Galveston, Cocktail-Buffet, Emerald Room, Shamrock Hilton Hotel, 7-9 p.m. Miss Mildred M. Robertson, University of Texas Medical Branch Alumni Association, Galveston; and Dr. Frederick R. Lummis, Houston, in charge.

Alumni Association

Officers and Board of Trustees, University of Texas Medical Branch Alumni Association, will meet Friday, 2 p.m., in the Minuet Room of the Towers Hotel.

Class Reunions

University of Texas Medical Branch class reunion-cocktail parties, scheduled for Saturday, are being arranged under the general direction of Miss Mildred M. Robertson, University of Texas Medical Branch Alumni Association, Galveston; and Dr. Carl J. Lind, Jr., Houston.

Tickets will be on sale in the registration area. Events planned, together with location, time, and person in charge, are as follows:

Class of 1914, Emerald Room, Shamrock Hilton Hotel, 7 p.m. Dr. E. W. Breihan, Dallas, in charge.

Class of 1924, Nile Room, Shamrock Hilton Hotel, 6:30 p.m. Dr. George D. Huff, San Diego, Calif., in charge.

Class of 1934, Emerald Room, Shamrock Hilton Hotel, 7 p.m. Dr. John A. Wall, Houston, in charge.

Class of 1939, Emerald Room, Shamrock Hilton Hotel, 7 p.m., Dr. Phillip A. Belleggie, Houston, in charge.

Class of 1944, Criterion Club, 16th Floor, Fannin State Bank Bldg., 1020 Holcombe Blvd., 5:30 p.m., Dr. W. Tom Arnold and Dr. Mylie E. Durham, Jr., Houston, in charge.

Class of 1954, Normandy B, Shamrock Hilton Hotel, 6:30 p.m. Dr. Charles D. Chamberlain, Jr., Houston, in charge.

Class members are invited to join the University of Texas Medical Branch alumni for buffet following the individual class reunion-cocktail parties. The buffet will be held Saturday in the Emerald Room, Shamrock Hilton Hotel, 7 to 9 p.m. Tables will be set up and marked for specific classes.

Fraternity Parties

Fraternity parties are planned from 6:30 to 7:30 p.m., Saturday. Tickets will be on sale in the registration area. Dr. Carl J. Lind, Jr., Houston, is general chairman for these events. Individual receptions, location, and the Houston physician making arrangements follow:

Phi Chi, Normandy A, Shamrock Hilton Hotel, Dr. Edward Griffey (1805 Medical Towers)

Theta Kappa Psi, Gold Room, Tidelands Inn Motor Hotel, Dr. William R. Kerr (6436 Fannin)

Women Physicians

Texas Women Physicians Association will meet at 7:30 a.m., Saturday, in the Nile Room of the Shamrock Hilton Hotel for breakfast. Tickets will be on sale in the registration area and at the door of the Nile Room. Dr. Karin Aileen Petri, Houston, is making arrangements.

Houston Society of Clinical Pathologists

A reception is planned for the Houston Society of Clinical Pathologists on Saturday, April 25, at 6:30 p.m. in the Grecian Room of the Shamrock Hilton Hotel. Dr. Carl J. Lind, Jr., Houston, is making arrangements.

Sports

A golf tournament will be held Friday at the Westwood Country Club, Richmond Road, Houston. Directions to the Club may be obtained at the Sports Events desk located in the Lobby of the Shamrock Hilton Hotel. Information regarding the tournament is given below:

Tee off time: Anytime after 12 Noon.
Cost: Fee of $3.00 includes green fees and prizes.
Rules of Play: USGA Rules.
Prizes: Trophies will be awarded to flight winners. Other prizes will be given.
Handicap Rules: Players presenting certified handicaps signed by home professional may play in handicap tournament.
Equipment: Physicians should bring own clubs.
Foursomes: Physicians should make up own foursomes.

Dr. Homer A. Taylor, Jr., 1406 Memorial Professional Building, Houston, is chairman in charge of sports events and is handling arrangements for the golf tournament.

A skeet shoot may be offered during the annual session, if enough interest in the event is indicated. Physicians interested in participating in a skeet shoot may register at the Sports Events desk. Dr. Jack N. Bevil and Dr. Edward T. Clarke, both of Houston, are in charge of the skeet shoot.

BUSINESS ACTIVITIES

House of Delegates

The three sessions of the House of Delegates will be held as follows in the Emerald Room of the Shamrock Hilton Hotel:

Thursday, 2 p.m.
Friday, 7:30 p.m.
Sunday, 8:30 a.m.

A dinner for the House of Delegates will be held at 6 p.m. Friday in the Azalea and Bluebonnet Rooms of the Shamrock Hilton Hotel.

The agenda, with reports and resolutions available at press time and a list of officers and committees, are included in a Handbook for Delegates and may be obtained by others upon request. All members of the Association may attend meetings of the House but may not participate in discussion or voting unless they are designated as delegates. However, all members of the Association have the privilege of appearing before reference committees of the House of Delegates in support of or in opposition to any proposal.

Reference Committees

Reference Committees will hold their first meetings to consider business assigned to them by the Speaker of the House of Delegates at 7 p.m. Thursday. Additional meetings will be at such other times as the chairmen of the committees find necessary. Meeting places other than for the first meetings will be assigned at the Message Center in the Shamrock Hilton Hotel. Committee chairmen are urged to inform the Message Center staff when they have called meetings so that inquiries may be answered. Meetings of reference committees Thursday at 7 p.m. will be held in the Shamrock Hilton Hotel as follows:

Constitution and By-Laws, Normandy B.
Legislation and Public Relations, Normandy A.
Medical Service and Insurance, Walnut Room.
Miscellaneous Business, Venetian Room.
Reports of Officers and Committees, Castilian C.
Scientific Work, Castilian A.
Board of Trustees, Room 1558.
Board of Councilors, Room 1620.

Chairmen of reference committees are asked to attend a luncheon meeting with the Speaker of the House of Delegates at 12 noon Thursday in the Venetian Room of the Shamrock Hilton Hotel to discuss procedures and expedite business.

Board, Council, and Committee Meetings

A number of Texas Medical Association board, council, and committee meetings are being scheduled during the annual session. Those scheduled at press time follow:

Wednesday

6:30 p.m. Board of Trustees (dinner), Room 1558, Shamrock Hilton Hotel.
7:00 p.m. Committee on Physicians Benevolent Fund (dinner), Venetian Room, Shamrock Hilton Hotel.
7:30 p.m. Committee for American Medical Association Education and Research Foundation (dinner), Normandy A, Shamrock Hilton Hotel.
7:30 p.m. Committee on Health Insurance (dinner), Nile Room, Shamrock Hilton Hotel.
7:30 p.m. Committee on Mental Health (dinner), Normandy B, Shamrock Hilton Hotel.
7:30 p.m. Committee on Nuclear Medicine (dinner), Walnut Room, Shamrock Hilton Hotel.

7:30 p.m. **Council on Constitution and By-Laws** (dinner), Castilian A, Shamrock Hilton Hotel.
8:00 p.m. **Council on Medical Jurisprudence**, Castilian C, Shamrock Hilton Hotel.
8:00 p.m. Committee on Liaison with State Bar of Texas, Room 225, Towers Hotel.

Thursday

7:00 a.m. Committee on Nuclear Medicine (breakfast), Walnut Room, Shamrock Hilton Hotel.
7:00 a.m. **Council on Medical Education and Hospitals** (breakfast), Venetian Room, Shamrock Hilton Hotel.
7:30 a.m. **Council on Scientific Advancement** (breakfast), Garden Room, Shamrock Hilton Hotel.
7:30 a.m. Delegates to American Medical Association (breakfast), Nile Room, Shamrock Hilton Hotel.
9:00 a.m. Committee on Association Insurance Programs, Hide-away, Shamrock Hilton Hotel.
9:00 a.m. **Board of Councilors**, Suite 1620, Shamrock Hilton Hotel.
10:00 a.m. Committee on Aging, Room 225, Towers Hotel.
10:00 a.m. Committee on Medical Careers, Pool Bar, Shamrock Hilton Hotel.
10:00 a.m. Committee on Public Health, Minuet Room, Towers Hotel.
10:00 a.m. Committee on Workmen's Compensation Insurance, Normandy B, Shamrock Hilton Hotel.
10:00 a.m. **Council on Medical Service and Insurance**, Walnut Room, Shamrock Hilton Hotel.
10:00 a.m. **Council on Public Relations and Public Service**, Normandy A, Shamrock Hilton Hotel.
10:30 a.m. Committee on Athletics, El Charro Room, Shamrock Hilton Hotel.
12:00 Noon **Council on Annual Session** (luncheon), Nile Room, Shamrock Hilton Hotel.
7:30 p.m. Committee on Cardiovascular Diseases (dinner), Minuet Room, Towers Hotel.

Friday

7:30 a.m. Committee on Medicine and Religion (breakfast), Minuet Room, Towers Hotel.
7:30 a.m. Committee on Professional Insurance (breakfast), Room 225, Towers Hotel.
10:00 a.m. Committee on School Health, Room 225, Towers Hotel.
10:30 a.m. Committee on Cancer, Minuet Room, Towers Hotel.
2:30 p.m. Committee on Disaster Medical Care, Tower Room, Towers Hotel.
4:00 p.m. Committee on Contract Medicine, Room 225, Towers Hotel.
5:30 p.m. Committee on Industrial Health, Nile Room, Shamrock Hilton Hotel.
6:00 p.m. Committee on Rehabilitation (dinner), Venetian Room, Shamrock Hilton Hotel.
7:30 p.m. Committee on Scientific Exhibits (dinner), Walnut Room, Shamrock Hilton Hotel.

Saturday

7:00 a.m. Texans for Political Action Committee (breakfast), Continental Room, Shamrock Hilton Hotel.
8:00 a.m. Committee on Maternal Mortality (breakfast), Bayou Room, Towers Hotel.
8:00 a.m. Committee on Nutrition (breakfast), Room 225, Towers Hotel
10:00 a.m. Committee on Public Health, Venetian Room, Shamrock Hilton Hotel.
6:30 p.m. Committee on Medical History (dinner), Garden Room, Shamrock Hilton Hotel.

TexPac Breakfast

The Texans for Political Action Committee will meet for a breakfast session Saturday at 7 a.m. in the Continental Room of the Shamrock Hilton Hotel. Rep. Bob Casey of Houston will be guest speaker.

Blue Cross-Blue Shield Dinner

Blue Cross-Blue Shield of Texas will have a dinner for members of the House of Delegates on Friday at 6 p.m. in the Azalea and Bluebonnet Rooms of the Shamrock Hilton Hotel. Mr. W. R. McBee, executive director, Blue Cross-Blue Shield of Texas, Dallas, is in charge of arrangements.

ANNUAL SESSION COMMITTEES

The Council on Annual Session, which has coordinated plans for the Annual Session, consists of Drs. Herman C. Sehested, Fort Worth, Chairman; Dennis M. Voulgaris, Wharton; John P. McGovern, Houston; W. Tom Arnold, Houston; John C. Meadows, Jr., San Antonio; Madison J. Lee, Jr., Tyler; John W. Chriss, Corpus Christi; Robert Mayo Tenery, Waxahachie, President (ex officio); and Mr. C. Lincoln Williston, Austin, Executive Secretary (ex officio). Mrs. Dale Willimack, Austin, serves as Staff Coordinator of Annual Session.

The Committee on General Arrangements for the 1964 Annual Session, all members from the Harris County Medical Society, includes Dr. Orceneth A. Fly, Jr., Chairman, and Dr. Benjamin F. Kitchen, Jr., Vice Chairman. Subcommittee chairmen (all from Houston) are listed below:

Dr. Carl J. Lind, Jr. (alumni and fraternities)
Dr. Thomas S. Behr (first aid)
Dr. Charles A. Dwyer (housing)
Dr. Irving W. Moody (information)
Dr. Jared E. Clarke (memorial service)
Dr. Arthur M. Jansa (public lectures)
Dr. Lloyd J. Gregory, Jr. (publicity)
Dr. John P. McGovern (scientific exhibits)
Dr. Homer A. Taylor, Jr. (sports)
Dr. E. Trowbridge Wolf (technical exhibits)

Mr. Bill E. Robertson is Executive Secretary of the Harris County Medical Society. Texas Medical Association staff members assisting with phases of the Annual Session include: Mr. Donald M. Anderson, Assistant Executive Secretary, hotels and equipment; Mr. Jon Hornaday, publicity and public rela-

tions; Mr. Dale Werner, scientific and technical exhibits; and Miss Pauline Duffield, memorial service and film program.

Mrs. George H. Brandau of Houston is convention chairman for the Woman's Auxiliary.

ALSO WATCH FOR—

TMA DAILY BULLETIN to be distributed at the session.

Memorial Service program, with names of those paid tribute, to be distributed at the service.

Handbook for Delegates, containing agenda of House of Delegates, annual reports of committees, and officer and committee list, available for distribution at registration.

Woman's Auxiliary Program, containing convention details, to be distributed at Auxiliary registration.

Speakers

GUEST SPEAKERS

Dr. Willard M. Allen

Professor of Obstetrics and Gynecology, Washington University School of Medicine, St. Louis.

Functional Bleeding and Secondary Amenorrhea, Refresher Course, Friday, 8:30-9:45 a.m.
Precocious and Delayed Puberty, Section on Obstetrics and Gynecology, Friday, 2:30-3 p.m.
Cardiac Arrest (Panel), Joint Session of Section on Obstetrics and Gynecology, Section on Surgery, and Texas Society of Anesthesiologists, Friday, 4-5 p.m.
Progestational Agents in Endometrial Carcinoma, Section on Obstetrics and Gynecology, Saturday, 2:30-3 p.m.
Sponsor: Donald M. Paton, Houston.
Cosponsor: John A. Wall, Houston.
Guest of Section on Obstetrics and Gynecology, Texas Medical Association.

Dr. Edward R. Annis

President, American Medical Association, Miami, Fla.

It's an Amazing Story, Annual Luncheon Address, Saturday, 1:40-2 p.m.
Sponsor: Milford O. Rouse, Dallas.
Cosponsor: Denton Kerr, Houston.
Guest of American Medical Association.

Dr. R. G. Bell

Executive Director, The Donwood Foundation; Medical Director, The Bell Clinic, Willowdale, Ontario, Canada.

A Three-Phase Treatment Program for Alcohol Addiction, Symposium on Alcoholism, Thursday, 8 p.m.
Clinical Management of Defensive Thinking, Refresher Course, Friday, 8:30-9:45 a.m.
Sponsor: Exter F. Bell, Jr., Houston.
Cosponsor: Mr. Richard Hume, Houston.
Guest of Texas Commission on Alcoholism, Houston Council on Alcoholism, and University of Texas.

Dr. Mark B. Coventry

Head, Section of Orthopaedic Surgery, Mayo Clinic, Rochester, Minn.

Athletic Injuries in the Winter Sports, Texas Society of Athletic Team Physicians, Thursday, 2:30-3 p.m.
Low Back Pain, Closed-Circuit Television Presentation, General Meeting, Friday, 11 a.m.-12 Noon.
Treatment of Malum Coxae Senilis, Texas Orthopaedic Association, Saturday, 2-3 p.m.
Sponsor: Bruce M. Cameron, Houston.
Cosponsor: Gerald S. Ahern, Corpus Christi.
Guest of Texas Orthopaedic Association and Texas Society of Athletic Team Physicians.

Dr. Charles L. Eckert

Professor and Chairman, Department of Surgery, Albany Medical College, Albany, N. Y.

Inflammatory Lesions of the Intestinal Tract, Refresher Course, Friday, 8:30-9:45 a.m.
Therapeutic Indications in Breast Cancer, Section on Surgery, Friday, 3-3:30 p.m.
Cardiac Arrest (Panel), Joint Session of Section on Obstetrics and Gynecology, Section on Surgery, and Texas Society of Anesthesiologists, Friday, 4-5 p.m.
Diseases of the Thyroid (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 2:30-3:40 p.m.
Sponsor: Wilson G. Brown, Houston.
Cosponsor: Frank H. Kidd, Jr., Dallas.
Guest of Section on Surgery, Texas Medical Association.

Dr. Max Ellenberg

Associate Attending Physician, Mt. Sinai Hospital, New York.

Diabetic Nephropathy, General Meeting Address, Saturday, 10-10:25 a.m.
Diagnosis of the Prediabetic State, Section on General Practice, Saturday, 4-4:30 p.m.
Diabetic Diarrhea, Texas Diabetes Association, Sunday, 10:50-11:20 a.m.
Thyroid Disease and Diabetes (Panel), Texas Diabetes Association, Sunday, 1 p.m.
Sponsor: Harold L. Dobson, Houston.
Cosponsor: Sidney Schnur, Houston.
Guest of Texas Diabetes Association and Texas Medical Association.

Dr. John A. Evans

Radiologist-in-Chief, The New York Hospital-Cornell Medical Center, New York.

Sclerosing Retroperitonitis and Associated Lesions, Section on Radiology, Friday, 3:40-4:10 p.m.
Specialized Roentgen Techniques in the Diagnosis of Abdominal Disease, Refresher Course, Saturday, 8:30-9:45 a.m.
Gastrointestinal Polyps (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 3:50-5 p.m.
Sponsor: Vincent P. Collins, Houston.
Cosponsor: Giles L. Andrews, Beaumont.
Guest of Section on Radiology, Texas Medical Association.

Dr. D. Bernard Foster

Director, Division of Neurology and Neurosurgery, The Menninger Clinic, Topeka, Kan.

The Differential Diagnosis of Vertigo, Refresher Course, Saturday, 8:30-9:45 a.m.
Mental Retardation, Texas Neuropsychiatric Association, Sunday, 9:45-10:15 a.m.
Pain in the Neck-Shoulder-Arm, Section on Neurology, Neurosurgery and Allied Fields, Texas Neuropsychiatric Association, Sunday, 3-3:30 p.m.
Sponsor: James K. Peden, Dallas.
Cosponsor: Charles L. Bloss, Dallas.
Guest of Texas Neuropsychiatric Association and Texas Medical Association.

Dr. A. Stone Freedberg

Associate Professor of Medicine, Harvard University Medical School, Boston.

The Laboratory Diagnosis of Coronary Artery Disease and Insufficiency, Section on Internal Medicine, Friday, 3-3:30 p.m.
Diseases of the Thyroid (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 2:30-3:40 p.m.
Diagnostic Tests of Thyroid Function, Texas Diabetes Association, Sunday, 10:20-10:50 a.m.
Thyroid Disease and Diabetes (Panel), Texas Diabetes Association, Sunday, 1 p.m.
Sponsor: Leonard R. Robbins, Houston.
Cosponsor: Mavis P. Kelsey, Houston.
Guest of Texas Diabetes Association.

Dr. S. Raymond Gambino

Director of Laboratories, Englewood Hospital, Englewood, N.J.

Screening for Clotting Abnormalities. A History Form and Basic Tests, Section on Pathology, Friday, 2:30-3 p.m.
Coagulation Problems in the Laboratory (Panel), Section on Pathology, Friday, 3:05-4:20 p.m.
The Liver Panel. The Differential Diagnosis of Liver Diseases on the Basis of Laboratory Tests, Section on Pathology, Friday, 4:30-5 p.m.
Diseases of the Thyroid (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 2:30-3:40 p.m.
Sponsor: Elwood E. Baird, Galveston.
Cosponsor: Eugene L. Bigelow, Houston.
Guest of Section on Pathology, Texas Medical Association.

Dr. Albert B. Hagedorn

Associate Professor, Section of Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minn.

Hematological Clues to Malignant Disease, Section on Internal Medicine, Friday, 3:30-4 p.m.
Anemias Not Responding to Ordinary Hematinics, Refresher Course, Saturday, 8:30-9:45 a.m.
Gastrointestinal Polyps (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 3:50-5 p.m.
Sponsor: Gene E. Burke, Houston.
Cosponsor: Clarence P. Alfrey, Houston.
Guest of Section on Internal Medicine, Texas Medical Association.

Dr. Patrick H. Hanley

Associate Professor of Clinical Surgery and Head, Department of Colon and Rectal Surgery, Tulane University School of Medicine; Head, Department of Colon and Rectal Surgery, Ochsner Clinic, New Orleans.

Nonspecific Ulcerative Colitis, Surgical Indications and Management, Texas Proctologic Society, Thursday, 2:30-3 p.m.
Present Day Management of Diverticulitis, General Meeting Address, Friday, 10:30-10:55 a.m.
Anal Fissures, Abscesses, and Fistulas, Refresher Course, Friday, 8:30-9:45 a.m.
Sponsor: Herbert T. Hayes, Houston.
Cosponsor: John McGivney, Galveston.
Guest of Texas Proctologic Society and Texas Medical Association.

Dr. John N. Hunt

Department of Physiology, Guy's Hospital Medical School, London, England.

The General Principles in the Interpretation of Test of Gastric Function, Refresher Course, Saturday, 8:30-9:45 a.m.
Duodenal Receptors, Section on Digestive Diseases, Sunday, 9-9:30 a.m.
Symposium on Newer Aspects of Treatment of Peptic Ulcer Disease, Section on Digestive Diseases, Sunday, 10:45 a.m.-12:15 p.m.
Sponsor: John R. Kelsey, Jr., Houston.
Cosponsor: Philip S. Bintlif, Houston.
Guest of Section on Digestive Diseases, Texas Medical Association.

Dr. Albert S. Hyman

Associate Clinical Professor of Medicine, New York Medical College, New York.

Estimation of Physical Fitness of the Cardiovascular System, Texas Society of Athletic Team Physicians, Thursday, 2:10-2:30 p.m.
The Cardiac Athlete, Texas Society of Athletic Team Physicians, Thursday, 3:55-4:15 p.m.
Sponsor: James R. Whitehurst, Houston.
Cosponsor: Harry L. Kaplan, Houston.
Guest of Texas Society of Athletic Team Physicians.

Dr. William K. Ishmael

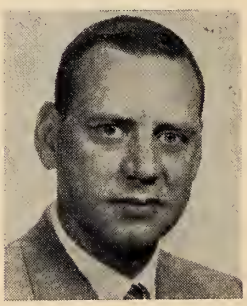
McBride Clinic, Inc., Oklahoma City.

Osteoporosis, Present Concepts, Texas Society on Aging, Clinical Medicine Section, Sunday, 10-10:30 a.m.
Conservative Care of the Back and Neck, Texas Society on Aging, Clinical Medicine Section, Sunday, 2-2:30 p.m.
Sponsor: William P. Price, Houston.
Guest of Texas Society on Aging, Clinical Medicine Section, and Texas Medical Association.

Dr. Margaret H. Jones

Professor of Pediatrics, University of California at Los Angeles School of Medicine, Los Angeles, Calif.

The Early Diagnosis of Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 9:05-9:45 a.m.
Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy (Panel), Symposium on Cerebral Palsy, Saturday, 11 a.m.-12 Noon.
The Research Challenge in Cerebral Palsy, Luncheon Address, Symposium on Cerebral Palsy, 12:30 p.m.
High Risk Babies, Section on General Practice, Saturday, 3:30-4 p.m.
Sponsor: E. Burke Evans, Galveston.
Cosponsor: Ralph Hanna, Austin.
Guest of United Cerebral Palsy Association of Texas, Inc.



Dr. Kenneth K. Keown

Professor of Anesthesiology, University of Missouri Medical Center, University of Missouri School of Medicine, Columbia, Mo.

Cardiac Arrest (Panel), Joint Session of Section on Obstetrics and Gynecology, Section on Surgery, and Texas Society of Anesthesiologists, Friday, 4-5 p.m.
Anesthesia for Patients with Heart Disease, Texas Society of Anesthesiologists, Sunday, 11-11:30 a.m.
The Recognition of Heart Failure in the Anesthetized Patient, Texas Society of Anesthesiologists, Sunday, 3:30-4 p.m.
Sponsor: Lawrence F. Schulmacher, Jr., Houston.
Cosponsor: Fred P. Thomas, Houston.
Guest of Texas Society of Anesthesiologists and Texas Medical Association.

Dr. Saul Krugman

Professor and Chairman, Department of Pediatrics, New York University School of Medicine, New York.

Natural History and Prevention of Infectious Hepatitis, Section on Pediatrics, Friday, 3-3:30 p.m.
Measles Vaccines, Section on General Practice, Friday, 4-5 p.m.
The Physician and the Rash, Section on General Practice, Saturday, 3-3:30 p.m.
Sponsor: Nathan Cotlar, Houston.
Cosponsor: Russell J. Blattner, Houston.
Guest of Section on General Practice, Texas Medical Association.

Dr. Sydney G. Margolin

Professor of Psychiatry, University of Colorado School of Medicine, Denver, Colo.

Pathological Obesity and Psychopathology, Section on Internal Medicine, Friday, 4-4:30 p.m.
Phobias, Depressions, and Addictions, Texas Neuropsychiatric Association, Sunday, 10:30-11 a.m.
The Essentials of Psychotherapy, Section on Psychiatry and Psychoanalysis, Texas Neuropsychiatric Association, Sunday, 3-3:30 p.m.
Sponsor: H. Harlan Crank, Houston.
Cosponsor: Shervert H. Frazier, Houston.
Guest of Texas Neuropsychiatric Association and Texas Medical Association.

Dr. Clarence S. Livingood

Chairman, Department of Dermatology, Henry Ford Hospital, Detroit.

The Diagnosis and Management of Selected Common Skin Diseases, Refresher Course, Friday, 8:30-9:45 a.m.
The Management of Bacterial Infections of the Skin—20 Years After the Introduction of Antibiotics, Texas Dermatological Society, Saturday, 9:30-10 a.m.
The Iatrogenic Diseases Due to Treatment of Cutaneous Diseases, Section on Pediatrics, Saturday, 2:30-3 p.m.
Sponsor: Edward M. Shapiro, Pasadena.
Cosponsor: Everett R. Seale, Houston.
Guest of Texas Dermatological Society and Texas Medical Association.

Dr. Walter T. Murphy

Chief of Radiation Therapy, Buffalo General Hospital, Buffalo.

Irradiation of Carcinoma of the Urinary Bladder, Section on Radiology, Friday, 2:50-3:20 p.m.
Diseases of the Thyroid (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 2:30-3:40 p.m.
Sponsor: Gilbert H. Fletcher, Houston.
Cosponsor: Lowell S. Miller, Houston.
Guest of Texas Division, American Cancer Society.

Dr. George H. Lowrey

Professor of Pediatrics, University of Michigan Medical Center, University of Michigan, Ann Arbor, Mich.

Thyrotoxicosis and Its Treatment, Section on Pediatrics, Friday, 2:30-3 p.m.
Obesity in Childhood, Section on Pediatrics, Saturday, 3-3:30 p.m.
Iatrogenic Disorders in the Fetus and Neonatal Period (Panel), Joint Session of Section on Obstetrics and Gynecology and Section on Pediatrics, Saturday, 4-5 p.m.
The Adolescent with Diabetes Mellitus, Texas Diabetes Association, Sunday, 9:35-10:05 a.m.
Sponsor: A. Thomas Adkins, Houston.
Cosponsor: C. William Daeschner, Galveston.
Guest of Section on Pediatrics, Texas Medical Association.

Dr. Oglesby Paul

Chief, Division of Medicine, Passavant Memorial Hospital, Chicago.

The Management of the Patient with an Arrhythmia, Refresher Course, Friday, 8:30-9:45 a.m.
The Patient with Severe Heart Failure, Section on Internal Medicine, Friday, 2:30-3 p.m.
Cardiac Arrest (Panel), Joint Session of Section on Obstetrics and Gynecology, Section on Surgery, and Texas Society of Anesthesiologists, Friday, 4-5 p.m.
Sponsor: R. William Baird, Jr., Houston.
Cosponsor: James E. Kreisle, Austin.
Guest of Texas Heart Association and Texas Medical Association.

Dr. Brittain F. Payne

Clinical Professor of Ophthalmology, New York University-Bellevue Medical School, New York.

Early Treatment of Common Eye Injuries (primarily for non-ophthalmologists), Refresher Course, Friday, 8:30-9:45 a.m.
Pathological Appraisal of Retinal Detachment Operations, Section on Ophthalmology, Friday, 3:30-4:30 p.m.
Prevention of Complications in Cataract Surgery, Section on Ophthalmology, Saturday, 4-5 p.m.
Sponsor: Everett L. Goar, Houston.
Cosponsor: Jacob F. Schultz, Houston.
Guest of Section on Ophthalmology, Texas Medical Association.

Dr. Charles E. Smith

Dean, School of Public Health, University of California, Berkeley, Calif.

Contributions of the Ethics of Medical Consultation to the Understanding of the Medical Practitioner and His Health Department, Texas Association of Public Health Physicians, Thursday, 8 p.m.
Some Problems and Challenges in Public Health, Section on Public Health, Friday, 2:30-3 p.m.
Accreditation in Public Health—Advice Welcomed, Conference of City and County Health Officers, Saturday, 2:30-3 p.m.
Sponsor: Charles A. Pigford, Houston.
Cosponsor: J. E. Peavy, Austin.
Guest of Conference of City and County Health Officers and Texas Medical Association.

Dr. G. O. Proud

Professor and Chairman, Department of Otorhinolaryngology, University of Kansas School of Medicine, Kansas City, Kan.

Evaluation of the Dizzy Patient, Refresher Course, Friday, 8:30-9:45 a.m.
Eustachian Tube Function Tests, Section on Otolaryngology, Friday, 2:30-3:15 p.m.
Otitis Externa and Laryngoscopy in Children, Texas Otolaryngological Association, Saturday, 10-10:45 a.m.
Middle Ear Effusion, Section on Otolaryngology, Saturday, 2:30-3:15 p.m.
Sponsor: Ben T. Withers, Houston.
Cosponsor: Louis E. Adin, Jr., Dallas.
Guest of Section on Otolaryngology, Texas Medical Association.

Dr. Fredrick J. Stare

Professor and Chairman, Department of Nutrition, School of Public Health, Harvard University, Boston.

Nutrition in Cardiovascular Disease, General Meeting Address, Friday, 10-10:25 a.m.
Nutritional Nonsense, General Meeting Luncheon Address, Friday, 1:25-1:55 p.m.
Sponsor: Ralph G. Greenlee, Midland.
Cosponsor: William Levin, Galveston.
Guest of Council on Annual Session, Texas Medical Association.

Dr. Woodrow D. Schlosser

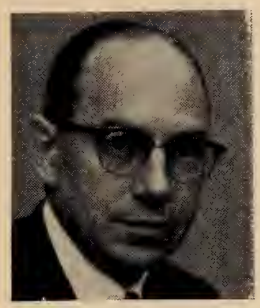
Assistant Director, Institute of Otology of the Presbyterian Hospital, Philadelphia, Pa.

Surgical Treatment and Findings in Mixed Congenital Deafness, Texas Otolaryngological Association, Friday, 10-10:45 a.m.
Some Aspects of Tympanoplasty, Texas Otolaryngological Association, Friday, 11:15 a.m.-12 Noon.
The Treatment of the Draining Ear in Children, Section on Otolaryngology, Friday, 3:45-4:30 p.m.
The Diagnosis and Treatment of Acoustic Neuroma, Texas Otolaryngological Association, Saturday, 11:15 a.m.-12 Noon.
Sponsor: William K. Wright, Houston.
Cosponsor: Ben M. Nail, Jr., Houston.
Guest of Texas Otolaryngological Association.

Dr. H. Saul Sugar

Clinical Professor of Ophthalmology, Wayne State University College of Medicine, Detroit.

Pupil Block and Pupillary Block Glaucoma, Texas Ophthalmological Association, Friday, 10:30 a.m.-12 Noon.
Deceptive Diagnosis in Lesions of the Ocular Fundus, Refresher Course, Saturday, 8:30-9:45 a.m.
Heterochromic Cyclitis, Texas Ophthalmological Association. Saturday, 10:30 a.m.-12 Noon.
Sponsor: Ray K. Daily, Houston.
Cosponsor: Henry N. Ricci, San Angelo.
Guest of Texas Ophthalmological Association.



Dr. Chester A. Swinyard

Director, Children's Division, Institute of Physical Rehabilitation, New York University Medical Center, New York.

The Individual with Postnatally Acquired Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 9:45-10:30 a.m.
Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy (Panel), Symposium on Cerebral Palsy, Saturday, 11 a.m.-12 Noon.
Sponsor: Oscar O. Selke, Jr., Houston.
Cosponsor: Lewis M. Helfer, San Antonio.
Guest of United Cerebral Palsy Association of Texas, Inc.

Dr. Watts R. Webb

Chairman, Division of Cardiovascular Surgery, Department of Surgery, The University of Texas Southwestern Medical School, Dallas.

Holes in the Lungs—Diagnosis and Management, Section on Surgery, Friday, 3:30-4 p.m.
Respiratory Emergencies in the Newborn, Refresher Course, Saturday, 8:30-9:45 a.m.
Pulmonary Transplantation—Experimental and Clinical Observations, Texas Chapter, American College of Chest Physicians, Sunday, 11:30 a.m.-12:15 p.m.
Present Status of Surgery of the Heart, Curbstone Consultation, Friday, 8 p.m.
Sponsor: James E. Daily, Houston.
Cosponsor: Milton V. Davis, Dallas.
Guest of Texas Chapter, American College of Chest Physicians, and Texas Medical Association.

Dr. Nathan A. Womack

Professor and Head, Department of Surgery, University of North Carolina School of Medicine, Chapel Hill, N.C.

Chronic Cystic Mastitis, General Meeting Address, Saturday, 10:30-10:55 a.m.
Gastrointestinal Polyps (Panel), Joint Session of Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology, and Surgery, Saturday, 3:50-5 p.m.
Paré and Sixteenth Century Surgery, Committee on Medical History, Saturday, 6:30 p.m.
Symposium on Newer Aspects of Treatment of Peptic Ulcer Diseases, Section on Digestive Diseases, Sunday, 10:45 a.m.-12:15 p.m.
Sponsor: James E. Pittman, Houston.
Cosponsor: Adam N. Boyd, Houston.
Guest of Texas Division, American Cancer Society.

SPECIAL SPEAKERS

Tina E. Bangs, Ph.D., Director, Speech and Hearing Center, Houston.
Panel: Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 11 a.m.

J. P. Boland, Resident in Thoracic Surgery, Parkland Memorial Hospital, Dallas.
A New Technique for Division of the Difficult Patent Ductus Arteriosus, and Its Successful Use in a Sixty-Year-Old Woman, Texas Chapter, American College of Chest Physicians, Sunday, 2:15 p.m.

Oliver H. Bown, Ph.D., Associate Professor of Educational Psychology and Assistant Director of Personality Research Center, University of Texas, Austin.
Panel: Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 11 a.m.
The Need for More Impactful Counseling as a Component of the Total Treatment Program, Symposium on Cerebral Palsy, Saturday, 3:45 p.m.

Charles H. Brown, Gastroenterologist, Cleveland Clinic, Cleveland.
Discussion, Eosinophilic Enteritis Syndrome, Section on Digestive Diseases, Sunday, 10:30 a.m.

Mrs. G. Bedford Brown, Auxiliary State Chairman for Memorial Service, Angleton.
Memorial Address for Deceased Members of Woman's Auxiliary, Memorial Service, Sunday, 11:30 a.m.

George T. Bryan, Assistant Professor in Pediatrics; Assistant Director, Clinical Study Center, University of Texas Medical Branch, Galveston.
Current Methods for the Evaluation of Aldosterone Secretion, Texas Diabetes Association, Sunday, 11:40 a.m.

Rep. Bob Casey, 22nd Congressional District, Houston.
Address, TexPac Breakfast, Saturday, 7 a.m.

Charles E. Cash, D.D.S., Pedodontist, Fort Worth.
Panel: Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 11 a.m.

Lt. Col. Ray G. Cowley, Chief, Pulmonary Disease Service, Brooke General Hospital, Fort Sam Houston.
Bronchogenic Carcinoma in Young Patients, Texas Chapter, American College of Chest Physicians, Sunday, 9 a.m.

John D. Daugherty, Federal Aviation Agency Regional Flight Surgeon, Fort Worth.
Local Air Safety Should Use the Epidemiological Approach, Texas Air-Medics Association, Sunday, 9 a.m.
Moderator, Federal Aviation Agency Panel Discussion, Texas Air-Medics Association, Sunday, 10:30 a.m.

Arnold Drake, National Institutes of Health Training Fellow in Gastroenterology, University of Texas Medical Branch, Galveston.
Collagen Disease Primarily Affecting the Gastrointestinal Tract, Section on Digestive Diseases, Sunday, 10 a.m.

Charles D. Dukes, Ph.D., Baylor University College of Medicine, Houston.

Curbstone Consultation: Vaginitis, Friday, 8 p.m.

Tom Dutcher, Clinical Pathologist, Department of Pathology, University of Texas Southwestern Medical School, Dallas.
Panel: Coagulation Problems in the Laboratory, Section on Pathology, Friday, 3:05 p.m.

Robert E. Eakin, Ph.D., Professor of Chemistry, University of Texas; Research Scientist, Clayton Foundation Biochemical Institute, University of Texas, Austin.
Metabolic Mechanisms in Cells: The Unity in Biochemistry, Symposium on Nutrition, Friday, 2:41 p.m.
Question and Answer Panel, Symposium on Nutrition, Friday, 4:50 p.m.

Marvin Erons, Resident, University of Texas Medical Branch, Galveston.
Production of Carcinoma in Burn Scars, Texas Society of Plastic Surgeons, Thursday, 10:45 a.m.

Willson Fahlberg, Ph.D., Associate Professor of Micro-Biology, Baylor University College of Medicine, Houston.
Panel: Use and Abuse of Gamma Globulin, Section on Pediatrics, Friday, 4 p.m.

Donald P. Ford, Clinical Assistant Professor of Ophthalmology, Baylor University College of Medicine, Houston.
Carbohydrate and Vascular Defects in Families of Known Diabetics, Texas Diabetes Association, Sunday, 2:40 p.m.

Mr. Philip W. Gauss, Jr., Administrative Assistant, Commissioner of Health for Texas, Austin.
The Medical Self-Help Program, Symposium on Disaster Medical Care, Friday, 9:35 a.m.

Armond Goldman, Assistant Professor of Pediatrics, University of Texas Medical Branch, Galveston.
Panel: Use and Abuse of Gamma Globulin, Section on Pediatrics, Friday, 4 p.m.

Luis Amezcua Gonzales, Chief, Department of Aviation Medicine, Mexico, D. F.
Aeromedical Indoctrination for Flight Crews, Texas Air-Medics Association, Saturday, 11:30 a.m.

Morton I. Grossman, Editor, *Gastroenterology*, and Senior Medical Investigator, Veterans Administration Center, Los Angeles, Calif.
Panel: Symposium on Newer Aspects of Treatment of Peptic Ulcer Disease, Section on Digestive Diseases, Sunday, 10:45 a.m.

Mr. Bobby Gunn, Athletic Trainer, Department of Intercollegiate Athletics, Lamar State College of Technology, Beaumont.
Training the High School Athlete, Texas Society of Athletic Team Physicians, Thursday, 3 p.m.

Mr. George E. Haddaway, Editor, *Flight Magazine*, Dallas.
Where Do We Go From Here? Texas Air-Medics Association, Saturday, 10 a.m.

Capt. Robert E. Harris, United States Air Force Hospital, Lackland Air Force Base.
Varicella Pneumonia in Adults, Texas Chapter, American College of Chest Physicians, Sunday, 9 a.m.

Rufus Hessberg, Crews Systems Division, Manned Spacecraft Center, National Aeronautics and Space Administration, Houston.
Dinner Address, Texas Society of Anesthesiologists, Saturday, 8 p.m.

Donald Hull, D.D.S., Houston.
Three Great Thrills of Life, Texas Chapter, Flying Physicians Association, Thursday, 7 p.m.

Ira Iscoe, Ph.D., Professor of Psychology, University of Texas, Austin.
Panel: Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 11 a.m.
The Comprehensive Psychological Assessment of Cerebral Palsied Children, Symposium on Cerebral Palsy, Saturday, 3:15 p.m.

Harold G. Jacobson, Chief, Division of Diagnostic Radiology, Montefiore Hospital, Bronx, N. Y.
Some Roentgen Aspects of Pituitary and Para-Pituitary Lesions, Section on Radiology, Friday, 4:30 p.m.

Cone Johnson, Consultant in Clinical Pulmonary Medicine, Scott and White Memorial Hospital, Temple.
Moderator, Texas Academy of General Practice Seminar, Thursday.
Office Pulmonary Function Evaluation, Texas Academy of General Practice Seminar, Thursday, 3:05 p.m.
Curbstone Consultation: Inhalation Therapy in Lung Disease, Friday, 8 p.m.

Joseph B. Kirsner, Professor of Medicine and Chief, Department of Gastroenterology, University of Chicago, Chicago.
Discussion, Eosinophilic Enteritis Syndrome, Section on Digestive Diseases, Sunday, 10:30 a.m.

James W. McBee, Gastroenterologist, Veterans Administration Hospital, Houston.
Eosinophilic Enteritis Syndrome, Section on Digestive Diseases, Sunday, 10:20 a.m.

Gordon McHardy, Clinical Professor of Medicine, Louisiana State University School of Medicine, New Orleans.
Moderator, Panel: Symposium on Newer Aspects of Treatment of Peptic Ulcer Disease, Section on Digestive Diseases, Sunday, 10:45 a.m.

Robert A. McNaughton, Clinical Assistant Professor of Medicine, University of Miami, Miami, Fla.
Discussion, Radiation Treatment in Reflux Esophagitis, Section on Digestive Diseases, Sunday, 8:30 a.m.

F. G. Marsh, Brooke General Hospital, Fort Sam Houston.
Porphyria Cutanea Tarda in the American Negro, Texas Dermatological Association, Saturday, 11:15 a.m.

Joseph L. Melnick, Ph.D., Professor of Epidemiology and Serology, Baylor University School of Medicine, Houston.
Curbstone Consultation: Newer Concepts in Immunizations, Friday, 8 p.m.

George R. Meneely, Professor of Nuclear Biochemistry, Baylor University Graduate Branch, M. D. Anderson Hospital, Houston.

Curbstone Consultation: Cardiac Emergencies, Friday, 8 p.m.

Jordan Miller, Resident in Plastic Surgery, Houston.
Elastic Splint Construction with Dental Silicone Impression Material, Texas Society of Plastic Surgeons, Thursday, 11:55 a.m.

Stanley R. Mohler, Director, Civil Aeromedical Research Institute, Oklahoma City.
Recent Medical Causes of Light Plane Crashes, Texas Air-Medics Association, Saturday, 10:45 a.m.

Col. John A. Moncrief, Instructor in Surgery, Emory University School of Medicine, Atlanta.
Discussion, Orthopedic Care of the Severely Burned Patient, Texas Orthopaedic Association, Saturday, 3:20 p.m.

Capt. Everett Rhoades, USAF (MC), Chief, Pulmonary and Infectious Disease Service, United States Air Force Hospital, Lackland Air Force Base.
Chemotherapy of Respiratory Infections, Texas Academy of General Practice Seminar, Thursday, 4:35 p.m.

Bettye A. Sayle, University of Texas Medical Branch, Galveston.
Curbstone Consultation: Use of Oral Agents in Diabetes Mellitus, Friday, 8 p.m.

Holbrooke S. Seltzer, Chief, Metabolic Service, Veterans Administration Hospital; Associate Professor of Medicine, University of Texas Southwestern School of Medicine, Dallas.
Discussion, Metabolic Mechanisms in Cells: The Unity in Biochemistry, Symposium on Nutrition, Friday, 3:11 p.m.
Insulogenic Response to Hyperglycemia After Chronic Treatment with Sulfonylurea, Texas Diabetes Association, Sunday, 2:20 p.m.

Albert L. Shirkey, Department of Surgery, Baylor University College of Medicine, Houston.
Surgical Treatment of Traumatic Wounds of the Heart and Blood Vessels, Texas Industrial Medical Association, Texas Physical Medicine and Rehabilitation Society, and Texas Traumatic Surgical Society, Thursday, 12:45 p.m.

Col. Ingalls H. Simmons, Chief, Preventive Medicine Branch, Fort Sam Houston.
Sanitation in Disaster, Symposium on Disaster Medical Care, Friday, 10:45 a.m.
Question and Answer Panel, Symposium on Disaster Medical Care, Friday, 11:20 a.m.

Lt. Col. J. R. Smith (MC), United States Air Force Hospital, Lackland Air Force Base.
Kidney Transplantation, Texas Society of Plastic Surgeons, Thursday, 9:45 a.m.

Col. John P. Stapp, Deputy Chief Scientist, Headquarters, Aerospace Medical Division, Brooks Air Force Base.
Moderator, Panel: Aerospace Medical Division, Brooks Air Force Base, Texas Air-Medics Association, Saturday, 2 p.m.

Frederick Steigmann, Director, Therapeutics and Gastroenterology, Cook County Hospital, Chicago; Associate Professor of Medicine, University of Illinois College of Medicine, Chicago.
Discussion, One Hundred Patient-Day Years of Anticholinergic Therapy, Section on Digestive Diseases, Sunday, 8:45 a.m.

Edward Teller, Ph.D., Director, Lawrence Radiation Laboratory, University of California, Berkeley, Calif. Operation Plowshare and Peacetime Uses of Atomic Energy, The Teller Lecture, Sunday, 12:45 p.m.

Robert P. Wagner, Ph.D., Professor of Zoology, University of Texas; Director, Genetics Foundation, University of Texas, Austin.
The Relation Between Genes and Proteins and Its Significance, Symposium on Nutrition, Friday, 4:11 p.m.
Question and Answer Panel, Symposium on Nutrition, Friday, 4:50 p.m.

Roger J. Williams, Ph.D., Professor of Chemistry, University of Texas; Research Scientist and Advisor, Clayton Foundation Biochemical Institute, Austin.
Cellular Nutrition and the Genetotrophic Principle, Symposium on Nutrition, Friday, 3:17 p.m.
Question and Answer Panel, Symposium on Nutrition, Friday, 4:50 p.m.

William G. Wolfe, Ph.D., Chairman of the Board, United Cerebral Palsy Association of Texas, Inc., Austin.
Moderator, Panel: Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy, Symposium on Cerebral Palsy, Saturday, 11 a.m.
Summation, Symposium on Cerebral Palsy, Saturday, 4:15 p.m.

Mr. Bill Yeoman, Head Coach, Athletic Department, University of Houston, Houston.
The Attitude of the Coach, Texas Society of Athletic Team Physicians, Thursday, 1:50 p.m.

Memorial Service

Sunday, April 26, 11:30 a.m.

Continental Room, Shamrock Hilton Hotel

Jared E. Clarke, M.D., Houston, Chairman.

Mrs. G. Bedford Brown, Angleton, Cochairman.

1. Organ Prelude. Mrs. Fred J. Stancliff, Houston.

2. Invocation. Dawson C. Bryan, D.D., Institute of Religion, Houston.

3. Special Music. Mrs. William C. Owsley, Jr., Houston.

4. Memorial Address for Deceased Members of Woman's Auxiliary. Mrs. G. Bedford Brown, Angleton.

5. Memorial Address for Deceased Physicians. Jared E. Clarke, M.D., Houston.

6. Special Music. Mrs. Owsley.

7. Benediction. Dr. Bryan.

8. Organ Postlude. Mrs. Stancliff.

General Meetings

Friday, April 24, 10 a.m.

Emerald Room, Shamrock Hilton Hotel

Robert Mayo Tenery, Waxahachie, President, Presiding.

1. (10:00) **Nutrition in Cardiovascular Disease.**
Fredrick J. Stare, Boston.

Improper nutrition is one of the important causative factors in cardiovascular disease, particularly those that are associated with atherosclerosis. Total caloric intake, fat and type of fat, carbohydrates and type of carbohydrate, and cholesterol content of the diet are among the nutritional factors involved. These will be discussed as they relate to various specific studies.

2. (10:30) **Present Day Management of Diverticulitis.**
Patrick H. Hanley, New Orleans.

3. (11:00) **Low Back Pain. (Closed-Circuit Television Presentation)**
Mark B. Coventry, Rochester, Minn.

Saturday, April 25, 10 a.m.

Emerald Room, Shamrock Hilton Hotel

William H. Gordon, Lubbock, Vice President, Presiding.

1. (10:00) **Diabetic Nephropathy.**
Max Ellenberg, New York.

Diabetic nephropathy represents a combination of glomerular, tubular, and infectious involvement. The nephrotic syndrome occurs in a minority of cases and has a poor correlation with the presence of the Kimmelstiel-Wilson lesion of nodular glomerulosclerosis. The K-W lesion is specific for the pathological documentation of diabetes. Its relationship to control is questionable and it may even occur without overt diabetes. This supports the concept that diabetes is a generalized multifaceted syndrome.

2. (10:30) **Chronic Cystic Mastitis.**
Nathan A. Womack, Chapel Hill, N. C.

3. (11:00) **Medical Support of Man in Space. (Closed-Circuit Television Presentation)**
Charles A. Berry, Houston.

GENERAL MEETING LUNCHEON

Friday, April 24, 12:30 p.m.

Continental Room, Shamrock Hilton Hotel

William H. Gordon, Lubbock, Vice President, Presiding.

1. (12:30) **Invocation.**
Edwin Goodall, Breckenridge.
Luncheon.
Recognition:
Past Presidents Association.

2. (1:10) **Presentation of second edition of work by Andreas Vesalius on anatomy published in 1555.**
Hampton C. Robinson, Houston, and William M. Crawford, Fort Worth.

3. (1:15) **Announcement of Anson Jones Awards.**
Glenn D. Carlson, Dallas, Chairman Council on Public Relations and Public Service.

4. (1:20) **Introduction of Speaker.**
Ralph G. Greenlee, Midland.

5. (1:25) **Nutritional Nonsense.**
Fredrick J. Stare, Professor and Chairman, Department of Nutrition, School of Public Health, Harvard University, Boston.

There is more sense in nutrition than nonsense, though sometimes one wonders if this is so. Food quacks and health charlatans abound from Texas to Massachusetts, and going the other way geographically, they seem to concentrate in the pleasant climate of Arizona and Southern California. Peddling their wares of organically fertilized foods, sea salts, natural vitamins, and honey and vinegar, they do a thriving business on the gullibility of mankind in his quest for health.

ANNUAL LUNCHEON AND INSTALLATION OF OFFICERS

Saturday, April 25, 12:30 p.m.

Continental Room, Shamrock Hilton Hotel

Robert Mayo Tenery, Waxahachie, President, Presiding.

1. (12:30) **Invocation.**
Howard O. Smith, Marlin.
Luncheon.
Recognition:
Fifty Year Club.
Registrants for Orientation Program.
Civic Guests.

2. (1:10) **Announcement of Scientific Exhibits Awards.**
Jasper H. Arnold, Houston, Chairman, Committee on Scientific Exhibits.

3. (1:15) **Installation of Officers of Texas Medical Association for 1964-65.**

4. (1:35) **Introduction of Speaker.**
Denton Kerr, Houston.

5. (1:40) **It's an Amazing Story.**
Edward R. Annis, Miami, Fla., President, American Medical Association.

PARTICIPATE IN CURBSTONE CONSULTATIONS

Friday, 8 p.m., Garden and Grecian Rooms,

Shamrock Hilton Hotel

Refresher Courses

The refresher course program for the 1964 annual session includes 14 courses, each consisting of approximately 45 minutes of lecture plus 30 minutes of questions and answers. The courses, presented by out-of-state medical leaders, are primarily for the benefit of physicians in general practice, but also are of value to specialists. Hour-for-hour accreditation will be granted by the Texas Academy of General Practice for each course.

The schedule runs from 8:30 to 9:45 a.m., Friday and Saturday, each course being complete in a single day. All courses are scheduled in the Shamrock Hilton Hotel. No tickets will be required for attendance at courses.

Following is the list of courses and instructors, together with meeting place for each course:

Friday, April 24, 8:30-9:45 a.m.

1. **Functional Bleeding and Secondary Amenorrhea.**
Willard M. Allen, St. Louis.
(Hide-away, Shamrock Hilton Hotel)

2. **Clinical Management of Defensive Thinking.**
R. G. Bell, Willowdale, Ontario, Canada.
(Walnut Room, Shamrock Hilton Hotel)

Defensive thinking in addiction occurs whenever the abnormal need or dependence is strong enough to perpetuate the addiction in spite of the victim's awareness of progressively serious problems in any or all areas—physical, psychological, and social. Many addicts who become locked into defensive thinking cannot possibly free themselves from its control without help. The widespread belief that one should wait until the alcohol or drug addict wants help dooms many of the victims to unnecessary and premature death. Physicians and others can learn to identify the main causes of defensive thinking in each case and help the patient to get started on a treatment program.

3. **Inflammatory Lesions of the Intestinal Tract.**
Charles L. Eckert, Albany, N.Y.
(Nile Room, Shamrock Hilton Hotel)

4. **Anal Fissures, Abscesses and Fistulas.**
Patrick H. Hanley, New Orleans.
(Castilian A, Shamrock Hilton Hotel)

5. **The Diagnosis and Management of Selected Common Skin Diseases.**
Clarence S. Livingood, Detroit.
(Venetian Room, Shamrock Hilton Hotel)

6. **The Management of the Patient with an Arrhythmia.**
Oglesby Paul, Chicago.
(Castilian C, Shamrock Hilton Hotel)

7. **Early Treatment of Common Eye Injuries. (Primarily for Nonophthalmologists.)**
Brittain F. Payne, New York.
(Grecian Room, Shamrock Hilton Hotel)

8. **Evaluation of the Dizzy Patient.**
G. O. Proud, Kansas City, Kan.
(Normandy A and B, Shamrock Hilton Hotel)

Saturday, April 25, 8:30-9:45 a.m.

9. **Specialized Roentgen Techniques in the Diagnosis of Abdominal Disease.**
John A. Evans, New York.
(Hide-away, Shamrock Hilton Hotel)

10. **The Differential Diagnosis of Vertigo.**
D. Bernard Foster, Topeka, Kan.
(Normandy A, Shamrock Hilton Hotel)

11. **Anemias Not Responding to Ordinary Hematinics.**
Albert G. Hagedorn, Rochester, Minn.
(Venetian Room, Shamrock Hilton Hotel)

12. **The General Principles in the Interpretation of Test of Gastric Function.**
John N. Hunt, London.
(Normandy B, Shamrock Hilton Hotel)

The anatomical and physiological background tests of gastric function will be given. The interpretation of such parameters as concentration of acid, volume of secretion, concentration of pepsin, response to maximal doses of histamine, ratio of pepsin to acid, etc., will be discussed.

13. **Deceptive Diagnosis in Lesions of the Ocular Fundus. (Primarily for Ophthalmologists.)**
H. Saul Sugar, Detroit.
(Grecian Room, Shamrock Hilton Hotel)

14. **Respiratory Emergencies in the Newborn.**
Watts R. Webb, Dallas.
(Walnut Room, Shamrock Hilton Hotel)

Respiratory distress in the newborn often creates a critical emergency, which in many instances can be resolved by prompt medical or surgical therapy. The major facets of this problem will be presented along with diagnostic clues from representative cases.

Curbstone Consultations

Ten informal, unrehearsed, round table discussions, sponsored by the Texas Medical Association, will be held Friday, 8-9:30 p.m., in the Grecian and Garden Rooms of the Shamrock Hilton Hotel. All registrants are invited to attend.

Physicians attending the sessions are encouraged to ask questions and to comment on the various aspects of each subject under discussion. Physicians are encouraged to bring case histories and to obtain the opinion of the discussion leaders. Physicians may move from one table to another, if and when they wish. Refreshments will be served on a complimentary basis.

Topics, moderators, and discussion leaders are listed below. Tables 1-6 will be placed in the Grecian Room. Tables 7-10 will be placed in the Garden Room.

Table 1. **Present Status of Surgery of the Heart.**
Hugh E. Wilson, Dallas, Moderator.
Robert H. LePere, San Antonio.

R. Maurice Hood, Austin.
Watts R. Webb, Dallas.
Denton A. Cooley, Houston.

Table 2. Inhalation Therapy in Lung Disease.
Robert J. Carabasi, Temple, Moderator.
Ivan E. Cushing, Dallas.
George S. Schools, Dallas.
Cone Johnson, Temple.
Theodore J. Haywood, Houston.

Table 3. Modern Concepts in the Management of the Postmenopausal Patient.
John C. Hundley, Fort Stockton, Moderator.
Charles A. Stephens, Odessa.
Haden E. McKay, Jr., Humble.
Harry A. Tubbs, Post.
Garland H. Lang, Midland.

Table 4. Use of Oral Agents in Diabetes Mellitus.
Raymond L. Gregory, Galveston, Moderator.
Harold L. Dobson, Houston.
Alfred L. Lane, Corpus Christi.
Leonard R. Robbins, Houston.
Bettye A. Sayle, Galveston.

Table 5. Backache.
Paul C. Williams, Dallas, Moderator.
Isaac S. McReynolds, Houston.
Louis J. Levy, Fort Worth.
Marvin P. Knight, Dallas.
Richard H. Eppright, Houston.
Robert A. Murray, Temple.

Table 6. Vaginitis.
Herman L. Gardner, Houston, Moderator.
William F. Guerriero, Dallas.
E. B. Mendel, Dallas.
Daniel H. Eames, LaMarque.
Charles D. Dukes, Ph.D., Houston.

Table 7. Newer Concepts in Immunizations.
Martha D. Yow, Houston, Moderator.
Quellin T. Box, Galveston.
Joseph L. Melnick, Ph.D., Houston.
Fred S. Brooksaler, Dallas.
A. Aaron Mintz, Houston.
Fred T. Matthes, Bay City.

Table 8. Adolescent Problems.
Victor C. Calma, Galveston, Moderator.

Table 9. Psychiatric Problems in Clinical Practice.
Shervert H. Frazier, Houston, Moderator.
William A. Cantrell, Houston.
Harry K. Davis, Galveston.
Jerry M. Lewis, Dallas.
Robert L. Stubblefield, Dallas.
Robert B. White, Galveston.

Table 10. Cardiac Emergencies.
Robert E. Leslie, El Campo, Moderator.
James K. Alexander, Houston.
John R. Derrick, Galveston.
Alfred W. Harris, Dallas.
George C. Morris, Jr., Houston.
George R. Meneely, Houston.

Section Meetings

SECTION ON DIGESTIVE DISEASES

Chairman—John R. Kelsey, Jr., Houston.

Secretary—Marcel Patterson, Galveston.

Saturday, April 25, 2:30-5 p.m.

Azalea and Bluebonnet Rooms, Shamrock Hilton Hotel

Joint Session: Sections on Digestive Diseases, Internal Medicine, Pathology, Radiology and Surgery.

1. (2:30) Panel: Diseases of the Thyroid.
Moderator: Mavis P. Kelsey, Houston.
Panelists: A. Stone Freedberg, Boston, Internal Medicine.
Charles L. Eckert, Albany, N. Y., Surgery.
Walter T. Murphy, Buffalo, Radiology.
William O. Russell, Houston, Pathology.
S. Raymond Gambino, Englewood, N. J., Pathology.

2. (3:50) Panel: Gastrointestinal Polyps.
Moderator: George L. Jordan, Jr., Houston.
Panelists: Albert B. Hagedorn, Rochester, Minn., Internal Medicine.
John A. Evans, New York, Radiology.
Nathan A. Womack, Chapel Hill, N. C., Surgery.
Harlan J. Spjut, Houston, Pathology.

Sunday, April 26, 8:30 a.m.-12:15 p.m.

Garden Room, Shamrock Hilton Hotel

3. (8:30) Radiation Treatment in Reflux Esophagitis.
Ralph D. Eichhorn, Houston.

Gastric reflux is responsible for the pain of distal esophagitis, with and without associated hiatal hernia, and for the distal esophageal erosion, ulceration, and probably bleeding. Standard treatment for gastric reflux is buffering of the gastric content, anticholinergic therapy, diet, and elevation of the head of the bed. This is a plea for an extension of radiation therapy to decrease gastric acid pepsin production. Several case presentations where this treatment has been beneficial will be presented.

Discussion: Robert A. McNaughton, Miami.

4. (8:45) One Hundred Patient-Day Years of Anticholinergic Therapy.
Ivan E. Danhof, F. Clark Douglas, Milford O. Rouse, Cecil O. Patterson, Herbert A. Bailey and John W. Fisher, Dallas.

Five anticholinergic agents (atropine sulfate, pentapiperide methyl sulfate, oxyphencyclimine hydrochloride, propantheline bromide, and glycopyrrolate) were evaluated using the double blind technique in 206 patients with well-documented peptic ulcerative disease of the duodenum. Comparative appraisal of the five agents studied will be delineated. The study represents an accumulative experience with anticholinergic medication in excess of 110 years of patient-days of therapy.

Discussion: Frederick Steigmann, Chicago.

5. (9:00) **Duodenal Receptors.**
John N. Hunt, London.

The duodenum is the meeting place of the gastric contents, the bile, the pancreatic secretions and the secretions of Brunner's glands. The mixing which occurs there is not haphazard; the flow of each of the components is regulated, mainly through reflex arcs activated by receptors in the walls of the duodenum. The properties of these receptors as discovered in man will be discussed.

(9:30) **Intermission.**

6. (10:00) **Collagen Disease Primarily Affecting the Gastrointestinal Tract.**
Arnold Drake, Edward Lefeber and Marcel Patterson, Galveston.

Uncommonly, the gastrointestinal tract is the main target organ of a collagen disease; other manifestations of the disease being slight or absent. These physicians have seen two such patients. The problems in recognition and management will be presented.

7. (10:10) **Primary Nonspecific Ulcer of the Small Intestine.**
Belton G. Griffin, Houston.

The syndrome of primary nonspecific ulcer of the small intestine is characterized in its early stages by no symptoms or vague and nonspecific abdominal complaints. The diagnosis is usually not made until one of the complications of obstruction, perforation, or hemorrhage occur. Five cases of ulcer of the small intestine, each causing a partial small bowel obstruction, will be described. In two, a possible cause of the ulcer was apparent.

8. (10:20) **Eosinophilic Enteritis Syndrome.**
Philip S. Bentlif and James W. McBee, Houston.

Study of several patients who have diffuse eosinophilic enteritis has been made. These patients have demonstrated a fairly well-defined pattern of symptoms, course of disease, and response to therapy, with small bowel biopsy picture of diffuse eosinophilic infiltration of the jejunal submucosa and mucosa. The syndrome will be discussed on the basis of material obtained from patients and from those described in the literature.

Discussion: Joseph B. Kirsner, Chicago; Charles Brown, Cleveland, Ohio; and Henry Laurens, Jr., Temple.

9. (10:45) **Symposium on Newer Aspects of Treatment of Peptic Ulcer Disease.**
Moderator: Gordon McHardy, New Orleans.
Panelists: Nathan A. Womack, Chapel Hill, N. C., Surgery.
George L. Jordan, Jr., Houston, Surgery.
Morton I. Grossman, Los Angeles, Gastroenterology.
John N. Hunt, London, Physiology.

(12:15) **Adjourn.**

1 p.m.

Criterion Club, 16th Floor, Fannin State Bank Building, 1020 Holcombe Blvd.

10. (1:00) **Luncheon.**

ALUMNI EVENTS, CLASS REUNIONS, FRATERNITY PARTIES
Plan to Attend. See list on page 263.

SECTION ON GENERAL PRACTICE

Chairman—Harry A. Tubbs, Post.
Secretary—George A. Burgess, Eagle Pass.

Friday, April 24, 2:30-5 p.m.
Emerald Room, Shamrock Hilton Hotel

1. (2:30) **Rehabilitation in Instances of Hearing Loss.**
James L. Smith, Houston.

Many persons, previously disabled by incapacitating hearing loss, can now be restored to normal hearing through modern surgical techniques. Others can be aided through auditory and speech training, allowing them to live useful, active lives. To the family physician falls the responsiblity of recognizing hearing loss in early childhood when rehabilitation measures are most successful and irreparable personality damage has not occurred.

2. (3:00) **The Postconcussion Syndrome and the Convulsive Equivalents.**
Lewis M. Helfer, San Antonio.

Because of the difficulty in "proving" such symptoms as headache, dizziness, forgetfulness, difficulty in concentration, blacking or blanking-out, tiredness, and changes in personality in persons who have had a head injury, the patient may be looked upon with suspicion as exaggerating symptoms for personal gain. A linking of these symptoms with those known to be associated frequently with the convulsive disorders can be shown to exist and consequent rational treatment offered.

Discussion: John L. Wright, Jr., Big Lake.

3. (3:30) **New Experiences with Mammography.**
Alfred G. Swearingen, Houston.

One thousand consecutive roentgenographic examinations of the breast, performed at the University of Texas M. D. Anderson Hospital and Tumor Institute between July, 1962, and May, 1963, and interpreted by the author, have been reviewed. The series was comprised of 514 patients. The radiographic criteria of benign and malignant lesions of the breast are evaluated with reference to the experience of this series.

Discussion: John E. Martin, Houston.

4. (4:00) **Measles Vaccines.**
Saul Krugman, New York.

The licensing of measles vaccine on March 21, 1963, was a significant event in the history of American medicine. During the past year, several million children have received live attenuated measles vaccine and gamma globulin, inactivated measles vaccine, or a combination of both killed and live vaccines. The recent development of a live further attenuated measles vaccine will obviate the need for gamma globulin. The virtues and limitations of the various measles immunization regimens will be discussed in detail.

5. (4:30) **Menopause.**
Don G. Harrel, Dallas.

With increasing frequency the questions are being asked, "Is the menopause physiological or pathological? If it is pathological should it be treated as a deficiency disease?" The evidence will be examined, and the advantages and pitfalls of the routine use of estrogen, estrogen-testosterone, and estrogen-progesterone mixtures will be considered. The questions of possible carcinogenesis and the problem of the production of withdrawal bleeding will be examined.

Saturday, April 25, 2:30-5 p.m.
Emerald Room, Shamrock Hilton Hotel

6. (2:30) **Laboratory Services for the Individual Practitioner.**

Jarrett E. Williams, Abilene.

The secret of excellence in laboratory service concerns the selection of competent personnel, equipment, and quarters. An overriding secret is the employment of a technique known as quality control which enables any laboratory, large or small, to develop a reproducible norm in all phases of its work for comparison with abnormal findings.
This paper is an attempt to share some thoughts concerning those laboratory services which can be performed in the office, small laboratory, or in larger reference laboratories. Medical ethics in laboratory work cannot be overlooked.

7. (3:00) **The Physician and the Rash.**

Saul Krugman, New York.

The physician who is confronted by a patient with a rash is charged with a grave responsibility. A correct diagnosis may have a significant effect on the patient, on the immediate contacts, and on the community. For example, early recognition of the rash of meningococcemia may avert a potential fatality. An erroneous diagnosis of rubella in pregnancy will cause unnecessary anxiety and possible pressure for an unnecessary therapeutic abortion. In this era of jet travel the risk of imported smallpox is significant. Small outbreaks of the disease occurred in Great Britain in 1961 and 1962. The presentation will include a discussion of more than 100 Kodachrome slides of rashes seen by the speaker in this country and abroad.

8. (3:30) **High Risk Babies.**

Margaret H. Jones, Los Angeles.

9. (4:00) **Diagnosis of the Prediabetic State.**

Max Ellenberg, New York.

Prediabetes may be defined as that period in the life of the patient that begins at birth and ends with the discovery of diabetes. Clinical awareness of the presence of complications in the absence of manifest diabetes, plus studies of metabolic and structural derangements in prediabetes, all indicate that this is not a dormant period, but rather is an active, dynamic phase. This concept represents a significant and highly promising breakthrough in diabetes.

10. (4:30) **Pediatric Cardiovascular Surgical Emergencies.**

Grady L. Hallman, Jr., Houston.

Congenital anomalies of the heart and great vessels may cause acute cardiac failure and hypoxemia of such severity in infancy that they offer an immediate threat to survival. Indeed, studies have shown that without surgical therapy, 60 per cent of infants with these anomalies will die during the first year of life. Experience with 500 operations, many on an emergency basis, in infants less than one year of age with cardiovascular anomalies has demonstrated that gratifying results can be obtained, and places the burden of early diagnosis on the general practitioner and pediatrician. The report will concern diagnosis and therapy in a group of patients many of whom were previously considered unsalable.

SECTION ON INTERNAL MEDICINE

Chairman—Raymond D. Pruitt, Houston.

Secretary—Horace E. Cromer, Austin.

Friday, April 24, 2:30-5 p.m.

Bluebonnet Room, Shamrock Hilton Hotel

1. (2:30) **The Patient with Severe Heart Failure.**

Oglesby Paul, Chicago.

2. (3:00) **The Laboratory Diagnosis of Coronary Artery Disease and Insufficiency.**

A. Stone Freedberg, Boston.

3. (3:30) **Hematological Clues to Malignant Disease.**

Albert B. Hagedorn, Rochester, Minn.

4. (4:00) **Pathological Obesity and Psychopathology.**

Sydney G. Margolin, Denver.

5. (4:30) **Recognition of the Psychotic Patient.**

Shervert H. Frazier, Jr., Houston.

Saturday, April 25, 2:30-5 p.m.

Azalea and Bluebonnet Rooms,
Shamrock Hilton Hotel

Joint Session: Sections on Internal Medicine, Digestive Diseases, Pathology, Radiology, and Surgery.

6. (2:30) **Panel: Diseases of the Thyroid.**

Moderator: Mavis P. Kelsey, Houston.

Panelists: A. Stone Freedberg, Boston, Internal Medicine.
Charles L. Eckert, Albany, N. Y., Surgery.
Walter T. Murphy, Buffalo, Radiology.
William O. Russell, Houston, Pathology.
S. Raymond Gambino, Englewood, N. J., Pathology.

7. (3:50) **Panel: Gastrointestinal Polyps.**

Moderator: George L. Jordan, Jr., Houston.

Panelists: Albert B. Hagedorn, Rochester, Minn., Internal Medicine.
John A. Evans, New York, Radiology.
Nathan A. Womack, Chapel Hill, N. C., Surgery.
Harlan J. Spjut, Houston, Pathology.

SECTION ON OBSTETRICS AND GYNECOLOGY

Chairman—Stephen W. Cobb, Dallas.

Secretary—Wayne F. Baden, Temple.

Friday, April 24, 2:30-5 p.m.

Continental Room, Shamrock Hilton Hotel

1. (2:30) **Precocious and Delayed Puberty.**

Willard M. Allen, St. Louis.

(3:00) **General Discussion.**

2. (3:15) **Management of Obstetrical Emergencies in a Small Hospital.**

J. Collier Rucker, Jacksonville.

Today, with the application of our clinical knowledge, essentially all obstetrical emergencies can be successfully managed in any size hospital that proposes acceptance of obstetrical cases. Only basic facilities are needed, the most important one being an attending physician who is obstetrically alert. Certain basic precautions and procedures are to be reviewed. These will anticipate and prevent, or will successfully manage most obstetrical emergencies in any hospital, regardless of its size.

Discussion: Robert H. Benbow, Bryan.

Joint Meeting of Section on Obstetrics and Gynecology, Section on Surgery, and Texas Society of Anesthesiologists

Azalea Room, Shamrock Hilton Hotel

4. (4:00) **Panel: Cardiac Arrest.**
Moderator: Charles L. Eckert, Albany, N. Y.
Panelists:
Oglesby Paul, Chicago.
Kenneth K. Keown, Columbia, Mo.
Willard M. Allen, St. Louis.
J. Alan Alexander, Houston.

(4:45) **General Discussion.**

Saturday, April 25, 2:30-5 p.m.

Continental Room, Shamrock Hilton Hotel

5. (2:30) **Progestational Agents in Endometrial Carcinoma.**
Willard M. Allen, St. Louis.

(3:00) **General Discussion.**

6. (3:15) **Vaginal Cytology As An Aid in Predicting Normal Pregnancy.**
Garland H. Lang, Midland.

1) A short resumé of vaginal cytology during pregnancy and interpretations of smears by one method will be given.
2) Results of cytology smears taken during the first 16 weeks of gestation in 763 pregnancies has been reported as follows: 6 per cent of pregnancies with normal smear aborted; 7 per cent of pregnancies with borderline abnormal smear aborted; 22 per cent of pregnancies with abnormal smears aborted. Smears with a karyopyknotic index of less than 20 per cent were considered normal.
3) Hormone therapy for threatened abortion in the presence of normal smears was to no avail, all patients in this category having aborted. Fifty per cent of women with abnormal smears treated for threatened abortion with hormones continued to term.

Discussion: Raymond H. Kaufman, Houston.

Joint Meeting of Section on Obstetrics and Gynecology and Section on Pediatrics

Continental Room, Shamrock Hilton Hotel

7. (4:00) **Panel: Iatrogenic Disorders in the Fetus and Neonatal Periods.**
Moderator: C. William Daeschner, Galveston.
Panelists:
George H. Lowrey, Ann Arbor, Mich.
Myron M. Nichols, Galveston.
Robert R. Franklin, Houston.

(4:45) **General Discussion.**

PLAN TO SEE
SCIENTIFIC AND TECHNICAL EXHIBITS
SEE PAGES 303, 304 FOR DETAILS.

SECTION ON OPHTHALMOLOGY AND TEXAS OPHTHALMOLOGICAL ASSOCIATION

Chairman, Section on Ophthalmology, and President, Texas Ophthalmological Association—Louis Daily, Houston.

Secretary, Section on Ophthalmology, and Vice President, Texas Ophthalmological Association—Everett R. Veirs, Temple.

Friday, April 24, 8:30 a.m.-5 p.m.

Grecian Room, Shamrock Hilton Hotel

1. (8:30) **Early Treatment of Common Eye Injuries. (Refresher Course primarily for nonophthalmologists.)**
Brittain F. Payne, New York.

Satisfactory treatment of common eye injuries to preserve vision, to prevent scarring and insure rapid healing often depends on the initial care given the patient. The history and the visual acuity of the patient should be recorded before the physician begins his examination. The topics to be discussed will include: superficial foreign bodies of the cornea and conjunctiva, intraocular foreign bodies, contusions of the orbit, eyelids and globe, and chemical burns.

2. (10:00) **President's Address: The Texas Ophthalmological Association—A Report on Some of Its Activities.**
Louis Daily, Houston.

3. (10:30) **Pupil Block and Pupillary Block Glaucoma.**
H. Saul Sugar, Detroit.

Pupillary block following cataract surgery is due to blocking of the pupillary opening, as well as any iridectomy or iridotomy communications between the anterior and posterior chambers by postoperative inflammatory adhesions. Experience with 19 cases will be described. In 13, pupillary block glaucoma was present. The diagnosis and treatment are to be outlined, with special emphasis on one case which appears to show the relation between malignant glaucoma and pupillary block glaucoma.

(12:00) Recess.

12:30 p.m.

Castilian A, B, and C, Shamrock Hilton Hotel

4. (12:30) **Luncheon of Texas Ophthalmological Association.**

1:30 p.m.

Grecian Room, Shamrock Hilton Hotel

5. (1:30) **Business Meeting. (Members Only)**

2:30 p.m.

Grecian Room, Shamrock Hilton Hotel

6. (2:30) **Office Planning for the Ophthalmologist.**
Dan A. Russell, Jr., San Antonio.

In any office, efficiency can be increased without impairing proper patient care. Arrangement of refraction lanes and treatment rooms, systems for filing correspondence and records, and equipment and forms are discussed.

7. (3:00) Clinical Aspects of Lethal Midline Granuloma.

John E. Eisenlohr, Dallas.

Lethal midline granuloma is an uncommon and poorly understood entity characterized by progressive ulceration of midline facial structures including the nose, oral cavity, sinuses and orbits. The disease almost invariably has a fatal outcome despite vigorous efforts to arrest its progress. A description of some typical aspects of this disease, as well as some comments on therapy, will be presented.

8. (3:30) Pathological Appraisal of Retinal Detachment Operations.

Brittain F. Payne, New York.

A brief review of the normal anatomy of the retina is necessary to discuss the pathological appraisal of retinal detachment operations. The final result depends on the artificial adhesions of the retina to the choroid, the evacuation of the subretinal fluid and the shortening of the globe. The new school of retinal surgeons seem to emphasize the anatomical restoration rather than visual results. The patient is concerned with the amount of sight obtained after the mental and the physical trauma of a major surgical procedure.

(4:30) Visit Exhibits.

Saturday, April 25, 8:30 a.m.-5 p.m.

Grecian Room, Shamrock Hilton Hotel

9. (8:30) Deceptive Diagnosis in Lesions of the Ocular Fundus. (Refresher Course primarily for ophthalmologists.)

H. Saul Sugar, Detroit.

A number of difficult or confusing diagnosis pictures of the ocular fundus will be presented. Among these are macular lesions associated with pits in the optic disc, senile macular degeneration, Coats' disease, senile miliary aneurysms, vascular conditions, melanocytoma, lesions simulating tumors, and inflammatory lesions misdiagnosed as noninflammatory ones. This course will be in the nature of a course in diagnosis of clinical fundus pathology.

10. (10:00) Experiences with Fibrinolytic Agents in Treatment of Total Hyphemia.

Rufus A. Roberts, Jr., Fort Worth.

Deficiencies in the surgical treatment of total hyphemia with secondary glaucoma following blunt trauma has stimulated a revival of interest in the purified fibrinolytic agents. Animal studies and 11 clinical cases of total hyphemia treated with fibrinolytic enzymes will be reviewed.

11. (10:30) Heterochromic Cyclitis.

H. Saul Sugar, Detroit.

Heterchromic cyclitis constitutes a syndrome consisting of mild chronic cyclitis associated with heterochromia iridis, often with cataract and sometimes with glaucoma. The findings in 18 cases are used to emphasize the diagnostic features and to suggest that the condition is heredodegenerative in nature. For this reason treatment is of little help except when cataract and glaucoma are present. Both of the latter are treated as if no cyclitis were present.

(12:00) Recess.

12. (2:30) Hemangioma of the Orbit.

Walter G. Morrow, El Paso.

Hemangioma is said to be one of the commonest primary tumors found within the orbit. However, in the private practice of the average ophthalmologist, few such tumors are seen. This presentation will consist of four interesting case reports with a few comments emphasizing the problems in diagnosis and management of this condition.

Discussion: Ralph G. Berkeley, Houston.

13. (3:00) Nematode Endophthalmitis.

Ralph G. Berkeley, Houston.

An unusual case of a child with systemic larvae migrans and proven nematode endophthalmitis is to be presented, and illustrated with photographs and illustrations taken during the several years that this child was followed. A brief review of the literature on this subject will be given with emphasis on the etiology, differential diagnosis, and treatment.

14. (3:30) Operating Room Minutiae.

Sam N. Key, Jr., Austin.

This presentation is intended for the average or "occasional" ophthalmic surgeon and is designed to discuss some of the smaller details of the arrangement, conduct, assistance, instrumentation, sterilization, drug handling, etc., during routine ophthalmic operation. Various methods and techniques seen or reported elsewhere, with their possible merit or disadvantage, will be mentioned.

15. (4:00) Prevention of Complications in Cataract Surgery.

Brittain F. Payne, New York.

The prevention of complications in cataract surgery depends on the training and the experience of the surgeon. The physical and mental condition of the patient must receive careful attention. A definite preoperative program should be followed before the patient enters the hospital for additional laboratory tests and preparation for the operation. The advantages of combined general with local anesthesia should be considered carefully. Other important things to consider are: the choice of conjunctival flap and sutures, the use of alphachymotrypsin, the use of forceps or erisophake, the restoration of the anterior chamber after the closure of the wound, the dressing and the return of the patient to his room.

SECTION ON OTOLARYNGOLOGY AND TEXAS OTOLARYNGOLOGICAL ASSOCIATION

Chairman, Section on Otolaryngology—Daniel B. Powell, Jr., Austin.

Secretary, Section on Otolaryngology—Ben T. Withers, Houston.

President, Texas Otolaryngological Association—William Skokan, Fort Worth.

Vice-President and Program Chairman, Texas Otolaryngological Association—J. Patrick Moran, Corpus Christi.

Friday, April 24, 8:30 a.m.-6 p.m.

Normandy A and B, Shamrock Hilton Hotel

1. (8:30) Evaluation of the Dizzy Patient. (Refresher Course.)

G. O. Proud, Kansas City, Kan.

Man attains posture and executes voluntary movements through constant muscle tonus and delicate coordination which are controlled by the central nervous system. The nervous system receives information from informer systems such as the eyes, the touch receptors in the soles of the feet, the proprioceptive receptors in muscles, tendons and joints, and the labyrinth. Disease of the informers makes itself known in the form of unsteadiness, dizziness or light-headedness. An investigation of the vestibulocerebellar complex is called for in a patient with one of these complaints. A discussion of the anatomy and physiology of this apparatus is given. Clinical testing, symptoms, and specific diseases leading to otoneurological phenomena are discussed.

2. (10:00) Surgical Treatment and Findings in Mixed Congenital Deafness.

Woodrow D. Schlosser, Philadelphia.

During the study of congenital deafness, a certain number of cases showed a conductive element superimposed upon the sensorineural deficit. After extensive audiologic study indicated that this was a bona fide finding, surgical exploration of a series of middle ears was performed. Anatomical deformities which were found in these patients can be categorized in four groups. However, the most common anomaly was fixation of the stapes. Following surgical intervention, many of these children, on a voluntary basis, did not show any improvement in their hearing. However, on involuntary hearing tests, these patients did reveal that the conductive element had been eliminated. In some cases, months passed before voluntary responses definitely showed the elimination of the conductive element. Even then, the hearing improvement has been of little use to the patient in discriminatory functions. It appears that there are other factors involved in the processes of audition other than improvement in the function of the conductive mechanism. Although neurological examination and electroencephalographic examinations do not show any neurologic deficit, some factors appear to be operant in the central auditory system which at present are not known nor understood.

3. (10:45) Glioma of the Nose.

Donald N. Matheson, Fort Worth.

4. (11:15) Some Aspects of Tympanoplasty.

Woodrow D. Schlosser, Philadelphia.

Tympanoplastic surgery has gone through many phases since surgical concept was introduced a number of years ago by Wullstein and Zöllner. At the present time, there are many variations of this type of surgery. Questions of how much surgery, and of what type of graft are numerous and the answers appear to be the result of personal experience. It seems that no two clinicians do tympanoplastic surgery in a similar manner. Nevertheless, there is a strong tendency to individualize each case rather than do all cases of chronic otitis media in a similar manner. Thus, the procedures may involve middle ear surgery in one case, middle ear and mastoid surgery in another case, or mastoid surgery in the form of a radical mastoidectomy with a graft to the middle ear area in others.

One of the problems in tympanoplastic surgery is eustachian tube function. This has been discussed and studied by many people. Nevertheless, in Dr. Schlosser's experience, inadequate tubal function seems to be one of the major causes of failure in tympanoplastic surgery. His feeling has been for some time that chronic otitis media is a disease that starts in the eustachian tube and then involves the middle ear and the mastoid cell system.

Surgery in the past has been confined primarily to the mastoid and middle ear areas. Many cases show initial good results and these results may last for a year or more. However, as time goes by there is retraction of the graft until eventually it makes contact with the medial wall and no longer is a good vibrating surface. This result then resembles chronic adhesive otitis media with reduced hearing.

(12:00) Recess.

5. (2:30) Eustachian Tube Function Tests.

G. O. Proud, Kansas City, Kan.

A simple office test for the evaluation of eustachian tube patency and function has been devised. Although it has yielded some information on the physiologic mechanisms of the auditory tube of experimental animals, its chief value lies in its use as a method for the preoperative assessment in subjects for whom tympanoplasty is contemplated. A test solution, fluorescein, is introduced into the middle ear and sought for in the nasopharynx with ultraviolet light.

6. (3:15) Pyrogenic Reaction Following Tonsillectomy and Adenoidectomy.

Ben T. Withers and Ben M. Nail, Jr., Houston.

7. (3:45) The Treatment of the Draining Ear in Children.

Woodrow D. Schlosser, Philadelphia.

In spite of modern antibiotic therapy, children still continue to have intermittent otitis media with drainage. Many of these patients are treated with an antibiotic therapy which clears up the infection, but it may flare up in a short period of time. Additional therapy again may improve the situation with a change in antibiotics after cultures of the drainage have been taken. However, in a certain number of cases a point is reached where antibiotic therapy only suppresses the disease process. As soon as the child is taken off the antibiotic therapy without any upper respiratory infection or any initiating disease, the ear spontaneously begins to drain again. Surgical exploration of the mastoid in these cases is necessary because the site of infection is most often the mastoid cell system. A number of cases of this type have been operated upon. The operation performed was a simple mastoidectomy. In some cases after the mastoid has been cleaned, the perforation of the tympanic membrane seals itself off without any further tympanoplastic procedures. A simple mastoidectomy in an intermittent draining ear in a child still is our best weapon in our surgical armamentarium for the treatment of these cases.

8. (4:30) Business Meeting.

9. (5:00) Social Hour.

Saturday, April 25, 10 a.m.-4:15 p.m.

Normandy A and B, Shamrock Hilton Hotel

10. (10:00) Otitis Externa and Laryngoscopy in Children.

G. O. Proud, Kansas City, Kan.

Meticulous mechanical cleansing of the ear canal is probably the most important segment of the treatment of otitis externa. Surgical complete denudation of the canal epithelium will, on occasions, effect a cure when the condition has been a refractory one.

Four methods are available for laryngoscopic examination in children. Each has its limitations and advantages. A lens system inspection is a safe, inexpensive and speedy technique which may be used with outpatients.

11. (10:45) Stapedectomy: An Evaluation of Current Techniques.

William L. Draper, Houston.

12. (11:15) The Diagnosis and Treatment of Acoustic Neuroma.

Woodrow D. Schlosser, Philadelphia.

The early diagnosis of acoustic neuroma usually is an otologic diagnosis. After symptoms have been present for many years and neurologic signs such as fifth nerve paresis develop, the diagnosis is relatively easy. However, in otologic examination of patients, Dr. Schlosser believes any patient who has a unilateral sensorineural deficit with reduced speech discrimination should be considered as a possible case of acoustic neuroma unless proven otherwise. Many of these patients show tinnitus as the earliest sign, followed later on by a hearing loss and marked reduction in auditory discrimination. In the otologic diagnostic armamentarium, the caloric test usually will show either reduced function or no function of the involved labyrinth. The Békésy audiometer will many times be of some diagnostic aid. Recruitment may or may not be present. Further studies would include X-rays of the temporal bone, involving tomograms and pneumoencephalographic studies performed by the neurosurgeon. When the diagnosis of an early acoustic neuroma is made, the best method of approach is either by the lateral temporal approach or the translabyrinthine approach. If the tumor is very small and situated in the internal auditory meatus, the translabyrinthine approach would be the method of choice. When the tumor is somewhat larger, the transtemporal approach can be utilized. For extremely large tumors, the posterior fossa approach still remains the method of choice.

(12:00) Recess.

13. (2:30) Middle Ear Effusion.

G. O. Proud, Kansas City, Kan.

Eustachian tube obstruction is not an inevitable etiologic agent in middle ear effusion. The complication of cholesteatoma is not rare, and careful long range observation is essential. Only through this vigilance can the complication be recognized and dealt with early and permanent hearing loss prevented.

14. (3:15) Congenital Sensory–Neural Deafness.
Bobby R. Alford, Houston.

15. (3:45) Audio-Otology in the Office.
William P. King, Corpus Christi.

SECTION ON PATHOLOGY

Chairman—Alice Smith, Dallas.

Secretary—Jack P. Abbott, Houston.

(The Texas Society of Pathologists, Inc., will hold a business meeting Sunday, April 26, 9 a.m. to 1 p.m., Walnut Room, Shamrock Hilton Hotel.)

(The Houston Society of Clinical Pathologists will sponsor a seminar on "Respiratory System, Gastrointestinal System, Genitourinary System and Bone," Saturday, April 25, 9 a.m. to 4 p.m., in the Jesse H. Jones Medical Library Auditorium, Texas Medical Center. The seminar will be conducted by Dr. Lewis B. Woolner, professor of pathology, Mayo Foundation Graduate School of Medicine, University of Minnesota, Rochester, Minn., and by Dr. Harold G. Jacobson, chief, Division of Diagnostic Radiology, Montefiore Hospital, Bronx, N. Y.)

Friday, April 24, 2:30-5 p.m.

Castilian A, B, and C, Shamrock Hilton Hotel

1. (2:30) Screening for Clotting Abnormalities. A History Form and Basic Tests.
S. Raymond Gambino, Englewood, N. J.

2. (3:05) Panel: Coagulation Problems in the Laboratory.
Moderator: Jack P. Abbott, Houston.
Panelists: Charles F. Pelphrey, Austin.
George C. Mani, San Antonio.
Andrew D. Gwynne, Lubbock.
Tom Dutcher, Dallas.
Discussion: S. Raymond Gambino, Englewood, N. J.

3. (4:30) The Liver Panel. Differential Diagnosis of Liver Disease on the Basis of Laboratory Tests.
S. Raymond Gambino, Englewood, N. J.

Saturday, April 25, 2:30-5 p.m.

Azalea and Bluebonnet Rooms, Shamrock Hilton Hotel

Joint Session: Sections on Pathology, Digestive Diseases, Internal Medicine, Radiology and Surgery.

4. (2:30) Panel: Diseases of the Thyroid.
Moderator: Mavis P. Kelsey, Houston.
Panelists: A. Stone Freedberg, Boston, Internal Medicine.
Charles L. Eckert, Albany, N. Y., Surgery.
Walter T. Murphy, Buffalo, Radiology.
William O. Russell, Houston, Pathology.
S. Raymond Gambino, Englewood, N. J., Pathology.

5. (3:50) Panel: Gastrointestinal Polyps.
Moderator: George L. Jordan, Jr., Houston.
Panelists: Albert B. Hagedorn, Rochester, Minn., Internal Medicine.
John A. Evans, New York, Radiology.
Nathan A. Womack, Chapel Hill, N. C., Surgery.
Harlan J. Spjut, Houston, Pathlogy.

SECTION ON PEDIATRICS

Chairman—C. William Daeschner, Jr., Galveston.

Secretary—David M. Mumford, Houston.

Friday, April 24, 2:30-5 p.m.

Nile Room, Shamrock Hilton Hotel

1. (2:30) Thyrotoxicosis and Its Treatment.
George H. Lowrey, Ann Arbor, Mich.

A presentation of experiences in the treatment of 43 children with thyrotoxicosis will be given. The majority of these children have been treated by subtotal thyroidectomies. A comparison of results with those published in the literature indicates that surgical therapy is superior to medical therapy. Fewer complications and more complete remissions were found in the patients using the surgical approach.

2. (3:00) Natural History and Prevention of Infectious Hepatitis.
Saul Krugman, New York.

The control of infectious hepatitis is one of the most important unsolved problems in the field of preventive medicine. Since 1960 more than 2,000 cases of viral hepatitis have been reported in Texas each year. Progress in preventing hepatitis has been retarded by repeated failures to isolate the causative virus or viruses, an important prerequisite for the development of a vaccine. Despite these drawbacks, recent studies have added to knowledge of the natural history of infectious hepatitis and the prevention of this disease with gamma globulin.

3. (3:30) Tuberculous Meningitis in Pediatrics: A Ten-Year Experience at the Medical Branch.
Quellin T. Box, Galveston.

4. (4:00) Panel: Use and Abuse of Gamma Globulin.
Moderator: David M. Mumford, Houston.
Panelists: Willson Fahlberg, Ph.D., Houston.
Armond Goldman, Galveston.
Orville C. Thomas, Houston.

(4:45) General Discussion.

Saturday, April 25, 2:30-5 p.m.

Nile Room, Shamrock Hilton Hotel

5. (2:30) The Iatrogenic Diseases Due to Treatment of Cutaneous Diseases.
Clarence S. Livingood, Detroit.

6. (3:00) Obesity in Childhood.
George H. Lowrey, Ann Arbor, Mich.

A Metabolic Clinic was recently established in the Department of Pediatrics at the University of Michigan Medical Center. The purpose of this clinic is to treat obesity in children. An analysis of observations reveals that endocrinopathies or in-

trinsic causes of obesity are seldom encountered. A high percentage of children who are obese in adolescence have been overweight since early childhood. Earlier recognition and control of the problem is indicated.

7. (3:30) Genetic Counseling.
Robert L. Tips, Pasadena.

Joint Meeting of Section on Pediatrics and Section on Obstetrics and Gynecology

Continental Room, Shamrock Hilton Hotel

8. (4:00) Panel: Iatrogenic Disorders in the Fetus and Neonatal Periods.
Moderator: C. William Daeschner, Galveston.
Panelists: George H. Lowrey, Ann Arbor, Mich.
Myron M. Nichols, Galveston.
Robert R. Franklin, Houston.
Reba M. Hill, Houston.

(4:45) General Discussion.

SECTION ON PUBLIC HEALTH

Chairman—Albert G. Randall, Amarillo.

Secretary—William R. Ross, San Antonio.

(The Texas Association of Public Health Physicians will have a dinner meeting Thursday at 7 p.m., Nile Room, Shamrock Hilton Hotel. Conference of City and County Health Officers will be held Saturday at 2:30 p.m., Walnut Room, Shamrock Hilton Hotel. See Specialty Programs section for details.)

Friday, April 24, 2:30-5 p.m.

Walnut Room, Shamrock Hilton Hotel

1. (2:30) Some Problems and Challenges in Public Health.
Charles E. Smith, Berkeley, Calif.

2. (3:00) The Development of a Home Care Program.
Francine Jensen, Houston.

Increasing emphasis is being directed toward the development of home care, a part of total medical care, as a means of meeting the problems of long-term illnesses. The implementation of a comprehensive home care program may take years, but a local health department can initiate the development of a home care service without elaborate expansion of staff or funds.

3. (3:30) Making Maximum Use of Facilities in School Health Services.
George A. Gray, Dallas.

Organization of the Health Service in Dallas Independent School District is coordinated with several other special services: research (psychological) department, special education, etc. None of these services stand alone; they are interdependent for normal functioning. Cases of children with problems which interfere with their learning, and practical solutions for these cases, will be discussed.

4. (4:00) Report of an Outbreak of Streptococcic Sore Throat.
Neal M. Atkins, Waco.

A report on the outbreak of streptococcic sore throats with rash in school children is outlined. Investigation of the outbreak and the study of the streptococcus organisms in relation to rheumatic fever is discussed.

5. (4:30) Migrant Labor Health Program in Texas.
Carl F. Moore, Jr., Austin.

A discussion of migrant labor health problems and the dual approach for these problems: (1) the program of the central office which is primarily interested in the areas not under the jurisdiction of local health departments or local grants, and (2) the program maintained in local health departments through funds made available directly to departments for migrant laborers in their jurisdiction.

SECTION ON RADIOLOGY

Chairman—Frederick J. Bonte, Dallas.

Secretary—George F. Crawford, Beaumont.

Friday, April 24, 2:30-5 p.m.

Garden Room, Shamrock Hilton Hotel

1. (2:30) Treatment Planning in Radiotherapy.
Philip T. Hudgins, Houston.

The first step in planning radiotherapy is to define an attainable purpose of radiotherapy which, in the field of cancer, may necessarily be short of cure. Particularly with supervoltage, maximum effectiveness with safety requires that radiation be precisely directed and sharply confined to tumor-bearing area so that normal tissue can be preserved. Techniques of treatment planning and beam direction will be presented.

2. (2:50) Irradiation of Carcinoma of the Urinary Bladder.
Walter T. Murphy, Buffalo.

Many conservative surgical measures, external irradiation, radioactive implantation, and intravesical radioactive source methods are able to control a large and similar percentage of the papillary or minimally infiltrative lesions. In the patient with more invasive lesions but with no extravesical involvement, surgery has given better results than external irradiation, but not statistically significantly better results than interstitial irradiation. In extensive and deeply infiltrative lesions with extravesical involvement, neither surgery nor irradiation has been too successful to date. Although high energy radiation sources have proved to have practical as well as physical and theoretical advantages over conventional or medium energy sources, their "curative" advantage has not been statistically significant to date at Roswell Park Memorial Institute.

Preliminary clinical observations appear encouraging as to the role of combined 5-FU and radiotherapy both as an adjuvant to extirpative surgery in infiltrating carcinoma of the bladder and as a primary therapy where local invasion within the confines of the pelvis precludes definitive surgery.

3. (3:20) Contrast Arthrography of the Knee.
William P. Pride and Neill B. Longley, Houston.

This is not a new procedure. The first examinations of this sort were done in 1905; and many contrast arthrographies have been used since. These physicians' experience has been that arthrography is an easy uncomplicated 20-minute procedure which is not routinely indicated, but is done on one out of five knee cases. Drs. Pride and Longley have had experience with 87 cases, 84 of which have gone to surgery and lead them to the conclusion that this examination should be performed and interpreted by the radiologist. There were no cases which had an arthrogram and which showed lesions which were not found in surgery, but they have seen additional lesions in surgery which were not noted at the time of the arthrogram. Consistently they have noted that more damage is present in the cartilage than they have been able to translate or interpret from the arthrogram.

4. (3:40) Sclerosing Retroperitonitis and Associated Lesions.
John A. Evans, New York.

5. (4:10) **Radiotherapeutic Approach to Carcinoma of the Retromolar Trigone.**

Ralph Bowen, Houston.

This paper suggests an approach to malignant lesions in the retromolar trigone. This area includes lesions of the posterior maxillary tuberosity, tonsillar pillar, tonsillar fossa, and lateral pharynx. Any of these lesions may have extensions to the tongue, alveolus, pharynx or lingual sulcus. Operative resection is rather disfiguring and represents great sacrifices of tissue and function. The technique describes a method of precise localization. Total radiotherapy is given by interstitial implants or radioactive material complimented by external supervoltage therapy. Accurate localization and positioning utilizing multiple small treatment fields permits careful irradiation and suppression of the malignancy and sparing of the adjacent normal tissue.

6. (4:30) **Some Roentgen Aspects of Pituitary and Para-Pituitary Lesions.**

Harold G. Jacobson, Bronx, N. Y.

The plain film findings in such lesions as chromophobe adenoma, eosinophilic adenoma, craniopharyngioma, carcinoma of the pituitary, metastatic disease to the sella turcica, lymphoma involving the sella turcica, and chordoma, will be illustrated. Some special features of the pneumoencephalographic and cerebral angiographic findings in the more common intrapituitary mass lesions will be shown.

Saturday, April 25, 2:30-5 p.m.

Azalea and Bluebonnet Rooms, Shamrock Hilton Hotel

Joint Session: Sections on Radiology, Digestive Diseases, Internal Medicine, Pathology and Surgery.

7. (2:30) **Panel: Diseases of the Thyroid.**

Moderator: Mavis P. Kelsey, Houston.

Panelists: A. Stone Freedberg, Boston, Internal Medicine.
Charles L. Eckert, Albany, N. Y., Surgery.
Walter T. Murphy, Buffalo, Radiology.
William O. Russell, Houston, Pathology.
S. Raymond Gambino, Englewood, N. J., Pathology.

8. (3:50) **Panel: Gastrointestinal Polyps.**

Moderator: George L. Jordan, Jr., Houston.

Panelists: Albert B. Hagedorn, Rochester, Minn., Internal Medicine.
John A. Evans, New York, Radiology.
Nathan A. Womack, Chapel Hill, N. C., Surgery.
Harlan J. Spjut, Houston, Pathology.

SECTION ON SURGERY

Chairman—William D. Seybold, Houston.

Secretary—R. H. Baskin, Waco.

Friday, April 24, 2:30-5 p.m.

Azalea Room, Shamrock Hilton Hotel

1. (2:30) **Massive Bowel Resection.**

John L. Doyle, San Antonio.

This paper demonstrates the many compensatory mechanisms that the gastrointestinal tract undergoes following massive large and small bowel resection. Periodic radiological studies demonstrate bowel lengthening and lumen widening. Radioactive B-12 and fat studies demonstrate the importance of ileum in digestion. Suggestions in postoperative management of these patients are given. Well-oriented patients today may undergo radical bowel resections and live fairly comfortable lives with supportive regimens.

Discussion.

2. (3:00) **Therapeutic Indications in Breast Cancer.**

Charles L. Eckert, Albany, N. Y.

Discussion.

For many years radical mastectomy as independently described by Halsted and Meyer was the standard method of primary treatment of mammary cancer by all except a few skeptics who continued to use simple mastectomy. Starting with the Edinburgh experiment (McWhirter) just prior to World War II and continuing up to the present, a number of different methods of treatment have been evaluated. These include simple mastectomy, simple mastectomy and irradiation therapy, various extensions of radical mastectomy to include supraclavicular internal mammary and parasternal lymph node resections, pre- and postoperative radiotherapy, and the adjuvant use of chemotherapeutic agents. The reason for this ferment in an erstwhile complacent field was mainly the undiminished death rate from breast cancer in the face of a constant attack rate, despite the wider application of radical mastectomy by an increased number of qualified surgeons.

While it is apparent that complacency is unjustified and the need for further investigation to seek new approaches is evident from the deplorably high fatality rate from this disease, it remains necessary to fully evaluate new methods of treatment before they are accepted for clinical application.

A basic question which must be answered is concerned with whether one method of treatment applicable to all cases is preferable to selection of therapy to fit the individual case. The evidence for and against different therapeutic modalities will be critically reviewed and conclusions regarding the present status of this problem will be given.

3. (3:30) **Holes in the Lungs—Diagnosis and Management.**

Watts R. Webb, Dallas.

Patients presenting radiologic evidence of pulmonary excavation offer a wide range of diagnostic possibilities and therapeutic challenges. A rational classification and a step-wise approach to the diagnosis will be presented along with clinical experience in this regard. The diagnostic approach and management will be largely dictated by division into congenital, infectious or neoplastic origin.

Discussion.

Joint Meeting of Section on Surgery, Section on Obstetrics and Gynecology, and Texas Society of Anesthesiologists

Azalea Room, Shamrock Hilton Hotel

4. (4:00) **Panel: Cardiac Arrest.**

Moderator: Charles L. Eckert, Albany, N. Y.

Panelists: Oglesby Paul, Chicago.
Kenneth K. Keown, Columbia, Mo.
Willard M. Allen, St. Louis.
J. Alan Alexander, Houston.

(4:45) **General Discussion.**

Saturday, April 25, 2:30-5 p.m.

Azalea and Bluebonnet Rooms, Shamrock Hilton Hotel

Joint Session: Sections on Surgery, Digestive Diseases, Internal Medicine, Pathology and Radiology.

5. (2:30) Panel: Diseases of the Thyroid.

Moderator: Mavis P. Kelsey, Houston.
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John A. Evans, New York, Radiology.
Nathan A. Womack, Chapel Hill, N. C., Surgery.
Harlan J. Spjut, Houston, Pathology.

Specialty Programs

CONFERENCE OF CITY AND COUNTY HEALTH OFFICERS

(Related Organization)

Chairman—J. E. Peavy, Austin.

(The Texas Association of Public Health Physicians will have a dinner meeting Thursday at 7 p.m., Nile Room, Shamrock Hilton Hotel. The Section on Public Health of the Texas Medical Association will be held Friday from 2:30 to 5 p.m., Walnut Room, Shamrock Hilton Hotel. See Section Meetings section and elsewhere in this Specialty Programs section for details.)

Saturday, April 25, 2:30-5 p.m.

Walnut Room, Shamrock Hilton Hotel

1. (2:30) **Accreditation in Public Health—Advice Welcomed.**
Charles E. Smith, Berkeley, Calif.

2. (3:00) **Mental Health Planning Progress.**
Spencer Bayles, Houston.

(3:20) **General Discussion.**

3. (3:30) **City of Dallas Immunization Program.**
James W. Bass and Earle Standlee, Dallas.

In 1963 the City of Dallas Department of Public Health was awarded a U. S. Public Health Service grant of $58,792, under the terms of the Vaccination Assistance Act of 1962. The grant period extends from July 11, 1963, to July 10, 1964. Application for the grant was approved by the Dallas County Medical Society and the Texas State Department of Health.

Forty-nine immunization clinics were held during the first two quarters of the Dallas program, and 12,064 persons received a total of 17,321 immunizations. These clinics and the number immunized were in addition to the regularly scheduled clinics. The clinics were held at the State Fair, industrial plants, parochial schools, and on the Mobile Tuberculosis Detection Unit. Four immunization surveys have been completed and indicate that 7,615 children (12 per cent) in the one through four age group are not properly immunized for D-P-T. In the one through 39 age group, 162,847 (38 per cent) have not completed the oral polio series; 232,271 (53 per cent) need D-T immunizations; and 264,144 (61 per cent) need smallpox vaccinations.

Promotional letters were mailed to all physicians urging them to accelerate their immunization activities. Letters were also sent to clinics, hospitals, and industrial plants asking them to participate.

(3:50) **General Discussion.**

4. (4:00) **Cystic Fibrosis and Mental Retardation Program.**
Fred P. Helm, Austin.

(4:20) **General Discussion.**

5. (4:30) **Migratory Labor Program in Lubbock.**
David M. Cowgill, Lubbock.

In Lubbock County approximately 6,000 migrant laborers work from May until September, and 20,000 from September until February. A project, financed through the United States Public Health Service, was started July 1, 1963, for the improvement of the health of these migrants. The project includes public health nursing programs consisting of immunizations, well child conferences, tuberculin testing, x-ray, maternity conferences, and examination of migrants who live in Lubbock County. A mobile clinic has been improvised for use in the above clinics. The project also includes a program to improve the sanitation in the camps.

(4:50) **General Discussion.**

ORIENTATION PROGRAM OF TEXAS MEDICAL ASSOCIATION

Executive Secretary—C. Lincoln Williston, Austin.

Saturday, April 25, 8 a.m.

Azalea Room, Shamrock Hilton Hotel

1. (8:00) **Registration.**

2. (8:30) **Call to Order.**
James D. Murphy, Fort Worth.

3. (8:35) **Welcome: Objectives of the Orientation Program.**
Travis Smith, Abilene.

4. (8:40) **An Accounting of Stewardship: What Happens to Your Dues?**
Byron P. York, Houston.

5. (9:00) **The Best Medical Care for All Texans.**
Mr. C. Lincoln Williston, Austin.

6. (9:30) **Medical Ethics—Obligations of the Physician to Patients, Colleagues, and to the Profession.**
Charles E. Oswalt, Jr., Fort Stockton.

(10:00) **Coffee Break.**

7. (10:15) **Call to Order.**
Charles Max Cole, Dallas.

8. (10:15) **Serving the Doctors of Texas.**
Mr. Donald M. Anderson, Austin.

9. (10:35) **Doctor-Patient Relations.**
Walter Walthall, San Antonio.

10. (10:55) The Physician's Responsibility in Preserving Voluntary Health Insurance.
A. Rex Kirkley, Dallas.

11. (11:10) The Veterans Administration Hospital Program: Its Effect upon the Private Practice of Medicine.
Milton V. Davis, Dallas.

12. (11:25) Legal Aspects of Medical Practice: Malpractice—How to Avoid It.
Mr. Philip R. Overton, LL.B., Austin.

(12:15) Adjournment.

12:30 p.m.
Continental Room, Shamrock Hilton Hotel

13. (12:30) Annual Luncheon and Installation of Officers.
Robert Mayo Tenery, Waxahachie, President, Presiding.
It's an Amazing Story.
Edward R. Annis, Miami, Fla., President, American Medical Association.

SOCIETY OF LIFE INSURANCE MEDICAL DIRECTORS OF TEXAS

(The Society of Life Insurance Medical Directors will sponsor a Hospitality Suite in the Shamrock Hilton Hotel Friday until noon Sunday. All Texas Medical Association members are invited to visit the suite. Suite number will be posted in the lobby.)

President—Fred Dinkler, Houston.

Sunday, April 26, 12:15 p.m.

Venetian Room, Shamrock Hilton Hotel

1. (12:15) Luncheon Meeting.

SYMPOSIUM ON ALCOHOLISM

(The Symposium on Alcoholism is sponsored by Texas Commission on Alcoholism, The University of Texas, and the Houston Council on Alcoholism.)

Thursday, April 23, 8 p.m.

Grecian Room, Shamrock Hilton Hotel

1. (8:00) A Three-Phase Treatment Program for Alcohol Addiction.
R. G. Bell, Willowdale, Ontario, Canada.

Addiction is defined as an abnormal need for one or more chemicals. The clinical management of any type of addiction could be considered a three-stage operation:

Phase I includes treatment of intoxication, withdrawal or other medical or surgical disability, plus initial assessment of physical, psychological and social problems; Phase II is concerned with educating the patient concerning the problems discovered in Phase I and initiating a program to eliminate them or bring them under control; and Phase III involves coaching and supervising the patient during the first two years of learning how to live without alcohol, and how to deal with personal and social situations in a new way.

SYMPOSIUM ON CEREBRAL PALSY

(The Symposium on Cerebral Palsy is sponsored by United Cerebral Palsy Association of Texas, Inc., and The University of Texas Medical Branch, Galveston.)

Saturday, April 25, 8:30 a.m.

Garden Room, Shamrock Hilton Hotel

Edward M. Krusen, Jr., Dallas, Chairman, Medical Professional Advisory Board, United Cerebral Palsy Association of Texas, Inc., Presiding.

1. (8:30) Registration.

2. (9:00) Call to Order and Welcome.
Objectives of the Symposium.
Edward M. Krusen, Jr., Dallas.

3. (9:05) The Early Diagnosis of Cerebral Palsy.
Margaret H. Jones, Los Angeles.

4. (9:45) The Individual with Postnatally Acquired Cerebral Palsy.
Chester A. Swinyard, New York.

(10:30) Coffee Break.

5. (11:00) Panel: Answers to the Most Frequently Asked Questions Concerning Cerebral Palsy.
Moderator: William G. Wolfe, Ph.D., Austin.
Panelists: Oliver H. Bown, Ph.D., Austin, Counseling.
Charles E. Cash, D.D.S., Fort Worth, Dentistry.
Chester A. Swinyard, New York, Neurology.
E. Burke Evans, Galveston, Orthopedics.
Arr Nell Boelsche, Galveston, Pediatrics.
Margaret H. Jones, Los Angeles, Pediatrics.
Ira Iscoe, Ph.D., Austin, Psychology.
Tina E. Bangs, Ph.D., Houston, Speech and Hearing.
Mary W. C. Fletcher, Houston, Pediatric Ophthalmology.

12:30 p.m.
Hide-away, Shamrock Hilton Hotel

John B. Truslow, Galveston, Dean and Executive Director, The University of Texas Medical Branch, Presiding.

6. (12:30) Luncheon.
The Research Challenge in Cerebral Palsy.
Margaret H. Jones, Los Angeles, and Chester A. Swinyard, New York.

2:15 p.m.

Garden Room, Shamrock Hilton Hotel

Oscar O. Selke, Jr., Houston, Vice-Chairman, Medical Affairs, Medical-Professional Advisory Board, United Cerebral Palsy Association of Texas, Inc., Presiding.

7. (2:15) **The Role of the Orthopedic Surgeon in Cerebral Palsy.**

E. Burke Evans, Galveston.

8. (2:45) **The Current Status of Drug Therapy in Cerebral Palsy.**

Arr Nell Boelsche, Galveston.

9. (3:15) **The Comprehensive Psychological Assessment of Cerebral Palsied Children.**

Ira Iscoe, Ph.D., Austin.

10. (3:45) **The Need for More Impactful Counseling as a Component of the Total Treatment Program.**

Oliver H. Bown, Ph.D., Austin.

11. (4:15) **Summation.**

William G. Wolfe, Ph.D., Austin.

(4:30) **Adjournment.**

SYMPOSIUM ON DISASTER MEDICAL CARE

Chairman, Committee on Disaster Medical Care, Texas Medical Association—Ben A. Merrick, Dallas.

(The Symposium on Disaster Medical Care is sponsored by the Committee on Disaster Medical Care of the Texas Medical Association.)

Friday, April 24, 9 a.m.

Garden Room, Shamrock Hilton Hotel

1. (9:00) **Registration.**

2. (9:10) **The Emergency Medical and Health Organization in Texas.**

Joseph B. Copeland, Austin.

Upon declaration of an emergency by the Governor, the Commissioner of Health becomes the State Director of Emergency Medical and Health Services. The responsibilities of the State Health Department, and the organization of personnel and facilities to effectively carry on disaster medical care will be outlined.

3. (9:20) **Aims of the Texas Medical Care Program.**

Ben A. Merrick, Dallas.

The necessity for organizing for medical care in disaster situations will be detailed. The sources of personnel to accomplish this, and the means whereby such planning can be effected will be discussed.

4. (9:35) **The Medical Self-Help Program.**

Mr. Philip W. Gauss, Jr., Austin.

A program designed to train people for survival in a time of national disaster is one of the prime essentials in the nuclear age. The scope of this program and the methods for presenting it to the population is to be detailed.

5. (9:50) **Is Survival Worthwhile?**

James L. Johnson, Amarillo.

A nuclear attack does not mean that everyone will be killed or maimed. Proper precautions can save the lives and effectiveness of the major portion of the population. Information will be given regarding shelters and other means of adequate preparation for survival.

6. (10:00) **The Regional Support Plan.**

Jay P. Sanford, Dallas.

The statewide emergency organization is divided into 17 districts. It is desired that each such district should carefully plan and organize its medical and allied health resources for major disasters. When carried out by all 17 districts, there would be an effective means whereby a disaster-struck region could be aided medically by a neighboring region not so damaged. Presented will be the possible means of accomplishing this type of regional support.

(10:30) **Coffee Break.**

7. (10:45) **Sanitation in Disaster.**

Col. Ingalls H. Simmons, Fort Sam Houston.

Sanitation problems will be multiplied to great proportions in periods of disaster. The problems posed by insects, contaminated water, waste disposal, and epidemics will be discussed.

8. (11:00) **Expanded Function Training.**

Joseph R. Shaeffer, San Antonio.

Should nuclear disaster strike, there will understandably be a great disparity between casualty load and total medical resources. This will necessitate compromises with standard day-to-day medical and surgical procedures. Expanded function training envisages augmenting the medical resources in disaster situations by instructing allied health personnel to perform certain medical and surgical tasks not now considered in their province.

9. (11:20) **Question and Answer Panel.**

Moderator: Ben A. Merrick, Dallas.

Panelists: Joseph B. Copeland, Austin.
Jay P. Sanford, Dallas.
Joseph R. Shaeffer, San Antonio.
Col. Ingalls H. Simmons, Fort Sam Houston.

(12:00) **Adjournment.**

SYMPOSIUM ON NUTRITION

Chairman, Committee on Nutrition, Texas Medical Association—Ralph G. Greenlee, Midland.

Basic Sciences in Relation to Nutrition.

(The Symposium on Nutrition is sponsored by the Committee on Nutrition of the Texas Medical Association. Guest speakers will be introduced by D. J. Sibley, Jr., Austin.)

Friday, April 24, 2:30 p.m.

Venetian Room, Shamrock Hilton Hotel

1. (2:30) **Opening Remarks.**

Ralph G. Greenlee, Midland.

2. (2:41) **Metabolic Mechanisms in Cells: The Unity in Biochemistry.**

Robert E. Eakin, Ph.D., Austin.

(3:11) **Discussion: Holbrooke S. Seltzer, Dallas.**

3. (3:17) **Cellular Nutrition and the Genetotrophic Principle.**

Roger J. Williams, Ph.D., Austin.

(3:47) **Discussion: T. S. Painter, Jr., Austin.**

(3:52) **Coffee Break.**

4. (4:11) **The Relation Between Genes and Protein and Its Significance.**
Robert P. Wagner, Ph.D., Austin.

(4:41) **Discussion:** Walter K. Long, Austin.

5. (4:50) **Question and Answer Panel.**
Moderator: Robert S. Nelson, Houston.
Panelists: Robert E. Eakin, Ph.D., Austin.
Roger J. Williams, Ph.D., Austin.
Robert P. Wagner, Ph.D., Austin.

THE TELLER LECTURE

Chairman, Committee on Nuclear Medicine, Texas Medical Association—Herbert C. Allen, Jr., Houston.

(The Teller Lecture and luncheon is sponsored by the Committee on Nuclear Medicine of the Texas Medical Association.)

Sunday, April 26, 12:45 p.m.

Continental Room, Shamrock Hilton Hotel

1. (12:45) **Luncheon.**
Operation Plowshare and Peacetime Uses of Atomic Energy.
Edward Teller, Ph.D., Berkeley, Calif.

TEXAS ACADEMY OF GENERAL PRACTICE SEMINAR

Physiology and Pathology of the Lung

(The Texas Academy of General Practice, with the cooperation of the Texas Medical Association and the Education and Research Institute of General Medicine, offers a seminar on "Physiology and Pathology of the Lung." Attendance at the seminar is acceptable for five accredited hours by the American Academy of General Practice. All American Medical Association members are invited to register. Registration fee is $10. Dr. Cone Johnson, Consultant in Clinical Pulmonary Medicine, Scott and White Memorial Hospital, Temple, will moderate discussion following each presentation.)

Thursday, April 23, 12:30 p.m.

Azalea Room, Shamrock Hilton Hotel

Silas W. Grant, Hillsboro, Presiding.

1. (12:30) **Registration.**

2. (1:00) **Address of Welcome.**
Haden E. McKay, Jr., Humble.

3. (1:05) **Surgical Treatment of Pulmonary Malignancies.**
George V. Brindley, Jr., Temple.

(1:25) **Discussion.**

4. (1:35) **Radiation Treatment of Pulmonary Malignancies.**
James C. Moore, Temple.

(1:55) **Discussion.**

5. (2:05) **Chemotherapy of Pulmonary Malignancies.**
John D. Bonnet, Temple.

(2:25) **Discussion.**

6. (2:35) **Differentiation of Cardiac and Pulmonary Breathlessness.**
Per H. Langsjoen, Temple.

(2:55) **Discussion.**

7. (3:05) **Office Pulmonary Function Evaluation.**
Cone Johnson, Temple.

(3:25) **Discussion.**

8. (3:35) **Measures in Treatment of Obstructive Lung Disease.**
Robert J. Carabasi, Temple.

(3:55) **Discussion.**

9. (4:05) **Anesthetic Problems in Patients with Chronic Pulmonary Disease.**
Charles H. Gillespie, Temple.

(4:25) **Discussion.**

10. (4:35) **Chemotherapy of Respiratory Infections.**
Capt. Everett Rhoades,
Lackland Air Force Base.

(4:55) **Discussion.**

11. (5:05) **Panel Discussion.**
Moderator: John C. Hundley, Fort Stockton.

6-7 p.m.

Bluebonnet Room, Shamrock Hilton Hotel

12. (6:00) **Social Hour for Guest Speakers, Registrants, and Their Wives.**

TEXAS AIR-MEDICS ASSOCIATION
(Related Organization)

President—P. W. Malone, Big Spring.

Secretary-Treasurer—C. F. Miller, Waco.

Saturday, April 25, 8 a.m.

Tower Room, Towers Hotel

1. (8:00) **Registration.**

2. (9:00) **Invocation.**
P. W. Malone, Big Spring.

President's Address and Introduction of Guests.
P. W. Malone, Big Spring.

3. (10:00) **Where Do We Go from Here?**
Mr. George E. Haddaway, Dallas.

4. (10:45) **Recent Medical Causes of Light Plane Crashes.**
Stanley R. Mohler, Oklahoma City.

5. (11:30) Aeromedical Indoctrination for Flight Crews.
Luis Amezcua Gonzales, Mexico, D. F.

(12:15) Recess.

6. (2:00) Aerospace Medical Division, Brooks Air Force Base, Panel Discussion.
Moderator: Col. John P. Stapp, Brooks Air Force Base.
Clinical Aerospace Medicine.
Space Medicine Research.
Crash Investigators.
Training Aspects of Aerospace Medicine.

5 p.m.

Tower Room, Towers Hotel

7. (5:00) Social Hour.

8. (7:30) Annual Banquet.

Sunday, April 26, 8 a.m.

Tower Room, Towers Hotel

9. (8:00) Registration.

10. (8:30) Business Meeting. Election of Officers.

11. (9:00) Local Air Safety Should Use the Epidemiological Approach
John D. Daugherty, Fort Worth.

12. (9:45) Agricultural Poisons and Aerial Applicators.
George L. Gallaher, Harlingen.

13. (10:30) Federal Aviation Agency Panel Discussion.
Moderator: John D. Daugherty, Fort Worth.

(12:00) Adjourn.

TEXAS ASSOCIATION OF PUBLIC HEALTH PHYSICIANS
(Related Organization)

President—Charles A. Pigford, Houston.

Secretary-Treasurer—Luther P. Walter, Austin.

(The Section on Public Health of the Texas Medical Association will meet Friday from 2:30 to 5 p.m., Walnut Room, Shamrock Hilton Hotel. The Conference of City and County Health Officers will be held Saturday from 2:30 to 5 p.m., Walnut Room, Shamrock Hilton Hotel. See Section Meetings section and elsewhere in this Specialty Programs section for details.)

Thursday, April 23, 7 p.m.

Nile Room, Shamrock Hilton Hotel

1. (7:00) Dinner.

2. (8:00) Contributions of the Ethics of Medical Consultation to the Understanding of the Medical Practitioner and His Health Department.
Charles E. Smith, Berkeley, Calif.

TEXAS CANCER COORDINATING COUNCIL

Chairman—R. Lee Clark, Houston.

Thursday, April 23, 12 noon

Hide-away Room, Shamrock Hilton Hotel

1. (12:00) Luncheon Meeting.

TEXAS CHAPTER, AMERICAN ACADEMY OF PEDIATRICS

President—D. H. McDonald, Abilene.

Secretary-Treasurer—G. Kenneth Womack, Austin.

(The Section on Pediatrics of the Texas Medical Association will meet Friday and Saturday from 2:30 to 5 p.m. in the Nile Room, Shamrock Hilton Hotel. See Section Meetings section for details.)

Friday, April 24, 12 noon

Hide-away, Shamrock Hilton Hotel

1. (12:00) Luncheon.

TEXAS CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS
(Related Organization)

President—Milton V. Davis, Dallas.

Vice President and Program Chairman—Charles T. Meadows, Abilene.

Secretary-Treasurer—John L. Kee, Jr., Dallas.

Sunday, April 26, 9 a.m.

Castilian A, B and C, Shamrock Hilton Hotel

1. (9:00) Varicella Pneumonia in Adults.
Capt. Robert E. Harris, Lackland Air Force Base.

2. (9:15) Bronchogenic Carcinoma in Young Patients.
Lt. Col. Ray G. Cowley, Fort Sam Houston.

The increasing adoption of steady smoking habits by increasingly younger children in the United States is documented and related to an increasing incidence in lung cancer in patients under 40 years of age. A retrospective analysis of 49 lung cancer patients under 40 years of age treated at Fitzsimons General Hospital, Denver, was carried out to determine if the clinical course and prognosis differed appreciably from these features in older patients. Studies by others have indicated the presence of highly significant differences requiring a different diagnostic and therapeutic approach in the younger age groups. An analysis of 173 lung cancer patients over 40 years of age at the same institution provides data for comparison.

While the ultimate prognosis in the young and old appears similar, definite differences were found and will be discussed as to clinical importance. A heightened index of suspicion for lung cancer in younger age groups is urged as a major means available today to improve the current poor five year cure rate in this disease. Responsibilities of physicians in actively supporting the preventive medicine aspects of the lung cancer are outlined.

3. (9:30) Preliminary Observations in the Treatment of Acute Myocardial Infarction with Low Molecular Dextran.

Per H. Langsjoen, Temple.

Intravascular sludging is a well recognized phenomenon known to be damaging to many body tissues. It can be reversed by the intravenous administration of low molecular dextran (LMDx) with an average molecular weight of 41,000. Sludging occurs consistently in acute myocardial infarction. This study was designed to observe the effect of intravenous LMDx in clinical cases of acute myocardial infarction, to detect any hazards, and to establish the optimum technique for its administration.

Sixty-five consecutive cases of acute myocardial infarction were given LMDx by continuous intravenous infusion in dosages between 1.5 to 3.0 gm/Kg/24 hr. for the initial 76 hours of their illness in addition to the usual forms of therapy. Variations from the usual pattern of this illness consisted of a prolonged elevation of the serum glutamicoxalacetic transaminase (SGOT), absence of a leukocyte response, delayed elevation of the erythrocyte sedimentation rate, and fairly consistent variations in the usual evolutional EKG changes.

Because of the unavailability of a readily useable laboratory measurement for dextran in the blood, a useful method was devised for estimating the level by gross estimation of its renal excretion in the presence of a constant input.

Mortality in this preliminary study was 16.9 per cent in 65 cases. Mortality in the preceding 202 cases treated at this institution was 46.6 per cent.

4. (9:45) Venous Anomalies Found with Congenital Heart Defects.

Robert H. LePere, San Antonio.

The association of anomalous pulmonary veins with congenital heart defects has received more attention than have the systemic venous anomalies. A number of more common pulmonary venous anomalies and several unusual systemic venous anomalies are documented occurring with many different forms of congenital heart lesions. The most common systemic venous anomaly is the persistent left superior vena cava which occurs in all cases of the present series. It is associated with otherwise uncomplicated patent ductus arteriosus, atrial septal defect, tetralogy of Fallot, ventricular septal defect, atrioventricularis commune, and several more complicated defects. Most of these lesions were studied at operation though some were found during postmortem examination.

The practical application of these low pressure shunts is appreciated at surgery when the operative field may be deluged with blood when they are not previously recognized and dealt with. In each case wherein there was a persistent left superior vena cava as well as a right vena cava, the left was ligated. In one case the ligation of the persistent left superior vena cava corrected a near catastrophic right to left shunt with severe post-bypass cyanosis and cardiac arrhythmia.

The management of the various forms of anomalous drainage is discussed as well as the anatomy and the embryology.

5. (10:00) Pulmonary Tuberculosis in Afghanistan.

Robert R. Shaw, Dallas.

(10:15) Coffee Break.

6. (10:30) Total or Partial Bypass of the Esophagus for the Palliative and/or Curative Treatment of Constricting Lesions.

Richard W. Ernst, Fort Worth.

Discussion is to be of 15 cases, ranging in age from two to 75 years, in whom there were constricting lesions of the esophagus of three major types: carcinoma of the esophagus, lye stricture of the esophagus, and stricture due to peptic esophagitis. In the carcinoma cases and in most lye strictures a total bypass of the thoracic esophagus was accomplished. In some lye strictures and all strictures due to peptic esophagitis only the lower third of the thoracic esophagus was bypassed. The surgical mortality in this series is zero. The late mortality is confined to the carcinoma group of which there were seven cases total. Of these seven cases five have died, four of their disease and one from hypostatic pneumonia resulting from prolonged bed rest at home. The deaths occurred from two months to one year following surgery. All patients in the lye stricture and peptic stricture group are alive and well today. In all cases the transverse and left or right colon was the organ of choice for the esophageal replacement. A discussion of the surgical technique, postoperative management, and treatment of complications will be given. A relatively new approach to the treatment of esophageal carcinoma will be mentioned.

7. (10:45) A Simplified Technique of Open Heart Surgery.

Arthur C. Beall, Jr., Houston.

Development of satisfactory methods of temporary cardiopulmonary bypass made available successful surgical correction of many congenital and acquired lesions of the heart. Early techniques of open heart surgery required a pool of fresh, heparinized blood to prime various types of pump oxygenators, and such a pool of homologous blood presented certain difficulties, both in procurement and use. Recently, many adverse physiological changes associated with cardiopulmonary bypass have been attributed to the use of such pools of homologous blood. Therefore, a technique of open heart surgery has been adopted using plastic disposable oxygenators primed with 5 per cent dextrose in distilled water under normothermic conditions.

Extensive experimental and clinical investigations have demonstrated the superiority of this technique of open heart surgery to those requiring a blood prime in almost every respect. Technical aspects of cardiopulmonary bypass have been simplified considerably. Furthermore, numerous complications associated with the use of pooled homologous blood have been eliminated. Cardiopulmonary bypass now is immediately available in emergency situations, as well as in patients with religious objections to blood transfusion. During the past 18 months more than 500 open heart operations have been performed using this method, and the results have been most gratifying.

8. (11:00) Comparison of Methods of Mechanical Ventilation in Animals.

Loyde H. Hudson, Amarillo.

Under the extreme tests of this experiment it was possible to oxygenate and ventilate adequately a small animal both by endotracheal and external ventilatory devices. A combination of positive and negative pressure to the airway and chest wall was the most efficient method used. The use of a synchronized double chamber for the trunk and head was found to be extremely efficient and found to compare favorably with a Bennett-body tank respirator unit. A tank respirator alone was an efficient ventilating device. Under the conditions of this experiment hypoxia was produced in the animals when room air was used in a body chamber respirator. This could be corrected by using higher positive and negative pressures in the chamber.

9. (11:15) Clinical and Pathological Variation in Patients with Tumor of the Thymus.

John L. Kee, Jr., Dallas.

10. (11:30) Charles M. Hendricks Memorial Lecture: Pulmonary Transplantation — Experimental and Clinical Observations.

Watts R. Webb, Dallas.

The problems of time, technique, and control of the immune response in pulmonary transplantation with an experimental approach to their control will be presented. The first two of these have been solved sufficiently to make clinical transplantation feasible though not yet easy. Control of the immune response has been advanced sufficiently to justify clinical trial and the speaker's early clinical experience will be presented.

12:15 p.m.

Hide-away, Shamrock Hilton Hotel

11. (12:15) Luncheon.

2 p.m.

Castilian A, B and C, Shamrock Hilton Hotel

12. (2:00) Miliary Nocardiosis—A Fatal Complication in a Case of Hodgkin's Disease.
William S. Horn, Jr., Fort Worth.

13. (2:15) Recurrent Mitral Stenosis.
Harold C. Urschel and
Donald L. Paulson, Dallas.

14. (2:30) Correlation of the Water Test and Reflux Esophagitis with Hiatus Hernia.
Eldon K. Siebel, Dallas.

The "water test" is designed to demonstrate reflux of barium from the stomach into the water-filled esophagus. This presumahly should give an indication of the degree of gastroesophageal incompetence especially in patients who have symptoms of esophagitis, with or without hiatal hernia. The accuracy of this presumption is investigated hy esophageal mucosa hiopsy, clinical improvement following surgical correction, and finally comparison of pre- and postoperative "water tests."

15. (2:45) Clinical Applications of Vectorcardiography with Emphasis on Posterior Myocardial Infarction.
Thomas M. Runge, Austin.

Spatial vectorcardiography has been demonstrated to be of supplementary value to the electrocardiogram in a number of clinical conditions including various types of myocardial infarction, myocardial infarction in the presence of left bundle hranch hlock or left ventricular hypertrophy, myocardial fibrosis demonstrahle hy vectorcardiography hut not hy electrocardiography, and myocardial ischemia demonstrahle hy vectorcardiography but not electrocardiography. In addition, vectorcardiography is a useful means of differentiating right hundle branch block from right ventricular hypertrophy in questionable cases and is helpful in differentiating atrial septal defect of the primum type from the secundum type which has a practical application in surgical correction of the defect. Also the vectorcardiogram has been found to be fairly typical in congenital aortic stenosis, whereas the electrocardiogram may not he. In addition, the vectorcardiogram may show myocardial damage in the presence of the WPW phenomena which may mask myocardial damage in the electrocardiogram.

In each of these instances, the vectorcardiogram has heen found to offer information which is not always available in the electrocardiogram, thus establishing the usefulness of this rather difficult, time consuming, and expensive procedure.

Discussion and demonstration of instances in which vectorcardiography is particularly helpful in clinical practice will he presented.

16. (3:00) Excisional Therapy for Postinfarction Left Ventricular Aneurysm in 40 Patients.
Grady L. Hallman, Houston.

Left ventricular aneurysm is a major complication of myocardial infarction and may pose a threat to survival in the form of intractable cardiac failure or peripheral embolization. Between 1958 and 1963, 40 patients have undergone excision of postinfarction left ventricular aneurysms utilizing cardiopulmonary hypass. Eight patients died during hospitalization (20 per cent) and six have died within five months to four years following discharge from causes related to ischemic heart disease. Gratifying improvement was noted in the survivors as regards congestive heart failure and angina, and improvement in cardiac dynamics was confirmed hy postoperative cardiac catheterization. This presentation will be concerned with some of the diagnostic and surgical considerations in evaluation and treatment of this lesion. Clinical and physiologic data (cardiac catheterization) will he discussed and a follow-up evaluation extending over a period of five years will be presented.

(3:20) Recess.

17. (3:30) Methods of Quantitation of Pulmonary Pathology.
S. Donald Greenberg, Houston.

18. (3:45) Asthma in Infants.
John P. McGovern, Houston.

19. (4:00) Benign Lesions of the Esophagus.
William D. Seybold, Houston.

20. (4:15) Considerations in Mass Screening for Pulmonary Disease.
Daniel E. Jenkins, Houston.

A presentation of the problems encountered in screening in a community for pulmonary diseases is given. The purpose and arrangement for screening and the methods of analysis and the various facts obtained is reviewed. Screening includes a questionnaire relating to identifying data, smoking history, past pulmonary diseases, and symptoms regarding cough, sputum, hreathlessness, wheezing, and frequency of respiratory infections. In addition, pulmonary function data have been obtained consisting of the forced expiratory volume curve. Some 3,000 cases have been surveyed and data analyzed. Coordination of these facts with the findings on a microfilm x-ray survey have also heen carried out in most instances.

All of this information has been coded for computers, the facts analyzed and correlations of the data derived with information ohtained in the history. Correlations and trends with the smoking history, breathlessness, and other factors will be mentioned.

21. (4:30) The Use of Antibiotics in Long–Term Treatment of Emphysema.
Edwin G. Troutman, Fort Worth.

The presentation emphasizes the following points: (1) that continuous suppression of infection is necessary for proper management; (2) that the physical characteristics of the sputum do not always indicate the presence of infection; (3) that serial sputum cultures are necessary in order to determine the presence and type of infection; and (4) that periodic gram stains will frequently reveal pertinent information that cannot be obtained hy culture. Slide illustrations of gram stains and culture growths of the most pertinent organisms will be given.

22. (4:45) A New Technique for Division of the Difficult Patent Ductus Arteriosus, and Its Successful Use in a 60-Year-Old Woman.
J. P. Boland, Dallas.

Although it is generally accepted that the presence of a patent ductus arteriosus discovered during the first two decades of life is an indication for interruption or ligation of the abnormal communication, surgical treatment in the adult has been approached with some hesitancy hecause of the technical difficulties imposed hy the early degenerative changes and calcification present in and about patent ducti in older patients. Faced with a 60-year-old woman in severe cardiac failure due to a large calcified patent ductus arteriosus, a method for overcoming the technical dangers of patent ductus division in adults has been devised. This woman is the oldest reported patient to have successful division of a patent ductus arteriosus.

The patient herein reported was a 60-year-old female who had had a known patent ductus arteriosus for many years. She had had progressive weakness and exertional dyspnea for more than ten years. In 1952 she was placed on digitalis and diuretics hut had gradually become completely incapacitated. The physiological studies revealed moderate pulmonary hypertension with a cardiac output of only 2.3 liters per minute, and a pulmonary blood flow of 6.5 liters per minute. X-rays revealed a marked increase in pulmonary vascularity, an enlarged heart, and calcification not only in the aortic arch and isthmus hut also within the patent ductus arteriosus itself.

The method devised and utilized in this patient involves the use of a strip of prosthetic vascular prosthesis in such a manner as to avoid tearing of the ductus and aorta with clamps or sutures and also make crossclamping of the aorta or the utilization of partial cardiopulmonary hypass unnecessary.

Only four reports of successful surgical interruption or ligation of patent ductus arteriosus in patients more than 50 years of age have been found in the world literature.

WATCH FOR FURTHER ANNUAL

SESSION INFORMATION BY MAIL

TEXAS CHAPTER, FLYING PHYSICIANS ASSOCIATION
(Related Organization)

President—Frederick W. Lurting, Big Spring.

Secretary-Treasurer—Cyril J. Ruilmann, Austin.

Program Chairman—Leon J. Alger, Pasadena.

Thursday, April 23, 7 p.m.

Tower Room, Towers Hotel

1. (7:00) **Social Hour and Dinner.**
Three Great Events of Life.
Donald Hull, D.D.S., Houston.

Friday, April 24, 9 a.m.

Tower Room, Towers Hotel

2. (9:00) **Business Meeting and Scientific Program.**

TEXAS DERMATOLOGICAL ASSOCIATION
(Related Organization)

President—Earl R. Cockerell, Abilene.

Vice-President—H. Fred Johnson, Amarillo.

Secretary-Treasurer—M. Allen Forbes, Jr., Austin.

Program Chairman—Coleman Jacobson, Dallas.

Saturday, April 25, 9:30 a.m.

Bluebonnet Room, Shamrock Hilton Hotel

1. (9:30) **The Management of Bacterial Infections of the Skin—20 Years After the Introduction of Antibiotics.**
Clarence S. Livingood, Detroit.

(10:00) **Discussion.**

2. (10:15) **Experiences With the Basophil Degranulation Test.**
M. Allen Forbes, Jr., and Mac Brannen, Austin.

(10:35) **Discussion.**

3. (10:45) **Sporotrichosis in the Texas Panhandle.**
H. Fred Johnson, Amarillo.

(11:05) **Discussion.**

4. (11:15) **Porphyria Cutanea Tarda in the American Negro.**
F. G. Marsh, Fort Sam Houston.

(11:35) **Discussion.**

A VALUABLE ASSET
Wear your registration badge. It is your admission to the exhibit area.

TEXAS DIABETES ASSOCIATION
(Related Organization)

President—F. Peel Allison, Beaumont.

Secretary-Treasurer—Leonard R. Robbins, Houston.

(The following program is jointly sponsored by the Texas Diabetes Association and the Houston Area Diabetes Association.)

Sunday, April 26, 9 a.m.

Bluebonnet Room, Shamrock Hilton Hotel

1. (9:00) **Registration.**

2. (9:15) **A New Class of Antidiabetic Drugs—Experiences with Dimethylisoxazole.**
Robert S. Radding, Houston.

Clinicians have long recognized certain disadvantages to present use of sulfonylureas, and/or biguanides. Secondary failures, limited range of usefulness, alterations in hepatic metabolism, and gastrointestinal side effects have all stimulated a continuing search for other orally-effective hypoglycemogenic agents. One result has been the synthesis of 3.5 dimethylisoxazole and a secondary complex with zinc chloride.

From animal data Dulin and Gerritsen have reported that dimethylisoxazole was 188 times more potent orally than tolbutamide and, further, that it increased glucose oxidation. This complex has now been used in diabetes mellitus as an adjuvant to patients taking sulfonylureas, as the sole agent in untreated stable diabetics, and as a supplement to insulin-dependent diabetics. Following a review of the pharmacology, the present report will describe and analyze the results of clinical trials with this compound.

3. (9:35) **The Adolescent with Diabetes Mellitus.**
George H. Lowrey, Ann Arbor, Mich.

The problem of good control in the adolescent patient is greater than at any other time during childhood. This results from the natural rebellion against authority that characterizes the adolescent. In addition, physical factors such as rapid growth and sexual maturation complicate the picture. These factors will be discussed and suggestions toward their solution will be presented.

(10:05) **Coffee Break.**

4. (10:20) **Diagnostic Tests of Thyroid Function.**
A. Stone Freedberg, Boston.

Recent advances in our understanding of thyroid physiology and biochemistry have resulted in the development of a large number of laboratory tests of thyroid function. The present report will attempt to summarize such tests, their bases and clinical applicability. Emphasis will be placed on more recently described tests, attempting to measure the binding capacity and saturation of the serum proteins for the thyroid hormones, namely the resin uptake of 1-131 triiodothyronine or 1-131 thyroxine from plasma, the red cell uptake of triiodothyronine and thyroxine from plasma, and the saturation tests as measured electrophoretically.

5. (10:50) **Diabetic Diarrhea.**
Max Ellenberg, New York.

Diabetic diarrhea is a specific entity which may be differentiated from other diarrheas occurring in the diabetic. The necessity for careful differential diagnostic evaluation of diarrhea in the diabetic is to be emphasized in view of significantly differing therapeutic indications. This is underscored by malabsorption syndrome in diabetes; although its relationship to diabetic enteropathy, if any, is still undetermined, superb therapeutic results are attainable following its recognition.

6. (11:20) **Experimental Studies on the Role of Glucagon in Appetite Regulation.**
Harry S. Lipscomb, Houston.

Glucagon, the hyperglycemic, glycogenolytic factor produced by the α-cell of pancreatic insular tissue, is a peptide with no known physiologic role in man. While it is capable of elevating blood-sugar rapidly, and has been termed an "insulin-antagonist," no clinical conditions of deficit or excess have been described. It is the object of these studies to prove or disprove the hypothesis that glucagon, at levels much lower than those required to elevate blood-glucose, may be uniquely capable of depressing appetite by an effect exerted upon hypothalamic tissue in the region of *nucleus ventromedialis*, and that minute fluctuations of levels of circulating glucagon may play a critical role in regulation of food intake in man.

7. (11:40) Current Methods for the Evaluation of Aldosterone Secretion.

George T. Bryan, Galveston.

A review of the physiology of aldosterone suggests that an evaluation of aldosterone secretion is important in several disease states, namely hypertension, unexplained alkalosis, dwarfism and hypoadrenocorticism. The interpretation of such an evaluation requires that the intake of sodium and potassium be controlled, and with this, the changes of serum sodium and potassium become useful indicators of aldosterone secretion. The assay of urinary aldosterone by isotopic methods is reliable when normal or increased quantities are present. The indirect measurement of aldosterone secretion by isotope dilution-derivative methods offers the most reliable technique, particularly in patients with decreased secretion and in those situations where daily 24-hour urine collections are not practicable.

12 Noon

International Club Lounge, Shamrock Hilton Hotel

8. (12:00) Luncheon.

9. (1:00) Panel: Thyroid Disease and Diabetes.

Moderator: Leonard R. Robbins, Houston.

Panelists: A. Stone Freedberg, Boston.
Max Ellenberg, New York.
Harold L. Dobson, Houston.
Marc Moldawer, Houston.

2 p.m.

Bluebonnet Room, Shamrock Hilton Hotel

10. (2:00) Annual Business Meeting and Election of Officers.

11. (2:20) Insulogenic Response to Hyperglycemia After Chronic Treatment with Sulfonylurea.

Holbrooke S. Seltzer, Dallas.

12. (2:40) Carbohydrate and Vascular Defects in Families of Known Diabetics.

Harold L. Dobson, Donald P. Ford, James M. Gray, and John M. Lewis, Houston.

13. (3:00) Medical Student Award Presentation.

14. (3:20) The Liver in Diabetes Mellitus—Hepatic Metabolism Following Antidiabetic Drugs.

Robert S. Radding and Wilson G. Brown, Houston.

Continuing serial studies of hepatic function in patients exposed to sulfonylureas reveal an incidence of abnormalities in the metabolism of serum alkaline phosphatase, but its pathological significance is uncertain. Consequently, a series of studies has been initiated to define and understand the hepatic status of all persons with diabetes. The patients were classified as follows: (1) untreated, (2) insulin only, (3) phenformin, and (4) sulfonylureas. A liver biopsy was obtained simultaneously with specimens for serum alkaline phosphatase, serum glutamic oxalic transaminase, serum glutamic pyruvic transaminase, thymol turbidity, aldolase, isocitric dehydrogenase, lactic dehydrogenase, urea nitrogen, and cholesterol. The results will correlate the anatomic and functional data to demonstrate the hepatic pathophysiology of diabetes mellitus with or without exposure to sulfonylureas.

TEXAS INDUSTRIAL MEDICAL ASSOCIATION, TEXAS PHYSICAL MEDICINE AND REHABILITATION SOCIETY, AND TEXAS TRAUMATIC SURGICAL SOCIETY

(Related Organizations)

Texas Industrial Medical Association

President—Frederick Fink, Dallas.

Program Chairman—Roy E. Joyner, Texas City.

Texas Physical Medicine and Rehabilitation Society

President—James R. Oates, Houston.

Secretary—Jewel S. Daughety, Dallas.

Texas Traumatic Surgical Society

President—Frank R. Denman, Houston.

Secretary-Treasurer—C. M. Ashmore, Bellaire.

Thursday, April 23, 9 a.m.

Castilian A, B and C, Shamrock Hilton Hotel

1. (9:00) The Role of Industry in Cardiac Rehabilitation.

Francis W. Wilson, Port Arthur.

2. (9:30) Early Recognition and Management of Nonpenetrating Injuries of the Abdomen.

Joe R. Gandy, Houston.

(10:00) Coffee Break.

3. (10:15) A Reacquaintance with Some Simple Procedures in the Diagnosis of Vascular Occlusive Disease.

Robert J. Potts and Paul R. Ellis, Jr., Dallas.

4. (10:45) Peripheral Nerve Lesions:

(10:45) Clinical Diagnosis.

John Tennant, Houston.

(11:15) Electro Diagnosis.

Charles R. Peterson, Houston.

(11:45) Treatment, Emphasizing Rehabilitation.

James R. Oates, Houston.

(12:00) Recess.

12:15 p.m.

Garden Room, Shamrock Hilton Hotel

5. (12:15) Luncheon.

6. (12:45) Surgical Treatment of Traumatic Wounds of the Heart and Blood Vessels.
Albert L. Shirkey and Denton A. Cooley, Houston.

2 p.m.

Castilian A, B, and C, Shamrock Hilton Hotel

7. (2:00) The Management of Blunt Trauma to the Chest.
Howard T. Barkley, Jr., Houston.

8. (2:30) New Laboratory Methods in Industrial Toxicology.
Eric G. Comstock, Houston.

(3:00) Coffee Break.

9. (3:15) Alcoholism in Industry.
Robert L. Raleigh, Longview.

10. (3:45) Management of Traumatic Injuries of the Hand.
Livius L. Lankford, Dallas.

11. (4:30) Separate Annual Business Meetings.
Texas Industrial Medical Association, Castilian A and B, Shamrock Hilton Hotel.
Texas Traumatic Surgical Society, Castilian C, Shamrock Hilton Hotel.
Texas Physical Medicine and Rehabilitation Society, Walnut Room, Shamrock Hilton Hotel.

6:15 p.m.

El Charro Room, Shamrock Hilton Hotel

12. (6:15) Hospitality Hour.

TEXAS NEUROPSYCHIATRIC ASSOCIATION
(Related Organization)

President—H. Harlan Crank, Houston.

President-Elect—E. Ivan Bruce, Jr., Galveston.

Vice-President—Irvin M. Cohen, Houston.

Secretary-Treasurer—James K. Peden, Dallas.

(A social hour will be held at 7:30 p.m. Saturday, April 25, in the Ramada Club, top of the First City National Bank Bldg.)

Sunday, April 26, 8 a.m.

Grecian Room, Shamrock Hilton Hotel

1. (8:00) Registration.

2. (9:00) Opening Remarks.
H. Harlan Crank, Houston.

3. (9:15) Guy Witt Memorial Paper.

Presentation of Award.
Perry C. Talkington, Dallas.

The Use of Electronystagmography as a Diagnostic Tool.
V. William Cole, Houston.

Electrographic methods of recording eye movements have made it possible to record nystagmus objectively. The pattern of the tracing is useful in differentiating various clinical syndromes in which nystagmus occurs. Preliminary studies of this new diagnostic tool are reported.

4. (9:45) Mental Retardation.
D. Bernard Foster, Topeka, Kan.

(10:15) Coffee Break.

5. (10:30) Phobias, Depressions, and Addictions.
Sydney G. Margolin, Denver.

6. (11:15) Joint Business Session—Texas Neuropsychiatric Association and Texas District Branch Society of American Psychiatric Association.

(12:00) Appetizers.

12:45 p.m.

Emerald Room, Shamrock Hilton Hotel

7. (12:45) Luncheon.

Section on Neurology, Neurosurgery, and Allied Fields

Irvin M. Cohen, Houston, Presiding.

2 p.m.

Garden Room, Shamrock Hilton Hotel

8. (2:00) Spondylotic Cauda Equina Radiculopathy: Recognition and Management.
George J. Ehni, Houston.

There has been recognized with increasing frequency recently an affection of the cauda equina similar in many respects to the abnormality of the cervical spinal cord that occurs with degenerative or spondylotic osseous changes. When severe, the condition can be mistaken preoperatively for cauda equina tumor and in its less severe form, it is likely to be mistakenly diagnosed and treated as herniation of a disc. Interlaminal disc removal is ineffective therapy and accurate preoperative diagnosis is essential if these patients are to be given satisfactory relief. Attention to certain characteristics of the history and clinical examination as well as insistence upon good cross-table lateral films at the time of myelography should dispel diagnostic difficulties.

9. (2:30) Developmental Language Disorders in Children.
Morris H. Lampert, San Antonio.

A study of 400 children, all of whom have some form of perceptual disorder, is discussed. Defects include difficulty with reading (word recognition, phonics, comprehension), arithmetic, spelling, syntax, word meaning, comprehension (auditory or visual), or with writing. Associated disorders of spatial function (laterality, body schema, geometric or geographic constructional ability), speech function (usually dyspraxias of speech), motor function (pyramidal or extrapyramidal, cerebellar signs, or their variations) are discussed.

Congenital defects, intelligence, and etiologic factors are considered. Language, learning and diagnostic approaches are described.

10. (3:00) **Pain in the Neck-Shoulder-Arm.**
D. Bernard Foster, Topeka, Kan.

Section on Psychiatry and Psychoanalysis

E. Ivan Bruce, Jr., Galveston, Presiding.

2 p.m.

Grecian Room, Shamrock Hilton Hotel

11. (2:00) **Panel: Community Psychiatry.**
Moderator: H. Harlan Crank, Houston.
Concepts of Community Based Psychiatry.
Hamilton F. Ford, Galveston.
Effect of Use of Community Facilities on State Hospital Admissions.
Cyril J. Ruilmann, Austin.
Role of the Private Psychiatric Hospital in Community Psychiatry.
Perry C. Talkington, Dallas.
The University Medical Center and Community Psychiatry.
Moody C. Bettis, Houston.
Psychiatric Organizations and Community Psychiatry.
Alex D. Pokorny, Bellaire.

12. (3:00) **The Essentials of Psychotherapy.**
Sydney G. Margolin, Denver.

3:30 p.m.

Grecian Room, Shamrock Hilton Hotel

13. (3:30) **Joint Business Session—Texas Neuropsychiatric Association and Texas District Branch Society of American Psychiatric Association. Election of Officers.**

TEXAS OPHTHALMOLOGICAL ASSOCIATION AND SECTION ON OPHTHALMOLOGY

President, Texas Ophthalmological Association and Chairman, Section on Ophthalmology—Louis Daily, Houston.

Vice President, Texas Ophthalmological Association and Secretary, Section on Ophthalmology—Everett R. Veirs, Temple.

Secretary—Harold E. Hunt, Paris.

Treasurer—Harl D. Mansur, Jr., Wichita Falls.

Friday, April 24, 8:30 a.m.-5 p.m.

Grecian Room, Shamrock Hilton Hotel

1. (8:30) **Early Treatment of Common Eye Injuries. (Refresher Course primarily for nonophthalmologists.)**
Brittain F. Payne, New York.

Satisfactory treatment of common eye injuries to preserve vision, to prevent scarring and insure rapid healing often depends on the initial care given the patient. The history and the visual acuity of the patient should be recorded before the physician begins his examination. The topics to be discussed will include: superficial foreign bodies of the cornea and conjunctiva, intraocular foreign bodies, contusions of the orbit, eyelids and globe, and chemical burns.

2. 10:00) **President's Address: The Texas Ophthalmological Association—A Report on Some of Its Activities.**
Louis Daily, Houston.

3. (10:30) **Pupil Block and Pupillary Block Glaucoma.**
H. Saul Sugar, Detroit.

Pupillary block following cataract surgery is due to blocking of the pupillary opening, as well as any iridectomy or iridotomy communications between the anterior and posterior chambers by postoperative inflammatory adhesions. Experience with 19 cases is to be described. In 13, pupillary block glaucoma was present. The diagnosis and treatment will be outlined, with special emphasis on one case which appears to show the relation between malignant glaucoma and pupillary block glaucoma.

(12:00) **Recess.**

12:30 p.m.

Castilian A, B, and C, Shamrock Hilton Hotel

4. (12:30) **Luncheon of Texas Ophthalmological Association.**

1:30 p.m.

Grecian Room, Shamrock Hilton Hotel

5. (1:30) **Business Meeting. (Members Only)**

2:30 p.m.

Grecian Room, Shamrock Hilton Hotel

6. (2:30) **Office Planning for the Ophthalmologist.**
Dan A. Russell, Jr., San Antonio.

In any office, efficiency can be increased without impairing proper patient care. Arrangement of refraction lanes and treatment rooms, systems for filing correspondence and records, and equipment and forms will be discussed.

7. (3:00) **Clinical Aspects of Lethal Midline Granuloma.**
John E. Eisenlohr, Dallas.

Lethal midline granuloma is an uncommon and poorly understood entity characterized by progressive ulceration of midline facial structures including the nose, oral cavity, sinuses and orbits. The disease almost invariably has a fatal outcome despite vigorous efforts to arrest its progress. A description of some typical aspects of this disease, as well as some comments on therapy, will be presented.

8. (3:30) **Pathological Appraisal of Retinal Detachment Operations.**
Brittain F. Payne, New York.

A brief review of the normal anatomy of the retina is necessary to discuss the pathological appraisal of retinal detachment operations. The final result depends on the artificial adhesions of the retina to the choroid, the evacuation of the subretinal fluid and the shortening of the globe. The new school of retinal surgeons seem to emphasize the anatomical restoration rather than visual results. The patient is concerned with the amount of sight obtained after the mental and the physical trauma of a major surgical procedure.

(4:30) **Visit Exhibits.**

Saturday, April 25, 8:30 a.m.-5 p.m.

Grecian Room, Shamrock Hilton Hotel

9. (8:30) Deceptive Diagnosis in Lesions of the Ocular Fundus. (Refresher Course primarily for ophthalmologists.)

H. Saul Sugar, Detroit.

A number of difficult or confusing diagnosis pictures of the ocular fundus will be presented. Among these are macular lesions associated with pits in the optic disc, senile macular degeneration, Coats' disease, senile miliary aneurysms, vascular conditions, melanocytoma, lesions simulating tumors, and inflammatory lesions misdiagnosed as noninflammatory ones. This course will be in the nature of a course in diagnosis of clinical fundus pathology.

10. (10:00) Experiences with Fibrinolytic Agents in Treatment of Total Hyphemia.

Rufus A. Roberts, Jr., Fort Worth.

Deficiencies in the surgical treatment of total hyphemia with secondary glaucoma following blunt trauma has stimulated a revival of interest in the purified fibrinolytic agents. Animal studies and 11 clinical cases of total hyphemia treated with figrinolytic enzymes will be reviewed.

11. (10:30) Heterochromic Cyclitis.

H. Saul Sugar, Detroit.

Heterchromic cyclitis constitutes a syndrome consisting of mild chronic cyclitis associated with heterochromia iridis, often with cataract and sometimes with glaucoma. The findings in 18 cases are used to emphasize the diagnostic features and to suggest that the condition is heredodegenerative in nature. For this reason treatment is of little help except when cataract and glaucoma are present. Both of the latter are treated as if no cyclitis were present.

(12:00) Recess.

12. (2:30) Hemangioma of the Orbit.

Walter G. Morrow, El Paso.

Hemangioma is said to be one of the commonest primary tumors found within the orbit. However, in the private practice of the average ophthalmologist, few such tumors are seen. This presentation will consist of four interesting case reports with a few comments emphasizing the problems in diagnosis and management of this condition.

Discussion: Ralph G. Berkeley, Houston.

13. (3:00) Nematode Endophthalmitis.

Ralph G. Berkeley, Houston.

An unusual case of a child with systemic larvae migrans and proven nematode endophthalmitis will be presented, and illustrated with photographs and illustrations taken during the several years that this child was followed. A brief review of the literature on this subject will be given with emphasis on the etiology, differential diagnosis, and treatment.

14. (3:30) Operating Room Minutiae.

Sam N. Key, Jr., Austin.

This presentation is intended for the average or "occasional" ophthalmic surgeon and is designed to discuss some of the smaller details of the arrangement, conduct, assistance, instrumentation, sterilization, drug handling, etc., during routine ophthalmic operation. Various methods and techniques seen or reported elsewhere, with their possible merit or disadvantage, will be mentioned.

15. (4:00) Prevention of Complications in Cataract Surgery.

Brittain F. Payne, New York.

The prevention of complications in cataract surgery depends on the training and the experience of the surgeon. The physical and mental condition of the patient must receive careful attention. A definite preoperative program should be followed before the patient enters the hospital for additional laboratory tests and preparation for the operation. The advantages of combined general with local anesthesia should be considered carefully.

Other important things to consider are: the choice of conjunctival flap and sutures, the use of alphachymotrypsin, the use of forceps or erisophake, the restoration of the anterior chamber after the closure of the wound, the dressing and the return of the patient to his room.

TEXAS ORTHOPAEDIC ASSOCIATION

(Related Organization)

President—Charles B. Sadler, Amarillo.

Secretary-Treasurer—Margaret Watkins, Dallas.

Program Chairman—Frank O. McGehee, Houston.

Saturday, April 25, 9 a.m.

Castilian A, B, and C, Shamrock Hilton Hotel

1. (9:00) Welcome.

Charles B. Sadler, Amarillo.

2. (9:05) Indications for Open Reduction in Acetabular Fractures.

Robert F. Hyde, Amarillo.

The permanent alteration of the normal relationship between the femoral head and the superior dome of the acetabulum frequently seen following acetabular fractures results in progressive deterioration of the hip joint. Restoration of the acetabulofemoral relationship by open surgery in cases in which any distortion of this relationship exists is suggested. The mechanics of injury, the surgical planning and the operative procedure are discussed. Slides representing the more complicated fractures are to be presented.

Discussion: Hanes H. Brindley, Temple.

3. (9:25) Anatomical Correction of the Bunion Foot.

Rex J. Howard, Fort Worth.

The presentation shows the incisions, one in the web between the first and second toes and one on the medial side of the first MP joint.

The adductor is detached from the short flexor and the lateral sesamoid is removed, the exostoses are then removed from the medial side of the head of the first metatarsal, from the medial side of the proximal end of the first phalanx, from the lateral side of the head of the first metatarsal and the lateral side of approximately the first phalanx and the medial side of the head of the second phalanx. A complete arthroplasty and stripping of the capsule is done at the head neck of the first metatarsal. The adductor is transplanted underneath the neck of the first metatarsal. A capsulotomy is done on the proximal end of the first metatarsal on the medial aspect of the foot.

The foot is corrected by pushing together passively the heads of the first and second metatarsals and the suture through the adductor is then used to close the capsule on the medial side of the foot. Before the capsule is closed, the capsule is trimmed and this, in turn, transplants the long flexor underneath the head of the first metatarsal and exerts a medial pressure in the lateral direction against the head of the first metatarsal to keep the foot corrected, the capsule is then closed with cat-gut and the skin incisions are closed and an Ace bandage is applied.

Patient is allowed to walk within 24 to 48 hours, is put in the whirlpool the third or fourth day, and stitches are removed on the tenth day. Patient is then given gait training and instructions in use of the long flexor. Advantages of this operation include early ambulation, small amount of pain, and good functional results.

Discussion: Marvin P. Knight, Dallas.

4. (9:45) **Lumbar Spine Fusions—Comparative Results.**

Louis J. Levy, Fort Worth.

In an effort to evaluate and improve results in lower lumbar spine fusions, a group of 250 cases of this type was studied. This analysis indicates that one should expect almost 99 per cent fusions when only one joint is fused, and 92 per cent satisfactory fusions in two-joint fusion attempts, excluding spondylolisthesis. It is more difficult to obtain fusions in spondylolisthesis; and in this group, only 81 per cent of the cases obtained satisfactory fusions from the first surgical procedure. Following surgery, adequate immobilization for a sufficient period of time is necessary. The type of fusion is discussed and the necessity of obtaining sufficient autogenous bone is stressed.

The percentage of satisfactory clinical results is not as good as the percentage of solid fusions in comparable cases, due in part to the nature of the disease process itself. Usually, however, the prolonged convalescence or continued complaints even when solid fusion is obtained are seen in the medicolegal cases, in the obese patients, in those who cooperate poorly during their convalescence, and in patients with emotional instability. Screening cases preoperatively will help rule out some of these factors and improve the percentage of satisfactory clinical results.

Discussion: E. Burke Evans, Galveston.

5. (10:05) **Limited Spinal Fusion in Spondylolisthesis and Spondylolysis.**

William W. Sullivan, Galveston.

Considering the nature and pathological anatomy of spondylolisthesis and spondylolysis, it is possible that present methods of posterior element fusion are more extensive than desirable or necessary. These methods eliminate more joints from motion than is required for stabilization of the defective segment and thus may hasten the onset of degenerative arthritis above the fusion. A limited fusion technique has been devised. The results in seven cases have been analyzed.

Discussion: Phillip L. Day, San Antonio.

(10:20) **Coffee Break.**

6. (10:35) **Instrumentation of the Spine—600 Cases.**

Paul R. Harrington, Houston.

(11:20) **General Discussion.**

(11:30) **Appetizers.**

12:30 p.m.

Continental Room, Shamrock Hilton Hotel

7. (12:30) **Annual Luncheon of Texas Medical Association.**

It's an Amazing Story.

Edward R. Annis, Miami, Fla., President, American Medical Association.

2 p.m.

Castilian A, B, and C, Shamrock Hilton Hotel

8. (2:00) **Treatment of Malum Coxae Senilis.**

Mark B. Coventry, Rochester, Minn.

General Discussion.

9. (3:00) **Restoration of Opposition in the Thumb.**

Royce C. Lewis, Jr., Lubbock.

Complexities of opposition of the thumb are discussed. True abduction of the thumb, rotation and extension, and lateral deviation of the proximal phalanx on the first metacarpal bone are all essential features for normal function.

The presentation mentions various operational procedures, giving special attention to the tendon transfer operations which seem to fulfill the requirements of proper restoration of opposition most completely. The technique originally described by Riordan includes all essential features, and is described in slightly modified form in detail. Postoperative care, pitfalls, and causes of less than excellent functional results are discussed.

Consideration is given to bone operations to restore or establish opposition including metacarpal osteotomy and occasional associated pollicization of the radial digit when the thumb is completely absent; carpal osteotomy; and various types of operations incorporating bone grafts bridging the first and second metacarpals. Soft tissue operations are discussed including procedures to lyse contractures in the thumb web or adductor pollicis muscles, and tendon operations in the form of either tenodesis or tenoplasty.

Discussion: Robert A. Murray, Temple.

10. (3:20) **Orthopedic Care of the Severely Burned Patient.**

E. Burke Evans, Galveston.

In the care and treatment of severely burned patients, the orthopedic surgeon, by virtue of his special training, is well equipped to render service on a consultant basis. The complications of burns which invite his attention and interest are contractures, dislocations, muscle atrophy, bone atrophy, growth disturbances, ankylosis, heterotopic bone formation and fractures. His knowledge of the uses and efficacy of definitive splinting and pinning techniques and of physical therapeutic measures gives him an important role in the acute phase of burn treatment. His understanding of bone and joint pathology and his skill in surgery of the extremities makes him an essential member of the burn team in the chronic phase.

Discussion: Colonel John A. Moncrief, Atlanta, Ga.

11. (3:40) **Annual Business Meeting.**

(4:30) **Adjournment.**

TEXAS OTOLARYNGOLOGICAL ASSOCIATION AND SECTION ON OTOLARYNGOLOGY

President, Texas Otolaryngological Association—William Skokan, Fort Worth.

Vice-President and Program Chairman, Texas Otolaryngological Association—J. Patrick Moran, Corpus Christi.

Secretary, Texas Otolaryngological Association—Norman E. Wright, Amarillo.

Treasurer, Texas Otolaryngological Association—Ben M. Nail, Jr., Houston.

Chairman, Section on Otolaryngology—Daniel B. Powell, Jr., Austin.

Secretary, Section on Otolaryngology—Ben T. Withers, Houston.

Friday, April 24, 8:30 a.m.-6 p.m.

Normandy A and B, Shamrock Hilton Hotel

1. (8:30) **Evaluation of the Dizzy Patient. (Refresher Course.)**

G. O. Proud, Kansas City, Kan.

Man attains posture and executes voluntary movements through constant muscle tonus and delicate coordination which are con-

trolled by the central nervous system. The nervous system receives information from informer systems such as the eyes, the touch receptors in the soles of the feet, the proprioceptive receptors in muscles, tendons and joints, and the labyrinth. Disease of the informers makes itself known in the form of unsteadiness, dizziness or light-headedness. An investigation of the vestibulocerebellar complex is called for in a patient with one of these complaints. A discussion of the anatomy and physiology of this apparatus is given. Clinical testing, symptoms, and specific diseases leading to otoneurological phenomena are discussed.

2. (10:00) Surgical Treatment and Findings in Mixed Congenital Deafness.

Woodrow D. Schlosser, Philadelphia.

During the study of congenital deafness, a certain number of cases showed a conductive element superimposed upon the sensorineural deficit. After extensive audiologic study indicated that this was a bona fide finding, surgical exploration of a series of middle ears was performed. Anatomical deformities which were found in these patients can be categorized in four groups. However, the most common anomaly was fixation of the stapes. Following surgical intervention, many of these children, on a voluntary basis, did not show any improvement in their hearing. However, on involuntary hearing tests, these patients did reveal that the conductive element had been eliminated. In some cases, months passed before voluntary responses definitely showed the elimination of the conductive element. Even then, the hearing improvement has been of little use to the patient in discriminatory functions. It appears that there are factors involved in the processes of audition other than improvement in the function of the conductive mechanism. Although neurological examination and electroencephalographic examinations do not show any neurologic deficit, some factors appear to be operant in the central auditory system which at present are not known nor understood.

3. (10:45) Glioma of the Nose.

Donald N. Matheson, Fort Worth.

4. (11:15) Some Aspects of Tympanoplasty.

Woodrow D. Schlosser, Philadelphia.

Tympanoplastic surgery has gone through many phases since surgical concept was introduced a number of years ago by Wullstein and Zöllner. At the present time, there are many variations of this type of surgery. Questions of how much surgery, and of what type of graft are numerous and the answers appear to be the result of personal experience. It seems that no two clinicians do tympanoplastic surgery in a similar manner. Nevertheless, there is a strong tendency to individualize each case rather than do all cases of chronic otitis media in a similar manner. Thus, the procedures may involve middle ear surgery in one case, middle ear and mastoid surgery in another case, or mastoid surgery in the form of a radical mastoidectomy with a graft to the middle ear area in others.

One of the problems in tympanoplastic surgery is eustachian tube function. This has been discussed and studied by many people. Nevertheless, in Dr. Schlosser's experience, inadequate tubal function seems to be one of the major causes of failure in tympanoplastic surgery. His feeling has been for some time that chronic otitis media is a disease that starts in the eustachian tube and then involves the middle ear and the mastoid cell system.

Surgery in the past has been confined primarily to the mastoid and middle ear areas. Many cases show initial good results and these results may last for a year or more. However, as time goes by there is retraction of the graft until eventually it makes contact with the medial wall and no longer is a good vibrating surface. This result then resembles chronic adhesive otitis media with reduced hearing.

(12:00) Recess.

5. (2:30) Eustachian Tube Function Tests.

G. O. Proud, Kansas City, Kan.

A simple office test for the evaluation of eustachian tube patency and function has been devised. Although it has yielded some information on the physiologic mechanisms of the auditory tube of experimental animals, its chief value lies in its use as a method for the preoperative assessment in subjects for whom tympanoplasty is contemplated. A test solution, fluorescein, is introduced into the middle ear and sought for in the nasopharynx with ultraviolet light.

6. (3:15) Pyrogenic Reaction Following Tonsillectomy and Adenoidectomy.

Ben T. Withers and Ben M. Nail, Jr., Houston.

7. (3:45) The Treatment of the Draining Ear in Children.

Woodrow D. Schlosser, Philadelphia.

In spite of modern antibiotic therapy, children still continue to have intermittent otitis media with drainage. Many of these patients are treated with an antibiotic therapy which clears up the infection, but it may flare up in a short period of time. Additional therapy again may improve the situation with a change in antibiotics after cultures of the drainage have been taken. However, in a certain number of cases a point is reached where antibiotic therapy only suppresses the disease process. As soon as the child is taken off the antibiotic therapy without any upper respiratory infection or any initiating disease, the ear spontaneously begins to drain again. Surgical exploration of the mastoid in these cases is necessary because the site of infection is most often the mastoid cell system. A number of cases of this type have been operated upon. The operation performed was a simple mastoidectomy. In some cases after the mastoid has been cleaned, the perforation of the tympanic membrane seals itself off without any further tympanoplastic procedures. A simple mastoidectomy in an intermittent draining ear in a child still is our best weapon in our surgical armamentarium for the treatment of these cases.

8. (4:30) Business Meeting.

9. (5:00) Social Hour.

Saturday, April 25, 10 a.m.-4:15 p.m.

Normandy A and B, Shamrock Hilton Hotel

10. (10:00) Otitis Externa and Laryngoscopy in Children.

G. O. Proud, Kansas City, Kan.

Meticulous mechanical cleansing of the ear canal is probably the most important segment of the treatment of otitis externa. Surgical complete denudation of the canal epithelium will, on occasions, effect a cure when the condition has been a refractory one.

Four methods are available for laryngoscopic examination in children. Each has its limitations and advantages. A lens system inspection is a safe, inexpensive and speedy technique which may be used with outpatients.

11. (10:45) Stapedectomy: An Evaluation of Current Techniques.

William L. Draper, Houston.

12. (11:15) The Diagnosis and Treatment of Acoustic Neuroma.

Woodrow D. Schlosser, Philadelphia.

The early diagnosis of acoustic neuroma usually is an etologic diagnosis. After symptoms have been present for many years and neurologic signs such as fifth nerve paresis develop, the diagnosis is relatively easy. However, in otologic examination of patients, Dr. Schlosser believes any patient who has a unilateral sensorineural deficit with reduced speech discrimination should be considered as a possible case of acoustic neuroma unless proven otherwise. Many of these patients show tinnitus as the earliest sign, followed later on by a hearing loss and marked reduction in auditory discrimination. In the otologic diagnostic armanentarium, the caloric test usually will show either reduced function or no function of the involved labyrinth. The Békésy audiometer will many times be of some diagnostic aid. Recruitment may or may not be present. Further studies would include X-rays of the temporal bone, involving tomograms and pneumoencephalographic studies performed by the neurosurgeon. When the diagnosis of an early acoustic neuroma is made, the best method of approach is either by the lateral

temporal approach or the translabyrinthine approach. If the tumor is very small and situated in the internal auditory meatus, the translabyrinthine approach would be the method of choice. When the tumor is somewhat larger, the transtemporal approach can be utilized. For extremely large tumors, the posterior fossa approach still remains the method of choice.

(12:00) Recess.

13. (2:30) **Middle Ear Effusion.**
G. O. Proud, Kansas City, Kan.

Eustachian tube obstruction is not an inevitable etiologic agent in middle ear effusion. The complication of cholesteatoma is not rare, and careful long range observation is essential. Only through this vigilance can the complication be recognized and dealt with early and permanent hearing loss prevented.

14. (3:15) **Congenital Sensory-Neural Deafness.**
Bobby R. Alford, Houston.

15. (3:45) **Audio-Otology in the Office.**
William P. King, Corpus Christi.

TEXAS PROCTOLOGIC SOCIETY
(Related Organization)

President—Harry B. Burr, Houston.

Secretary—W. Truett Melton, Houston.

(The Texas Proctologic Society will sponsor a surgical clinic Thursday, April 23, 8 a.m. to 12 Noon, Memorial Baptist Hospital, 602 Lamar. A luncheon will be held at 12:30 p.m. at the Sheraton-Lincoln Hotel.)

Thursday, April 23, 2:30 p.m.

Venetian Room, Shamrock Hilton Hotel

1. (2:30) **Nonspecific Ulcerative Colitis, Surgical Indications and Management.**
Patrick H. Hanley, New Orleans.

2. (3:00) **Noninvasive Cancer in Hemorrhoids.**
Curtice Rosser, Dallas.
Discussion: Otho P. Griffin, Fort Worth.

3. (3:30) **Round Table Discussion.**

4. (5:00) **Business Meeting.**

7:30 p.m.

Hotel America

5. (7:30) **Banquet.**

TEXAS REHABILITATION CENTER OF GONZALES WARM SPRINGS FOUNDATION, MEDICAL ADVISORY COUNCIL

Medical Director—Marjorie Kirkpatrick, Gonzales.

Saturday, April 25, 2 p.m.

Venetian Room, Shamrock Hilton Hotel

1. (2:00) **Business Meeting.**

TEXAS SOCIETY OF ATHLETIC TEAM PHYSICIANS
(Related Organization)

President—J. Frank Beall, Nacogdoches.

Vice-President and Program Chairman—A. Ross Davis, Houston.

Secretary-Treasurer—Dan Spivey, Lufkin.

(The scientific program is open to all physicians, both members and nonmembers. Persons interested in joining the Society may write to Dr. Beall, 2516 North Street, Nacogdoches.)

Thursday, April 23, 1:30 p.m.

Bluebonnet Room, Shamrock Hilton Hotel

1. (1:30) **Opening Remarks and Presidential Address. Management of Trauma to a Solitary Kidney.**
J. Frank Beall, Nacogdoches.

Serious trauma to a solitary kidney is fortunately a rare occurrence. An example occurring in a child in whom conservative management resulted in recovery is described. Diagnosis and treatment of injuries is discussed, as well as the types and treatment of renal injuries in general. The importance of diagnostic procedures in determining degree of damage is stressed. The value of nonoperative treatment in management of such injuries is emphasized.

2. (1:50) **The Attitude of the Coach.**
Mr. Bill Yeoman, Houston.

3. (2:10) **Estimation of Physical Fitness of the Cardiovascular System.**
Albert S. Hyman, New York.

There is urgent need for simple tests to estimate physical fitness of the cardiovascular-pulmonary system in normal individuals. Recent computer studies in sports medicine have shown that a number of newly discovered correlation parameters may be readily employed by team physicians, physical educators, and coaches in such athletic examinations. These dynamic tests are based upon the observation of certain physiologic reactions produced by standardized exercise tolerance. Calculated in foot/pounds of work performance, multidiscipline reaction curves provide graphic data upon which adequate judgment may be based. The common parameters include pulse rate, blood pressure, vital capacity, inspiratory breath holding time, maximum expiratory pressure, maximum breathing capacity, the Dynamic Respiratory Index, and the Cardio-Pulmonary Index. The instrumental parameters include the electrocardiogram, ballistocardiogram, vibrocardiogram, phonocardiogram, arteriogram, and rheogram. The x-ray size of the heart and aorta constitutes the third group. In field and clinical practice, these parameters either singly or in multiple, are determined under three conditions of physiologic states: (1) after an initial resting period, (2) immediately after the given exercise tolerance test, and (3) after a five-minute resting period. Experience obtained in about 2,600 tests has shown that five grades of physical fitness of the cardiovascular-pulmonary system may be accurately determined. Slides and charts will illustrate these groups.

4. (2:30) **Athletic Injuries in the Winter Sports.**
Mark B. Coventry, Rochester, Minn.

5. (3:00) **Training the High School Athlete.**
Mr. Bobby Gunn, Beaumont.

(3:20) Coffee Break.

6. (3:35) **The Athletic Toll.**
A. Ross Davis, Houston.

7. (3:55) The Cardiac Athlete.
Albert S. Hyman, New York.

The cardiac athlete has always been a perplexing problem to team physicians, physical educators, and coaches. Individuals with advanced heart disease have been known to perform amazing feats of physical endurance while others with relatively insignificant cardiac abnormalities experience disturbing symptoms of the failing heart on only slight effort. The traditional belief that objective evidence of heart disease can be automatically correlated with functional capacity has finally been replaced by the physical fitness concept of structural change. In this concept, attention is focused upon actual performance of the cardiovascular system rather than upon the specific abnormalities present. In a recent large series of athletic examinations, five major types of cardiac disabilities predominated; in the order of frequency these were: (1) heart murmurs, (2) high blood pressure, (3) irregularities of rhythm, (4) persistent tachycardia, and (5) chest pain. Heart murmurs constituted about 64 per cent of the total group; one-half of these were considered to be functional. High systolic blood pressure was found in about 11 per cent; of these 92 per cent were psychosomatic in origin. The most common irregularity of rhythm was premature beats usually of ventricular origin. There was a close correlation between persistent tachycardia and the thyroid syndromes. Chest pain requires careful evaluation and is the most important cause for disqualification. Statistics on sudden death in athletes are presented.

8. (4:15) Panel Discussion.
Moderator: Louis J. Levy, Fort Worth.
Panelists: Harry L. Kaplan, Houston, Internist.
Howard G. Purnell, Houston, General Practitioner and Team Physician.
Edward T. Smith, Houston, Orthopedic Surgeon and Team Physician.

9. (4:45) Business Meeting.

TEXAS SOCIETY OF ANESTHESIOLOGISTS
(Related Organization)

President—Charles H. Gillespie, Temple.

Secretary-Treasurer—Leo S. M. Duflot, Galveston.

Program Chairman—William S. Derrick, Houston.

Friday, April 24, 4 p.m.

Azalea Room, Shamrock Hilton Hotel

(Joint Meeting of Texas Society of Anesthesiologists, Section on Obstetrics and Gynecology, and Section on Surgery.)

1. (4:00) Panel: Cardiac Arrest.
Moderator: Charles L. Eckert, Albany, N. Y.
Panelists: Oglesby Paul, Chicago.
Kenneth K. Keown, Columbia, Mo.
Willard M. Allen, St. Louis.
J. Alan Alexander, Houston.

(4:45) General Discussion.

Saturday, April 25, 4 p.m.

Second Floor Meeting Room, Jesse H. Jones Library Building, Texas Medical Center

2. (4:00) Executive Board Meeting.

7 p.m.

Doctors' Club, Jesse H. Jones Library Building, Texas Medical Center

3. (7:00) Social Hour.

4. (8:00) Dinner.

Address.
Rufus Hessberg, Houston.

Sunday, April 26, 8:30 a.m.
Azalea Room, Shamrock Hilton Hotel

William S. Derrick, Houston, Presiding.

5. (8:30) Report of Survey of Anesthesiologists' Activities in Texas.
Norman E. Halbrooks, Tyler.

6. (8:45) Post Spinal Headaches.
Arthur B. Tarrow, San Antonio.

7. (9:00) Patient Monitoring.
Stewart A. Wilber, Houston.

8. (9:30) Anesthetic Considerations for the Diabetic Patient.
Adolph H. Giesecke, Jr., Dallas.

9. (10:00) Developments in the American Society of Anesthesiologists.
Oliver F. Bush, Dallas.

(10:30) Recess.

10. (11:00) Anesthesia for Patients with Heart Disease.
Kenneth K. Keown, Columbia, Mo.

11. (11:30) Angiotensin.
John R. Derrick, Galveston.

12:15 p.m.

Bayou Room, Towers Hotel

12. (12:15) Luncheon.

13. (1:30) Business Meeting.

2:30 p.m.
Azalea Room, Shamrock Hilton Hotel

William S. Derrick, Houston, Presiding.

14. (2:30) Some Aspects of Patient Pre- and Post-anesthetic Care for Open Heart Procedures.
Nellie R. Luhn, Dallas.

15. (3:00) Newer Approaches to Block Anesthesia for the Extremities.
George P. Beck, Jr., Lubbock.

16. (3:30) The Recognition of Heart Failure in the Anesthetized Patient.
Kenneth K. Keown, Columbia, Mo.

TEXAS SOCIETY OF PATHOLOGISTS, INC.
(Related Organization)

President—Mervin H. Grossman, Dallas.

Secretary-Treasurer—Vernie A. Stembridge, Dallas.

(The Society will have its scientific session with the Section on Pathology Friday, April 24, 2:30 to 5 p.m. in Castilian A, B, and C of the Shamrock Hilton Hotel, and Saturday, April 25, 2:30 to 5 p.m. in the Azalea and Bluebonnet Rooms of the Shamrock Hilton Hotel. See Section Meetings section for details.)

(The Houston Society of Clinical Pathologists will sponsor a seminar on "Respiratory System, Gastrointestinal System, Genitourinary System and Bone," Saturday, April 25, 9 a.m. to 4 p.m. in the Jesse H. Jones Medical Library Auditorium, Texas Medical Center. The seminar will be conducted by Dr. Lewis B. Woolner, Professor of Pathology, Mayo Foundation Graduate School of Medicine, University of Minnesota, Rochester, Minn., and by Dr. Harold G. Jacobson, chief, Division of Diagnostic Radiology, Montefiore Hospital, Bronx, N. Y.)

Sunday, April 26, 9 a.m.

Walnut Room, Shamrock Hilton Hotel

1. (9:00) **Business Meeting.**

TEXAS SOCIETY OF PLASTIC SURGEONS
(Related Organization)

President—Raymond O. Brauer, Houston.

Vice-President—Dean C. Kipp, Dallas.

Secretary-Treasurer—Willard C. Sellman, Jr., Dallas.

Thursday, April 23, 9 a.m.

Grecian Room, Shamrock Hilton Hotel

1. (9:00) **Registration.**

2. (9:30) **Plastic Surgery in the Texas Prison System.**
Melvin Spira and S. Baron Hardy, Houston.

3. (9:45) **Kidney Transplantation.**
Lt. Col. J. R. Smith, Lackland Air Force Base.

4. (10:15) **Bone Grafts in Cleft Palate Reconstruction.**
John B. Lynch and Stephen R. Lewis, Galveston.

5. (10:45) **Production of Carcinoma in Burn Scars.**
Marvin Erons and John B. Lynch, Galveston.

(11:00) **Coffee Break.**

6. (11:15) **Skin Grafting in Epidermolysis Bullosum.**
David A. Grant, Fort Worth.

7. (11:30) **Simple Adhesive Elastic Sponge Compression Dressing.**
Bromley S. Freeman, Houston.

8. (11:45) **Palatal Shishkebab for Midface Fractures.**
Dean C. Kipp and E. Frank Dunton, Dallas.

9. (11:55) **Elastic Splint Construction with Dental Silicone Impression Material.**
Jordan Miller, Melvin Spira, and S. Baron Hardy, Houston.

12:30 p.m.

Continental Room, Shamrock Hilton Hotel

10. (12:30) **Luncheon.**
Report on International Symposium on Early Treatment of Cleft Lip and Palate, Zurich, Switzerland.
T. D. Cronin, Houston.

2 p.m.

Grecian Room, Shamrock Hilton Hotel

11. (2:00) **Round Table Discussion.**

12. (4:00) **Business Meeting (Members Only).**

7 p.m.

Cork Club, 5629 Fannin

13. (7:00) **Hospitality and Dinner-Dance.**

TEXAS SOCIETY ON AGING, CLINICAL MEDICINE SECTION
(Related Organization)

Chairman—Silas W. Grant, Hillsboro.

Chairman-Elect—Walter C. H. Kerr, Fort Worth.

Program Chairman—Allen M. Fain, Duncanville.

Sunday, April 26, 8:30 a.m.

Normandy A, Shamrock Hilton Hotel

1. (8:30) **Registration.**

2. (9:00) **Chairman's Remarks.**
Walter C. H. Kerr, Fort Worth.

3. (9:30) **Diagnosis and Treatment of Gouty Arthritis.**
Howard C. Coggeshall, Dallas.

4. (10:00) **Osteoporosis, Present Concepts.**
William K. Ishmael, Oklahoma City.

Osteoporosis remains a prevalent yet little understood bone disease. The cause remains unknown in many instances. Various types of osteoporosis will be differentiated, as well as other

disorders resulting in deterioration of bone. The relationship of osteoporosis to pain and its importance in the management of patients is stressed. Recent literature will be reviewed.

5. (10:30) **Treatment of Rheumatoid Arthritis.**
Evelyn V. Hess, Dallas.

6. (11:00) **Application of Hypnosis in Care of Cancer Patients.**
Oscar L. Morphis, Fort Worth.

(11:30) Recess.

12 Noon

Normandy B, Shamrock Hilton Hotel

7. (12:00) **Luncheon.**

2 p.m.

Normandy A, Shamrock Hilton Hotel

8. (2:00) **Conservative Care of the Back and Neck.**
Willliam K. Ishmael, Oklahoma City.

The painful spine results from three general contributing factors: organic disease, postural strain, and psychological problems. The bleak prospects for improvement of the condition, existing in the minds of both doctors and patients, stem from failure to recognize and correct all features contributing to the pain and disability. Differentiation of the contributing features in the painful back and neck will be made, and management discussed.

9. (2:30) **Diagnosis of Arthritis in Office Practice.**
Howard C. Coggeshall, Dallas.

10. (3:00) **Demonstration of Hypnosis Technique in Treatment of Cancer Patients.**
Oscar L. Morphis, Fort Worth.

11. (3:30) **Treatment of Infectious Arthritis.**
Evelyn V. Hess, Dallas.

12. (4:00) **Business Meeting.**

TEXAS STATE DIVISION, INTERNATIONAL COLLEGE OF SURGEONS

(R.S.V.P. Robert G. McCorkle, 7338 McHenry Street, Houston.)

Thursday, April 23, 6:30 p.m.

Doctors' Club, Jesse H. Jones Library Building, Texas Medical Center

1. (6:30) **Annual Dinner and Business Meeting.**

MESSAGES WILL NOT BE READ AT MEETINGS

Physicians are asked to check bulletin boards for indication of messages and to obtain messages from Message Center, Shamrock Hilton Hotel.

Exhibits

MOTION PICTURE PROGRAM

Friday, April 24, 10 a.m.

Nile Room, Shamrock Hilton Hotel

1. (10:00) **The Painful Breast—1963.***

2. (10:15) **The Doctor Is Cross-Examined—1960.***

This film carries the physician-witness through the second phase of his court appearance. It points out the different types of testimony which may be given by the attending physician and the examining physician. It discusses again the question of talking things over with the attorney before testifying. Primarily, it emphasizes the fact that the medical expert need have no fear of cross-examining so long as he prepares himself in advance, sticks to the facts of the case, does not volunteer testimony, and talks in language which the judge and jury can understand.

3. (10:45) **Football Injuries—Cause and Prevention—1963.***

4. (11:10) **Emergency Airways—Crisis in Action—1960.***

This film is a report of the medical problems encountered and techniques employed in creating emergency airways. Although the film is intended primarily for the otolaryngologist, surgeon, and medical student, it should also be helpful to nonsurgical personnel concerned with first aid in the emergency room and ambulance service, in combat, and in civil defense. The film advocates "tracheotomy drill" rehearsing visually and by palpation upon fellow students, colleagues, or relatives.

5. (11:35) **A New Diagnostic Technique for Stroke: Retrograde Cerebral Angiography—1962.***

This film demonstrates the importance of recognizing that there are differences in stroke mechanisms and that something can be done for many of the victims once a specific diagnosis is made. The story of a man who was hospitalized as a result of a slight stroke is demonstrated. Examination by a neurosurgeon, preoperative treatment, and the technique for exposure of the brachial artery, cannulation and injection of the radiopaque agent are shown. Also included are demonstration and description of different angiograms of other stroke patients.

6. (12:15) **Technique of Total Abdominal Hysterectomy—1963.**

The film demonstrates a simplified technique of total abdominal hysterectomy used at the Mayo Clinic for over 20 years. Emphasis is placed upon exposure and a section of all continuous structures that are subject to injury during hysterectomy, and methods of exposing the ureters throughout their pelvic course are demonstrated.

7. (12:40) **Is Smoking Worth It?—1963.***

This film is used to motivate high school students to discuss health hazards involved in cigarette smoking. It presents valid reasons for giving up smoking, and for those who have not begun to smoke, the advantages of not starting. There is also an adult sequence which dramatizes some of the difficulties encountered by adults in giving up cigarettes after a long period of smoking. It also shows the percentages of the increased death rate from lung cancer in the past 30 years; and demonstrates, with the use of a smoking machine and other laboratory techniques, the percentage of cancer-causing chemicals found in tobacco tar that remains in the smoker's lungs.

Saturday, April 25, 10 a.m.

Nile Room, Shamrock Hilton Hotel

1. (10:00) Diagnosis and Management of Fungous Infection of the Skin, Hair and Nails—no date.

2. (10:35) Penetrating Wounds of the Large Intestine—1962.

Mortality and morbidity from penetrating wounds of the colon have been reduced progressively during the 20th century through aggressive surgery. Technical procedures of choice, however, have remained controversial. The purpose of this film is to present one surgical approach used in various types of colon wounds and to illustrate its principles in two cases.

3. (11:00) Stroke—Early Restorative Measures in Your Hospital—1963.*

This film illustrates simple and effective methods and devices used in rehabilitation of stroke patients. The content of the film emphasizes early management and procedures in the rehabilitation of the stroke patient which should be instituted immediately upon admission to the general hospital. Employment of the film's method to prevent deformities, forestall psychological invalidism, and rescue stroke victims from unwarranted exile in institutions can mean returning patients from limbo to life.

4. (11:25) Clinical Medical Electronics—no date.

This film introduces the practicing physician to various medical electronic instruments now available for use in the office, hospital, laboratory, and home. Many of these are of advanced design and are of particular interest to the cardiologist, surgeon, neurologist, gastroenterologist, ophthalmologist, and biochemist. These new devices will be used in increasing frequency in coming years. Future possibilities and the field of biomedical engineering also are touched on briefly.

5. (12:00) Principles of the Local Burn Wound—1962.*

A program is outlined for management of the local burn wound which consists, in essence, of protection of the wound; mechanical cleanliness by soap-and-water washing combined with conservative debridement; employment of large sheets of skin, where possible, for grafts; and simplified techniques for handling of both transplants and donor sites.

6. (12:25) One Who Heals—1963.*

The role of the minister and the doctor in the care of the sick —a cooperative effort.

*These films as well as many others are available for use professionally and with lay groups. A brochure will be sent upon request from the Memorial Library of the Texas Medical Association.

SCIENTIFIC EXHIBITS

Scientific exhibits will be displayed in the Hall of Exhibits of the Shamrock Hilton Hotel Thursday from 12 noon until 5:15 p.m., Friday from 8 a.m. to 5:15 p.m., and Saturday from 8 a.m. to 5 p.m.

First, second, and honorable mention awards will be given in the four groups of exhibits: Group I, Individual; Group II, Group; Group III, Institutional; and Group IV, Educational-Promotional.

Exhibitors may be recognized by special ribbons attached to their identification badges.

Floor plans, showing location of scientific and technical exhibits, are available in the exhibit areas.

Following is a roster of exhibitors and their exhibits:

Group I, Individual Exhibits

Aorto–Iliac Surgery.
Jesse E. Thompson, M.D., Dale J. Austin, M.D., and C. Gene Wheeler, M.D., Dallas.

Organization of Cardiac Resuscitation.
Committee on Cardiovascular Diseases, Texas Medical Association.

Measly Muscle–Taenia Solium in Striated Muscle.
Morton H. Leonard, M.D., Z. W. Kosicki, M.D., L. W. Breck, M.D., O. H. Ellis, M.D., El Paso Orthopaedic Surgery Group, El Paso.

Detection of Unsuspected Cancer by Routine Sigmoidoscopy.
James S. O'Hare, M.D., Travis Clinic, Jacksonville.

Mammography in Private Practice.
Charles W. Yates, M.D., Leslie L. Lemak, M.D., John E. Martin, M.D., and James M. Keegan Associates, Houston.

Contrast Arthrography of the Knee Joint.
William P. Price, M.D., and Neill B. Longley, M.D., Houston.

Dynamic Activity of Vaginal Cells at the Molecular Level during Hormonal Administration, Estrogen and Progesterone.
Karl John Karnaky, M.D., Houston.

Group II, Group Exhibits

Basal Cell Nevus Syndrome.
J. B. Howell, M.D., Dallas; David E. Anderson, Ph.D., James L. McClendon, D.D.S., M. D. Anderson Hospital and Dental Branch, Houston.

Demineralized Whey Protein: Its Nutritional Value in Pediatrics.
P. T. McLoughlin, M.D., Wyeth Laboratories, Radnor, Pa.; A. M. Harmuth, M.D., Roselia Foundling Hospital, Pittsburgh.

Use of Low Molecular Dextran in Acute Myocardial Infarction.
Per H. Langsjoen, M.D., Temple.

Ten Per Cent Benzophrenone as a Sun Protectant.
M. Allen Forbes, Jr., M.D., and Mac Brannen, M.D., Austin.

Differentiation in Approach to Four Major Rheumatic Diseases.
Edward L. Tarpley, M.D., Nashville, Tenn.

Aortic Valve Replacement.
Arthur C. Beall, Jr., M.D., Thomas G. Nelson, M.D., Denton A. Cooley, M.D., and Michael E. DeBakey, M.D., Houston.

Open Heart Surgery Using Disposable Oxygenators and 5 Per Cent Dextrose Prime at Normothermia.
Denton A. Cooley, M.D., and Arthur C. Beall, Jr., M.D., Houston.

Study of Chronic Lung Disease by Inflation-Fixation Techniques.
Daniel E. Jenkins, M.D., David Bahar, M.D., S. Donald Greenberg, M.D., Robert M. O'Neal, M.D., and H. Irving Schweppe, Jr., M.D., Houston.

Use of Photography in Evaluation of Physical Disabilities.
Charles R. Peterson, M.D., Lewis A. Leavitt, M.D., and Richard Kenyon, Houston.

Antiarrhythmic and Antifibrillary Drugs with Especial Reference to Antazoline.
George R. Herrmann, M.D., Charles R. Secrest, M.D., Glory J. Vilbig, M.D., R. A. Quinn, M.D., and Anna H. Soyillial, Galveston.

Degenerative Diseases Involving Locomotion in Elderly.
Francis H. Stern, M.D., Philadelphia.

Cardiovascular Surgery During the First Year of Life.
Denton A. Cooley, M.D., and Grady L. Hallman, M.D., Houston.

Management of Leg Cramps in Pregnancy.
Herschel S. Murphy, M.D., Roselle, N. J.

Management of Surgical Complications in the Patient with Cystic Fibrosis.
Gunyon M. Harrison, M.D., and Franklin James Harberg, M.D., Houston.

Office Treatment of the Alcoholic.
Jackson A. Smith, M.D., and Lester H. Rudy, M.D., Chicago.

Nephrotic Syndrome in Adults.
P. A. Lang, M.D., A. R. Remmers, Jr., M.D., H. E. Sarles, M.D., and R. L. Gregory, M.D., Galveston.

Refractory Heart Failure: Pathophysiology and Therapy.
Albert N. Brest, M.D., Robert Seller, M.D., Gaddo Onesti, M.D., Charles Heider, M.D., and John Moyer, M.D., Philadelphia.

Modern Management of Hypertension in Private Practice.
Henry A. Cromwell, M.D., Houston.

Scintillation Photoscanning in Clinical Medicine.
Thomas P. Haynie, M.D., and William K. Otte, B.S., Galveston.

Organization of Cardiac Resuscitation.
Section of Cardiovascular Surgery, University of Texas Medical Branch, Galveston.

Radiologic Examination of the Larynx.
Melvin H. Schreiber, M.D., and Charles K. Hendrick, M.D., Galveston.

Use of Cryostat in Diagnosis of Squamous Carcinoma in Situ of Cervix.
R. Vernon Colpitts, M.D., and Michael L. Ibanez, M.D., Houston.

Topical Steroid Therapy of Ulcerative Colitis—A Ten Year Experience.
Marcel Patterson, M.D., John McGivney, M.D., Arnold Drake, M.D., and Helen Ong, M.D., Galveston.

Calculation of Radium Needle Implants Using a Digital Computer.
Marilyn Stovall, Robert J. Shalek, Gilbert H. Fletcher, M.D., Houston.

Group III, Institutional Exhibits

Carpal Tunnel Syndrome.
Charles W. Barrier, M.D., and Robert A. Murray, M.D., Temple.

Retinal Detachment.
Hermann Hospital Ophthalmology Department, Houston.

Hernia Repair with Polypropylene Mesh.
Francis C. Usher, M.D., Houston.

Otolaryngologist and the Hearing Aid Selection.
H. E. Maddox, III, M.D., Galveston.

Complications of Biliary Tract Surgery.
Gerald S. Dowdy, Jr., M.D., James L. Moore, M.D., Alfred E. Smith, M.D., and George W. Waldron, M.D., (deceased), Houston.

Group IV, Educational-Promotional Exhibits

Blood! What Quality?
Charles F. Pelphrey, M.D., T. G. Price, M.D., H. L. Harle, M.D., and J. R. Rainey, Jr., M.D., Austin.

Medicine and Religion.
Committee on Medicine and Religion, Texas Medical Association.

Hospital Cancer Registry.
American Cancer Society, Texas Division, Inc., Austin.

Doctor, Are You Sure of Your Lab Results?
Texas Society of Pathologists.

Have Your ECG Taken.
Texas Heart Association Houston, and Committee on Cardiovascular Diseases, Texas Medical Association.

Radiography in Differential Diagnosis of Heart Disease.
Texas Heart Association, Houston.

Eye Health of the Infant and Young Child—The Physician's Responsibility.
Texas Society for the Prevention of Blindness, Houston.

Liberty from Dystrophy.
Liberty Muscular Dystrophy Research Foundation, Inc., Liberty.

Radiological Health Services for Texas Physicians.
Committee on Nuclear Medicine, Texas Medical Association, and Division of Occupational Health and Radiation Control, Texas State Department of Health.

Syphilis Is Where You Find It.
City of Houston Health Department.

The Regulation of Dietary Fat.
Judith W. Boeck, Department of Foods and Nutrition, American Medical Association, Chicago.

Small Industry: An Opportunity for the Family Physician.
Henry F. Howe, M.D., Lee N. Hames, American Medical Association, Chicago.

The Role of the Family Doctor in Cerebral Palsy.
United Cerebral Palsy of Texas, Inc., Austin.

Texas Education Agency, Vocational Rehabilitation Division, Austin.

Texas Medical Assistants Association, Harris County Medical Assistants Society, Houston.

TECHNICAL EXHIBITS

All technical exhibits will be displayed in the Hall of Exhibits of the Shamrock Hilton Hotel. Representatives from the pharmaceutical industry, business equipment companies, insurance and investment companies, and other related fields will be present to discuss equipment and products with physicians. The Hall of Exhibits will be open Thursday from 12 noon to 5:15 p.m.; Friday from 8 a.m. to 5:15 p.m., and Saturday, from 8 a.m. to 5 p.m.

The President's Reception for Exhibitors will be held for a representative of each exhibiting company Thursday, 5:30 to 6:30 p.m., in the Garden Room of the Shamrock Hilton Hotel.

Floor plans, showing location of technical and scientific exhibits, will be available in the exhibit area.

Following is an alphabetical list of exhibits:

Abbott Laboratories, North Chicago, Booth 28.

Air-Shields, Inc., Hatboro, Pa., Booth 73.
Alcon Laboratories, Inc., Fort Worth, Booth 93.
Aloe Division, Brunswick Corp., Dallas, Booth 11.
Ames Co., Inc., Elkhart, Ind., Booth 16.
Armour Pharmaceutical Co., Chicago, Booth 49.
Arnar-Stone Laboratories, Inc., Mt. Prospect, Ill., Booth 81.
Astra Pharmaceutical Products, Inc., Worcester, Mass., Booth 40.
Atomic Energy Industrial Laboratories, Houston, Booth 24.
Ayerst Laboratories, Dallas, Booth 74.
Barnes-Hind Ophthalmic Products, Sunnyvale, Calif., Booth 87.
Bentex Pharmaceutical Co., Houston, Booth 9.
Blue Cross-Blue Shield of Texas, Dallas, Booth 94.
Borcherdt Co., Chicago, Booth 110.
Borden Co., Houston, Booths 115, 116, 117.
Breon Laboratories, Inc., New York, Booth 86.
Bristol Laboratories, Syracuse, N.Y., Booth 112.
Broilizer Sales, Los Angeles, Booth 83.
Brooke Laboratories, Dallas, Booth 101.
Brown Schools, Austin, Booth 89.
Burroughs Wellcome & Co., Inc., Tuckahoe, N. Y., Booth 68.
Burton, Parsons & Co., Inc., Washington, D. C., Booth 38.
Carnrick Laboratories, Summit, N. J., Booth 103.
Cary-Taylor Corp., Arlington, Booth 63.
Ciba Pharmaceutical Company, Summit, N. J., Booth 61.
Clark & Hart, Inc., Dallas, Booth 131.
Coca-Cola Co., Atlanta, Booth 136.
Contour Chair Lounge Co., Dallas, Booths 50, 51, 52.
Coreco Research Corp., New York, Booth 17.
Cutter Laboratories, Berkeley, Calif., Booth 66.
Dallas Radionics, Dallas, Booth 19.
Davies, Rose & Co., Inc., Boston, Booth 39.
Devereux Foundation, Victoria, Booth 67.
Dixon, Ford, Co., Dallas, Booth 85.
Dome Chemicals, Inc., New York, Booth 79.
Duke Laboratories, Inc., South Norwalk, Conn., Booth 71.
Edison, Thomas A., Industries, Inc., Houston, Booth 53.
Electrodyne Co., Inc., Garland, Booth 140.
Emerson Laboratories, Dallas, Booth 7.
Encyclopaedia Britannica, Chicago, Booth 41.
Endo Laboratories, Richmond Hill, N. Y., Booth 138.
Finley, Charles O., & Co., Austin, Booth 36.
First Texas Pharmaceuticals, Inc., Dallas, Booth 99.
Flint Laboratories, Inc., Morton Grove, Ill., Booth 59.
Geigy Pharmaceuticals, Ardsley, N. Y., Booth 45.
General Electric Co., X-Ray Department, Dallas, Booth 91.
Gerber Products Co., Fremont, Mich., Booth 132.
Gilbert X-Ray Co. of Texas, Dallas, Booth 8.
Great American Reserve Insurance Co., Dallas, Booth 44.
Great Books of the Western World, Chicago, Booth 82.
Health Insurance Council, New York, Booth 5.
Heinz, H. J., Co., Pittsburgh, Booth 100.
Holland-Rantos Co., Inc., New York, Booth 15.
International Pharmaceutical Corp., Norristown, Pa., Booth 139.
Johnson & Johnson, New Brunswick, N. J., Booth 108.
Kendall, C. B., Co., Inc., Indianapolis, Booth 22.
Knoll Pharmaceutical Co., Orange, N. J., Booth 105.
Kyle, W. A., Co., Houston, Booth 102.
Landry Pharmaceuticals, Inc., Houston, Booth 90.
Lanier Co., Houston, Booth 12.
Lederle Laboratories, Division of American Cynamid Co., Pearl River, N. Y., Booth 64.
Leeming, Thos., Co., New York, Booth 107.
Lilly, Eli, & Co., Indianapolis, Booth 62.
Lippincott, J. B., Co., Philadelphia, Booth 72.
Lloyd Brothers, Inc., Cincinnati, Booth 13.
Loma Linda Foods, Riverside, Calif., Booth 29.
Magill, A. E., & Co., Houston, Booth 121.
Majors, J. A., Co., Dallas, Booth 78.
Mead Johnson & Co., Evansville, Ind., Booth 133.
Medco Products Co., Inc., Tulsa, Okla., Booth 84.
Medical Protective Co., Fort Wayne, Ind., Booth 34.
Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa., Booth 70.
Merit Pharmaceutical Co., Inc., Houston, Booth 88.
Merrell, William S., Co., Cincinnati, Booth 137.
Metro Med., Inc., Houston, Booth 124.
Mission Pharmacal Co., San Antonio, Booth 35.
Mueller, V., & Co., Chicago, Booth 97.
Mutual Benefit Life Insurance Co., Newark, N. J., Booth 98.
National Drug Co., Philadelphia, Booth 126.
Neutrogena Corp., Santa Monica, Calif., Booth 143.
Organon, Inc., West Orange, N. J., Booth 75.
Ortho Pharmaceutical Corp., Raritan, N. J., Booth 69.
Pacific Medical Equipment Co., North Hollywood, Calif., Booth 111.
Pfizer, Chas., & Co., Inc., New York, Booth 26.
Pharmafac, Inc., Austin, Booth 6.
Physicians Products Co., Inc., Petersburg, Va., Booth 54.
Poythress, Wm. P., and Co., Inc., Richmond, Va., Booth 118.
Professional Pharmacal Co., Inc., San Antonio, Booth 120.
Reynolds, R. J., Tobacco Co., Winston-Salem, N. C., Booth 60.
Riker Laboratories, Inc., Northridge, Calif., Booth 76.
Ritter Co., Inc., Rochester, N. Y., Booth 135.
Robins, A. H., Co., Inc., Richmond, Va., Booth 14.
Roche Laboratories, Nutley, N. J., Booth 77.
Ross Laboratories, Columbus, Ohio, Booth 123.
Sandoz Pharmaceuticals, Hanover, N. J., Booth 92.
Savage Laboratories, Inc., Houston, Booth 18.
Schering Corp., Union, N. J., Booth 46.
Schmid, Julius, Inc., New York, Booth 47.
Searle, G. D., & Co., Chicago, Booth 144.
Seven-Up Developers of Texas, Waco, Booth 42.
Smith Kline & French Laboratories, Philadelphia, Booth 119.
Smith, Miller & Patch, Inc., New Brunswick, N. J., Booth 80.
Southern X-Ray Engineering Co., Houston, Booth 127.
Strasenburgh Laboratories, Rochester, N. Y., Booth 122.
Stuart Co., Division of Atlas Chemical Industries, Inc., Pasadena, Calif., Booth 125.
Surgical Mechanical Research, Inc., Los Angeles, Booth 10.
Syntex Laboratories, Inc., Palo Alto, Calif., Booths 109, 142.
Tab Products Co., San Francisco, Booth 4.
Tarco Aviation Service, Houston, Booth 43.
Terrell Supply Co., Inc., Fort Worth, Booth 37.

Texas Employers' Insurance Association, Dallas, Booth 113.
Texas Pharmacal Co., San Antonio, Booth 65.
U. S. Vitamin & Pharmaceutical Corp., New York, Booth 48.
Upjohn Co., Kalamazoo, Mich., Booth 114.
Wallace Laboratories, Cranbury, N. J., Booth 96.
Warner-Chilcott Laboratories, Morris Plains, N. J., Booth 134.
Westwood Pharmaceuticals, Buffalo, Booth 104.
White Laboratories, Inc., Kenilworth, N. J., Booth 106.
Winthrop Laboratories, Inc., New York, Booth 95.
W-T-S Pharmaceuticals, Rochester, N. Y., Booth 33.
Wyeth Laboratories, Philadelphia, Booth 27.

PLAN TO SEE

SCIENTIFIC AND TECHNICAL EXHIBITS

SEE PAGES 303-306 FOR DETAILS.

PROGRAM

Woman's Auxiliary

to the

Texas Medical Association

Forty-Sixth Annual Convention—Houston—April 23-26, 1964

Tickets to all functions may be obtained upon registration.

Wednesday, April 22

12:00 noon-4:00 p.m.—Registration, Mezzanine, Sheraton-Lincoln Hotel.

12:00 noon-4:00 p.m.—Hospitality Room Open, Massanet, Sheraton-Lincoln Hotel.

Thursday, April 23

8:00 a.m.-4:00 p.m.—Registration, Mezzanine, Sheraton-Lincoln Hotel.

9:00 a.m.-4:00 p.m.—Registration, Lobby, Shamrock Hilton Hotel.

8:00 a.m.-4:00 p.m.—Hospitality Room Open, Massanet, Sheraton-Lincoln Hotel.

12:30 p.m.—Executive Board Luncheon, East Ballroom Room, Sheraton-Lincoln Hotel. Mrs. Hamilton Ford, La Marque, President, presiding. (All County Presidents-Elect and incoming Board members are invited to attend.)
Invocation. Mrs. William K. Powell, Dallas.
Welcome. Mrs. R. Marion Johnson, Houston.
Response. Mrs. A. O. McCary, Freeport.
Presentation of President-Elect.
Greetings from Chairman of Texas Medical Association Advisory Committee to the Woman's Auxiliary, M. L. Towler, M.D., Galveston.
Introduction of Special Guests.
Presentation of Past Presidents.
Mrs. Payton R. Denman, Houston.
Business:
Appointment of Reading Committee: Mrs. Willis H. Jondahl, Harlingen; Mrs. Frank M. Posey, Jr., San Antonio; Mrs. Philip T. Weisbach, Beaumont.
Convention Rules.
Minutes.
Treasurer's Report.
Correspondence.
Recommendations of Officers and Committee Chairmen.
Announcements. Mrs. George H. Brandau, Houston, Convention Chairman.
Adjournment.

3:00 p.m.—Council Woman's Meeting and Tea, Mirador Room, Sheraton-Lincoln Hotel. Mrs. Van D. Goodall, Clifton, First Vice President, Organization and Membership Chairman, presiding.

6:30 p.m.—Past Presidents' Dinner, Gulf Coast Suite, Petroleum Club.

Friday, April 24

8:00 a.m.-4:00 p.m.—Registration, Mezzanine, Sheraton-Lincoln Hotel.

9:00 a.m.-4:00 p.m.—Registration, Lobby, Shamrock Hilton Hotel.

8:00 a.m.-4:00 p.m.—Hospitality Room Open, Massanet Room, Sheraton-Lincoln Hotel.

9:00 a.m.-12:00 noon—First Business Meeting, French Salon, Sheraton-Lincoln Hotel. Mrs. Hamilton Ford, La Marque, President, presiding.
Invocation. Mrs. William B. Allensworth, Mineral Wells.
Welcome. Mrs. George H. Brandau, Houston, Convention Chairman.
Response. Mrs. H. E. Griffin, Corpus Christi.
Credo and Pledge.
Mrs. Ramsay H. Moore, Dallas.

Eastern Region Reports (District and County).
Mrs. Melvin Marx, Clarksville, Eastern Regional Vice-President.
District 9. Mrs. Frank T. Smith, Jr., Sealy, Council Woman.
Austin-Grimes-Waller, Harris, East Harris (Chapter), Montgomery, Polk-San Jacinto, Walker-Madison-Trinity, and Washington-Burleson Counties.
District 10. Mrs. P. C. Caldwell, Beaumont, Council Woman.
Angelina, Hardin-Tyler, Jasper-Newton, Jefferson: Beaumont and Port Arthur Chapters, Liberty-Chambers, Nacogdoches, Orange, and Shelby-San Augustine-Sabine Counties.
District 11. Mrs. Hugh F. Rives, Jacksonville, Council Woman.
Anderson-Leon, Cherokee, Freestone, Henderson, Houston, Rusk-Panola, Smith, and Wood Counties.
District 15. Mrs. Henry K. Crawley, Kilgore, Council Woman.
Bowie, Camp - Morris - Titus, Cass - Marion, Gregg, Harrison, Red River, and Upshur Counties.

Western Region Reports (District and County).
Mrs. Russell L. Deter, Western Regional Vice-President.
District 1. Mrs. John D. Martin, El Paso, Council Woman.
El Paso, Pecos-Jeff Davis-Presidio-Brewster, and Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties.
District 2. Mrs. George Horton, Odessa, Council Woman.

Andrews-Ector-Midland, Dawson-Lynn-Terry-Gaines-Yoakum, Howard-Martin-Glasscock, and Nolan-Fisher-Mitchell-Scurry-Borden-Kent-Stonewall Counties.

District 3. Mrs. Ernest Nalle, Jr., Lubbock, Council Woman.
Armstrong-Donley-Childress-Collingsworth-Hall-Wheeler, Dallam-Hartley-Sherman-Moore, Deaf Smith-Parmer-Castro-Oldham-Swisher, Gray-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson, Hale-Floyd-Briscoe, Hardeman-Cottle-Foard-Motley-Dickens-King, Lamb-Bailey-Hockley-Cochran, Lubbock-Crosby-Garza, and Potter-Randall Counties.

District 4. Mrs. Roy F. Kemper, Coleman, Council Woman.
Brown-Comanche-Mills-San Saba, Coleman, Crane-Upton-Reagan, Kimble-Mason-Menard-McCulloch, Runnels, and Tom Green-Coke-Crockett-Concho-Irion-Sterling-Sutton-Schleicher Counties.

Northern Region Reports (District and County).
Mrs. James A. Hallmark, Fort Worth, Northern Regional Vice-President.

District 12. Mrs. Charles E. Gamble, Waco, Council Woman.
Bell, Bosque-Hamilton, Brazos-Robertson, Coryell, Erath-Hood-Somervell, Hill, Johnson, Limestone, McLennan, Milam, and Navarro Counties.

District 13. Mrs. William B. Allensworth, Mineral Wells, Council Woman.
Baylor-Knox-Haskell, Clay-Montague-Wise, Eastland-Callahan-Stephens-Shackelford-Throckmorton, Palo Pinto-Parker-Young-Jack-Archer, Tarrant, Taylor-Jones, Wichita, and Wilbarger Counties.

District 14. Mrs. Glenn D. Carlson, Dallas, Council Woman.
Collin, Cooke, Dallas, Denton, Ellis, Fannin, Grayson, Hopkins-Franklin, Hunt-Rockwall-Rains, Kaufman, Lamar-Delta, and Van Zandt Counties.

12:30 p.m.-2:30 p.m.—Luncheon Honoring County Presidents, Grand Ballroom, Sheraton-Lincoln Hotel. Mrs. Hamilton Ford, La Marque, President, presiding.
Invocation. Mrs. Donald E. Pohl, Austin.
Recognition of County Presidents.
Greetings from President of Woman's Auxiliary to the American Medical Association, Mrs. C. Rodney Stoltz, Watertown, South Dakota.
Greetings from President-Elect of Woman's Auxiliary to Southern Medical Association, Mrs. Jordan Kelling, Waverly, Mo.
Presentation of County Auxiliary Awards.
Mrs. Howard R. Dudgeon, Jr., Waco, Chairman, Historical-Awards Committee.

3:00 p.m.—Tea at Doctors' Club, honoring Mrs. Hamilton Ford, President; Past Presidents; 1963-64 Officers. (Transportation provided.)

7:30 p.m.—Report of the President of the Woman's Auxiliary, Mrs. Hamilton Ford, to the House of Delegates of the Texas Medical Association, Emerald Room, Shamrock Hilton Hotel.

Saturday, April 25

8:00 a.m.-4:00 p.m.—Registration, Mezzanine, Sheraton-Lincoln Hotel.

8:00 a.m.-4:00 p.m.—Hospitality Room Open, Massanet Room, Sheraton-Lincoln Hotel.

9:00 a.m.-12:00 noon—Second Business Meeting, French Salon, Sheraton-Lincoln Hotel. Mrs. Hamilton Ford, La Marque, President, presiding.
Invocation, Mrs. R. Cecil Marburger, Columbus.
Greetings from President of Texas Medical Association, Robert Mayo Tenery, M.D., Waxahachie.

Southern Region Reports (District and County).
Mrs. Paul Klinger, San Antonio, Southern Regional Vice-President.

District 5. Mrs. Herbert G. Liberty, Seguin, Council Woman.
Atascosa, Bexar, Comal, Gonzales, Guadalupe, Karnes-Wilson, Kerr-Kendall-Gillespie-Bandera, La Salle-Frio-Dimmitt, and Medina-Uvalde-Maverick-Val Verde-Edwards-Real-Kinney-Terrell-Zavala Counties.

District 6. Mrs. Willis H. Jondahl, Harlingen, Council Woman.
Bee-Live Oak-McMullen, Brooks-Duval-Jim Wells, Cameron-Willacy, Hidalgo-Starr, Kleberg-Kenedy, Nueces, San Patricio-Aransas, Refugio, and Webb-Zapata-Jim Hogg Counties.

District 7. Mrs. Robert Burns, Giddings, Council Woman.
Bastrop-Lee, Caldwell, Hays-Blanco, Lampasas-Burnet-Llano, Travis, and Williamson Counties.

District 8. Mrs. A. O. McCary, Freeport, Council Woman.
Brazoria, Colorado-Fayette, De Witt-Lavaca, Galveston, Victoria-Calhoun-Goliad, and Wharton-Jackson-Matagorda-Fort Bend Counties.

Reports of Committee Chairmen and Officers.
Recommendations from Executive Board.
Election of Officers.
Other Business.

12:30 p.m.-3:00 p.m.—Installation Luncheon and Style Show, staged by Battlestein's, Grand Ballroom, Sheraton-Lincoln Hotel, Mrs. Hamilton Ford, La Marque, President, presiding.
Invocation. Mrs. M. Lake Fowler, Jr., Galveston.
Courtesy Resolutions.
Mrs. Joseph L. Knapp, Dallas.
Installation of Officers.
Mrs. Mark H. Latimer, Houston.
Presentation of Gavel and President's Pin.
Mrs. Hamilton Ford, La Marque.
Acceptance of Gavel and President's Pin.
Mrs. W. D. Nicholson, Lake Jackson.
Presentation of Past President's Pin.
Mrs. R. C. L. Robertson, Houston.
Style Show.
Adjournment of 1963-64 Session.

3:30 p.m.-4:30 p.m.—Post-Convention Executive Board Meeting, French Salon, Sheraton-Lincoln Hotel. Mrs. W. D. Nicholson, Lake Jackson, President, presiding.
Invocation.
Mrs. E. W. Coyle, San Antonio, Chaplain.

Greetings from the President of Texas Medical Association, Max E. Johnson, M.D., San Antonio.
Introduction of TMA Advisors to the Woman's Auxiliary: A. O. McCary, M.D., Freeport; M. L. Towler, M.D., Galveston; (One to be appointed).
Introduction of Past Presidents.
Mrs. Joseph B. Foster, Houston.
Introduction of President-Elect.
Mrs. George Turner, El Paso.
Introduction of Officers, County Presidents, County Presidents-Elect, and Council Women.
Introduction of Committee Chairmen.
Business.
Announcements.
Adjournment.

Sunday, April 26

11:30 a.m.-12:00 noon—Memorial Service of Texas Medical Association and Woman's Auxiliary, Continental Room, Shamrock Hilton Hotel. Jared E. Clarke, M.D., Dallas, Chairman; Mrs. G. Bedford Brown, Angleton, Cochairman.
Organ Prelude. Mrs. Fred J. Stancliff, Houston.
Invocation. Dawson C. Bryan, D.D., Institute of Religion, Houston.
Special Music.
Mrs. William C. Owsley, Jr., Houston.
Memorial Address for Deceased Members of Woman's Auxiliary.
Mrs. G. Bedford Brown, Angleton.
Memorial Address for Deceased Physicians.
Jared E. Clarke, M.D., Houston.
Special Music. Mrs. Owsley.
Benediction. Dr. Bryan.
Organ Postlude. Mrs. Stancliff.

Related Activities

The report of the President of the Woman's Auxiliary to the Texas Medical Association will be given by Mrs. Hamilton Ford, La Marque, to the House of Delegates on Friday, April 24, 7:30 p.m., in the Emerald Room of the Shamrock Hilton Hotel. Members of the Auxiliary are invited to hear this report.

The Massanet Room, 3rd floor of the Sheraton-Lincoln, will serve as the Hospitality Room throughout the convention. Refreshments will be served through the courtesy of Great American Reserve Insurance Company of Dallas.

The Woman's Auxiliary to the Texas Medical Association will sponsor a tea at the Doctors' Club on Friday, April 24, at 3:00 p.m., honoring the President, Mrs. Hamilton Ford, Past Presidents, and 1963-64 Officers. Buses will be provided and will leave from the Sheraton-Lincoln promptly at 2:30 p.m.

Alumni banquets will be held at 8:00 p.m. on Saturday, April 25.

Fraternity parties will be held from 6:30 p.m. to 7:30 p.m. on Saturday night, April 25.

General information concerning baby sitters, sightseeing, antique shops, theaters, and so forth, may be obtained at the Auxiliary Information Desk on the Mezzanine of the Sheraton-Lincoln and at the TMA Information Desk in the Lobby of the Shamrock Hilton.

Registration and reservations for special Auxiliary functions may be made at the Auxiliary Information Desk in the Lobby of the Shamrock Hilton Hotel. Reservations must be made one day in advance of the event if made at the Shamrock Hilton, and will be handled as "Advance Registrations" at the Sheraton-Lincoln.

A shuttle bus, courtesy of Texas Medical Association, will provide transportation between the two hotels for the convenience of Auxiliary members.

Memorial Service will be held on Sunday, April 26, at 11:30 a.m. in the Continental Room of the Shamrock Hilton Hotel.

The Credo of a Doctor's Wife

1. I believe in the principles of the Woman's Auxiliary to the American Medical Association which foster fellowship, benevolence, education, and patriotism.

2. I do solemnly pledge that I will be loyal to the profession of medicine, and just and generous to its members.

3. I will not be adversely critical of Auxiliary objectives and ideals but will support its cause and will respect the rights of its members.

4. I solemnly promise to uphold the dignity of my husband's profession and will help him render service to humanity in seeking to secure for him the free unfettered practice of medicine.

Mrs. Frank N. Haggard, San Antonio.

Auxiliary Pledge

I pledge my loyalty and devotion to the Woman's Auxiliary to the American Medical Association. I will support its activities, protect its reputation, and ever sustain its high ideals.

Local Convention Chairmen

General Chairman. Mrs. George H. Brandau.
Cochairman. Mrs. R. Marion Johnson.
Social Chairman. Mrs. Richard G. Martin.
Hospitality Chairman. Mrs. Thomas J. Vanzant.
Courtesy Chairman. Mrs. J. Charles Dickson.
Decorations Chairman. Mrs. John E. Norris.
Finance Chairman. Mrs. E. L. Slataper.
Business Meetings Chairman. Mrs. John K. Glen.
Registration Chairman. Mrs. I. W. Moody.
Information Chairman. Mrs. L. L. D. Tuttle.
Publicity Chairman. Mrs. William C. Fisher, III.
Memorial Services. Mrs. Carlos R. Hamilton.
Mrs. J. Peyton Barnes.
Executive Board Luncheon.
Council Woman's Meeting and Tea.
Mrs. Thomas P. Shearer.
Luncheon Honoring County Presidents.
Mrs. J. Griffin Heard.
Installation and Style Show Luncheon.
Mrs. Thomas L. Royce.
Special Event. Mrs. Guy E. Knolle.

Officers and Committees

Past Presidents

Honorary Life Presidents

*Mrs. E. H. Cary, Dallas.
*Mrs. S. C. Red, Houston.
*Mrs. M. L. Graves, Houston.
*Mrs. W. A. Wood, Waco.
*Mrs. John O. McReynolds, Dallas.
Mrs. S. A. Collom, Texarkana.
*Mrs. E. V. DePew, San Antonio.
*Mrs. H. B. Trigg, Fort Worth.
*Mrs. Joe Gilbert, Austin.
*Mrs. H. C. Haden, Houston.
Mrs. O. M. Marchman, Dallas.
*Mrs. H. R. Dudgeon, Waco.
Mrs. G. V. Brindley, Temple.
Mrs. Frank N. Haggard, San Antonio.
*Mrs. Preston Hunt, Texarkana.
*Mrs. S. D. Whitten, Greenville.
*Mrs. John T. Moore, Houston.
*Mrs. R. B. Homan, El Paso.
Mrs. W. R. Thompson, Fort Worth.
Mrs. F. F. Kirby, Waco.
*Mrs. S. H. Watson, Waxahachie.
Mrs. Scott C. Applewhite, San Antonio.
Mrs. William Hibbitts, Texarkana.
Mrs. S. F. Harrington, Dallas.
Mrs. P. R. Denman, Houston.
Mrs. A. B. Pumphrey, Fort Worth.
*Mrs. Sam E. Thompson, Kerrville.
*Mrs. Charles B. Alexander, San Antonio.
Mrs. George Turner, El Paso.
Mrs. Edward C. Ferguson, Beaumont.
Mrs. S. Minter Hill, Dallas.
Mrs. Joseph B. Foster, Houston.
*Mrs. William M. Gambrell, Texas City.
Mrs. Oscar W. Robinson, Paris.
Mrs. Robert Farris Thompson, El Paso.
Mrs. E. W. Coyle, San Antonio.
Mrs. Mark H. Latimer, Houston.
Mrs. Joseph H. McCracken, Jr., Dallas.
Mrs. Richard C. Bellamy, Liberty.
Mrs. H. S. Renshaw, Fort Worth.
Mrs. John D. Gleckler, Denison.
Mrs. Haskell D. Hatfield, El Paso.
Mrs. Ramsay H. Moore, Dallas.
Mrs. Garland G. Zedler, Austin.
Mrs. R. C. L. Robertson, Houston.

*Deceased.

Honorary Life Members

Mrs. H. Leslie Moore, Dallas.

May Owen, M.D., Fort Worth.

Officers

President.—Mrs. Hamilton Ford, La Marque.

President-Elect.—Mrs. W. D. Nicholson, Lake Jackson.

First Vice-President.—Mrs. Van D. Goodall, Clifton.

Northern Vice-President.—Mrs. James A. Hallmark, Fort Worth.

Eastern Vice-President.—Mrs. Melvin Marx, Clarksville.

Southern Vice-President.—Mrs. Paul Klinger, San Antonio.

Western Vice-President.—Mrs. Russell L. Deter, El Paso.

Recording Secretary.—Mrs. Travis Smith, Abilene.

Corresponding Secretary.—Mrs. M. Lake Fowler, Jr., Galveston.

Treasurer.—Mrs. Ralph C. Patrick, Houston.

Publicity Secretary.—Mrs. Walter P. McCall, Ennis.

Parliamentarian.—Mrs. Garland G. Zedler, Austin.

Auxiliary Staff Secretary.—Mrs. Martha D. James, Austin.

Council Women

District 1.—Mrs. John D. Martin, El Paso.
District 2.—Mrs. George Horton, Odessa.
District 3.—Mrs. Ernest Nalle, Jr., Lubbock.
District 4.—Mrs. Roy F. Kemper, Coleman.
District 5.—Mrs. Herbert G. Liberty, Seguin.
District 6.—Mrs. Willis H. Jondahl, Harlingen.
District 7.—Mrs. Robert Burns, Giddings.
District 8.—Mrs. A. O. McCary, Freeport.
District 9.—Mrs. Frank T. Smith, Jr., Sealy.
District 10.—Mrs. P. C. Caldwell, Beaumont.
District 11.—Mrs. Hugh F. Rives, Jacksonville.
District 12.—Mrs. Charles E. Gamble, Waco.
District 13.—Mrs. William B. Allensworth, Mineral Wells.
District 14.—Mrs. Glenn D. Carlson, Dallas.
District 15.—Mrs. Henry K. Crawley, Kilgore.

Standing Committees

Advisory.—Mrs. R. C. L. Robertson, Houston, Chairman; Mrs. John D. Gleckler, Denison; Mrs. Haskell D. Hatfield, El Paso; Mrs. Ramsay H. Moore, Dallas; Mrs. Garland G. Zedler, Austin. Special Advisor to the President—Mrs. Ramsay H. Moore, Dallas.

Civil Defense.—Mrs. F. F. Rogers, Corpus Christi, Chairman; Mrs. H. E. Griffin, Corpus Christi, Cochairman.

Convention.—Mrs. George H. Brandau, Houston, Chairman; Mrs. R. Marion Johnson, Houston, Cochairman.

Courtesy Resolutions.—Mrs. Joseph L. Knapp, Dallas, Chairman.

Finance.—Mrs. R. E. Leslie, El Campo, Chairman; Mrs. Russell L. Deter, El Paso; Mrs. R. C. L. Robertson, Houston; Mrs. Ralph C. Patrick,

Houston; Mrs. Andrew G. Goesl, Texarkana, Mrs. Thomas P. Shearer, Houston.

Historical.—Mrs. Howard Dudgeon, Jr., Waco, Chairman; Mrs. W. L. Ford, Waco; Mrs. Charles Shellenberger, Waco; Mrs. Eldon B. Fine, Waco; Mrs. Walter B. King, Waco; Mrs. H. M. Anspach, Waco; Mrs. Wendel Louney, Gatesville; Mrs. Howard O. Smith, Marlin.

International Health Activities.—Mrs. Paul Collins, Wichita Falls, Chairman; Mrs. George T. Singleton, Wichita Falls, Cochairman; Mrs. J. Franklin Campbell, Fort Worth, Cochairman.

Legislation.—Mrs. John J. Andujar, Fort Worth, Chairman; Mrs. Warren Binion, Fort Worth, Cochairman, Mrs. W. H. McClure, Kermit, Western Region; Mrs. C. E. Willingham, Tyler, Eastern Region, Mrs. Earl Gaston, Kingsville, Southern Region.

Memorial Service.—Mrs. G. Bedford Brown, Angleton, Chairman.

Mental Health.—Mrs. Winston Cochran, Beaumont, Chairman, Mrs. C. M. Phillips, Levelland (Legislation); Mrs. Lee F. Scarborough, Austin (Rehabilitation), Mrs. E. P. Kalbaugh, Nederland (Education); Mrs. Bruce Beard, Fort Worth (Religion).

Nominating.—Mrs. R. C. L. Robertson, Houston, Chairman; Mrs. Glenn D. Carlson, Dallas; Mrs. L. Bonham Jones, San Antonio; Mrs. Richard B. Johns, Abilene; Mrs. I. L. Van Zandt, Fort Worth; Mrs. Joseph Greenwood, Temple; Mrs. Robert L. Carr, Lubbock.

Organization.—Mrs. Van D. Goodall, Clifton, Chairman; Mrs. James A. Hallmark, Fort Worth, Northern Region; Mrs. Melvin Marx, Clarksville, Eastern Region; Mrs. Paul Klinger, San Antonio, Southern Region; Mrs. Russell L. Deter, El Paso, Western Region.

Philanthropic Funds:

AMA-ERF.—Mrs. T. J. Archer, Austin, Chairman; Mrs. R. O. Swearingen, Austin, Cochairman; Mrs. B. J. Smith, Austin; Mrs. George W. Tipton, Austin; Mrs. Sam W. Wilborn, Austin.

Library Fund.—Mrs. V. C. Smart, Austin, Chairman; Mrs. A. F. Vickers, Austin, Cochairman.

Memorial Fund.—Mrs. Sam A. Alexander, Dallas, Chairman; Mrs. O. M. Marchman, Dallas, Cochairman.

Student Loan Fund.—Mrs. Wm. M. Palm, Houston, Chairman; Mrs. L. L. Bourdon, Houston, Cochairman; Mrs. M. L. Towler, Galveston; Mrs. Robert Sparkman, Dallas.

Program.—Mrs. C. R. Allen, Galveston, Chairman; Mrs. T. G. Blocker, Jr., Galveston; Mrs. C. T. Stone, Jr., Galveston; Mrs. Fred J. Wolma, Galveston.

Public Relations.—Mrs. James C. Price, Gonzales, Chairman; Mrs. A. E. Mgebroff, Yoakum, Cochairman; Mrs. Joseph L. Knapp, Dallas; Mrs. Robert Carr, Lubbock; Mrs. Walter B. Allen, Nacogdoches.

Publications:

Bulletin.—Mrs. Clinton E. Woodson, Wharton, Chairman.

News Letter.—Mrs. B. J. Smith, Austin, Editor; Mrs. Eugene P. Schoch, Jr., Austin, Coeditor.

Recruitment.—Mrs. Henry K. May, Lake Jackson, Chairman; Mrs. Tom C. Burditt, Abilene, T.A. F.N. Convention Chairman; Mrs. H. Ray Buzbee, Abilene, T.A.F.N. Convention Cochairman.

Reference.—Mrs. Horace A. Renshaw, Fort Worth, Chairman; Mrs. J. C. Terrell, Stephenville; Mrs. Edward W. Coyle, San Antonio.

Research and Romance of Medicine.—Mrs. O. W. Robinson, Paris, Chairman.

Revisions.—Mrs. John D. Gleckler, Denison, Chairman; Mrs. V. M. Longmire, Temple; Mrs. J. L. Jinkins, Galveston.

School of Instruction.—Mrs. Hal V. Norgaard, Denton, Chairman; Mrs. Scott Haggard, Denton, Cochairman.

Committees—Special Appointments

Doctors' Day.—Mrs. Mack Bowyer, Abilene, Chairman.

Medicine and Religion.—Mrs. Wm. J. Powell, Dallas, Chairman.

Rural Health.—Mrs. Edwin Franks, Iraan, Chairman.

Safety.—Mrs. Stephen B. Tucker, Nacogdoches, Chairman.

Senior Citizens.—Mrs. W. Lacey Smith, San Angelo, Chairman.

Science Fair.—Mrs. L. Bonham Jones, San Antonio, Chairman; Mrs. Frank M. Posey, Jr., San Antonio; Mrs. W. W. Bondurant, Jr., San Antonio; Mrs. Donald Kenedy, New Braunfels; Mrs. Charles Berger, New Braunfels.

TMA Advisory Committee.—Martin L. Towler, M.D., Galveston, Chairman; George W. Tipton, M.D., Austin; A. O. McCary, M.D., Freeport.

County Presidents

District 1

El Paso.—Mrs. Leigh Wilcox, El Paso.

Pecos-Jeff Davis-Presidio-Brewster (Big Bend).—Mrs. Edwin Franks, Iraan.

Reeves-Ward-Winkler-Loving-Culberson-Hudspeth.—Mrs. George Dowling, Kermit.

District 2

Andrews-Ector-Midland.—Mrs. John Hays, Andrews.

Dawson-Lynn-Terry-Gaines-Yoakum (Five Counties).—Mrs. Wayne C. Hill, Brownfield.

Howard-Martin-Glasscock (Permian Basin).—Mrs. John B. Norton, Big Spring.

Nolan-Fisher-Mitchell-Scurry-Borden-Kent-Stonewall (Colorado Basin).—Mrs. E. A. Rogers, Snyder.

District 3

Armstrong-Donley-Childress-Collingsworth-Hall-Wheeler (Greenbelt).—Mrs. Robert Clark, Memphis.

Dallam-Hartley-Sherman-Moore.—Mrs. Byron W. Wright, Dumas.

Deaf Smith-Parmer-Castro-Oldham-Swisher (Tierra Blanca).—Mrs. G. Lee Cranfill, Friona.

Gray-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson (Top O' Texas).—Mrs. Harvey Hays, Borger.

Hale-Floyd-Briscoe.—

Hardeman-Cottle-Foard-Motley-Dickens-King.—

Lamb-Bailey-Hockley-Cochran.—

Lubbock-Crosby-Garza.—Mrs. Leslie R. Ansley, Lubbock.

Potter-Randall.—Mrs. William E. Laur, Amarillo.

District 4
Brown-Comanche-Mills-San Saba.—Mrs. Wm. O. Sweet, Brownwood.
Coleman.—Mrs. John D. Murrell, Santa Anna.
Crane-Upton-Reagan.—
Kimble-Mason-Menard-McCulloch.—
Runnels.—
Tom Green-Coke-Crockett-Concho-Irion-Sterling-Sutton-Schleicher (Tom Green Eight).—Mrs. D. R. Reilly, San Angelo.
District 5
Atascosa.—
Bexar.—Mrs. Ralph Munslow, San Antonio.
Comal.—
Gonzales.—Mrs. David M. Shelby, Gonzales.
Guadalupe.—Mrs. George P. Bachman, Seguin.
Karnes-Wilson.—Mrs. Spencer Stubbs, Karnes City.
Kerr-Kendall-Gillespie-Bandera.—Mrs. W. H. Springall, Fredericksburg.
LaSalle-Frio-Dimmit.—
Medina-Uvalde-Maverick-Val Verde-Edwards-Real-Kinney-Terrell-Zavala (Nine Counties).—Mrs. Richard Rankin, Jr., Uvalde.
District 6
Bee-Live Oak-McMullen.—
Brooks-Duval-Jim Wells.—Mrs. Richard Penly, Falfurrias.
Cameron-Willacy.—Mrs. John Kuppinger, Harlingen.
Hidalgo-Starr.—Mrs. Ralph P. Panzer, Weslaco.
Kleberg-Kenedy.—Mrs. Walter Fosberg, Kingsville.
Nueces.—Mrs. James R. Riley, Corpus Christi.
San Patricio-Aransas-Refugio.—
Webb-Zapata-Jim Hogg.—Mrs. J. S. Reitman, Laredo.
District 7
Bastrop-Lee.—Mrs. Robert Burns, Giddings.
Caldwell.—
Hays-Blanco.—Mrs. B. M. Primer, Jr., San Marcos.
Lampasas-Burnet-Llano.—Mrs. Ray Shepperd.
Travis.—Mrs. R. N. Snider, Austin.
Williamson.—
District 8
Brazoria.—Mrs. F. C. Turner, Lake Jackson.
Colorado-Fayette.—Mrs. Cecil Marburger, Columbus.
DeWitt-Lavaca.—Mrs. A. E. Mgebroff, Yoakum.
Galveston.—Mrs. Reagan H. Gibbs, Galveston.
Victoria-Calhoun-Goliad.—Mrs. Winton Coleman, Victoria.
Wharton-Jackson-Matagorda-Fort Bend.—Mrs. Charles Bishop, Ganado.
District 9
Austin-Grimes-Waller.—Mrs. John C. Galbreath, Navasota.
Harris.—Mrs. George H. Brandau, Houston.
East Harris Chapter.—Mrs. Robert W. Pipkin, Baytown.
Montgomery.—
Polk-San Jacinto.—Mrs. Thomas L. Gardner, Livingston.
Walker-Madison-Trinity (Tri County).—
Washington-Burleson.—Mrs. W. F. Hasskarl, Brenham.
District 10
Angelina.—Mrs. Basil E. Atkinson, Jr., Lufkin.
Hardin-Tyler.—Mrs. John Tate, Kountze.
Jasper-Newton.—Mrs. Tommy Quick, Jasper.
Jefferson:
Beaumont Chapter.—Mrs. Philip T. Weisbach, Beaumont.
Port Arthur Chapter.—Mrs. Dan H. Byram, Port Arthur.
Liberty-Chambers.—Mrs. C. W. Castle, Liberty.
Nacogdoches.—Mrs. Stephen B. Tucker, Nacogdoches.
Orange.—Mrs. Joseph Tarride, Orange.
Shelby-San Augustine-Sabine.—
District 11
Anderson-Leon.—Mrs. Curtis R. Jordan, Palestine.
Cherokee.—Mrs. Harbert Davenport, Jacksonville.
Freestone.—
Henderson.—
Houston.—
Rusk-Panola.—Mrs. Paul Suehs, Henderson.
Smith.—Mrs. Madison J. Lee, Tyler.
Wood.—
District 12
Bell.—Mrs. Charles Gillespie, Temple.
Bosque-Hamilton.—Mrs. W. F. Key, Clifton.
Brazos-Robertson.—Mrs. K. L. Nelson, Bryan.
Coryell.—
Erath-Hood-Somervell (Tri County).—Mrs. D. L. Richardson, DeLeon.
Falls.—
Hill.—
Johnson.—
Limestone.—
McLennan.—Mrs. G. I. Anderson, Waco.
Milam.—Mrs. Elmer Oswald Smith, Jr., Cameron.
Navarro.—Mrs. Robert D. Mertz, Corsicana.
District 13
Baylor-Knox-Haskell.—
Clay-Montague-Wise.—
Eastland-Callahan-Stephens-Shackelford-Throckmorton.—Mrs. J. T. Lawrence, Breckenridge.
Palo Pinto-Parker-Young-Jack-Archer.—Mrs. Earl Russell, Weatherford.
Tarrant.—Mrs. W. V. Bradshaw, Jr., Fort Worth.
Taylor-Jones.—Mrs. Richard B. Johns, Abilene.
Wichita.—Mrs. George Slaughter, Wichita Falls.
Wilbarger.—Mrs. John Hardin, Jr., Vernon.
District 14
Collin.—Mrs. Leo L. Fitzgerald, Plano.
Cooke.—Mrs. Kenneth Dobbs, Muenster.
Dallas.—Mrs. John B. Bourland, Dallas.
Denton.—Mrs. S. J. Pedigo, Denton.
Ellis.—Mrs. John Compton, Waxahachie.
Fannin.—
Grayson:
South.—Mrs. Morris Britton, Sherman.
North.—Mrs. W. Herbert Brown, Denison.
Hopkins-Franklin.—Mrs. W. Ray Hanna, Sulphur Springs.
Hunt-Rockwall-Rains.—Mrs. Van Kaden, Greenville.
Kaufman.—
Lamar-Delta.—Mrs. C. E. Beachley, Jr., Paris.
Van Zandt.—
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Volunteers Organize Glaucoma-Detection Programs

Almost a million people in the United States are slowly losing their sight because of ignorance and neglect. That glaucoma can creep in insidiously and destroy sight sometimes within hours is unknown to much of the general population.

Yet statistics published by the National Society for the Prevention of Blindness indicate that one out of every 50 Americans over 40 years of age will have glaucoma.

To combat the devastating effects of glaucoma, Lions clubs, Junior Chambers of Commerce, Kiwanis clubs, and Delta Gamma alumnae groups in conjunction with the Texas Society for the Prevention of Blindness under the direction of medical advisory committees have organized glaucoma-detection programs. In November, 1963, 15,504 people in Texas had been screened for glaucoma in these programs.

It was just three years ago that the first public glaucoma-detection program was held in Texas. Under the direction of the Texas Society for the Prevention of Blindness and the Houston Ophthalmological Society, with the Houston Junior Chamber of Commerce, members of the Houston Delta Gamma Alumnae Chapter, and registered nurses of the Houston-Harris County Chapter-American Red Cross participating as volunteers in September, 1960, 761 persons were screened for glaucoma in Houston. Ten persons in the program had positive cases of glaucoma, four were suspicious, and 21 did not report back.

Since that time glaucoma-detection programs have been held in Houston, Dallas, Fort Worth, Longview, Breckenridge, Amarillo, Freeport, and Corpus Christi.

Six hundred and forty-one persons have been referred to physicians from these programs, and 84 positive cases of glaucoma have been reported. Seventy-seven cases of suspicious glaucoma were noted, and there were other patients that did not report back.

Mrs. Pauline Outland, Houston, executive director of the Texas Society for the Prevention of Blindness, comments: "Even though many positive and suspicious cases of glaucoma are detected in these community programs, the primary objective is education."

For that purpose, films such as "Hold Back the Night" have been shown, and educational material has been distributed at these programs.

All public glaucoma-detection programs which have been held in Texas have been cosponsored and coordinated by the Texas Society for the Prevention of Blindness under the direction of the Texas Ophthalmological Association and local medical societies.

More than one city has had trouble in establishing the glaucoma-detection program. Organization, assistance, publicity, and equipment are just a few of the areas to be considered. In time, however, these problems have been ironed out.

Lon D. Edwards, chairman, Glaucoma Screening Program for the seven Lions Clubs of Amarillo, said, "We at Amarillo struggled along for a year or more in our

A Delta Gamma alumna records the physician's findings as a volunteer assists in the 1962 glaucoma-detection program in Houston. Of 1,919 persons screened at this program, 98 were referred to physicians for further examinations. Ten positive cases of glaucoma and ten suspicious cases were found. Thirty-three patients did not report back.

planning before we knew that there was a Texas Society, an affiliate of the National Society for the Prevention of Blindness. When we finally got in touch with Mrs. Outland of the Texas Society and her State Medical Advisory Committee, and they agreed to help us in the program, our problems dissolved very fast."

The Amarillo group found that a program for screening 500 persons was outlined in detail by the Texas Society for the Prevention of Blindness under the direction of the local medical society. Educational literature was furnished for the program.

Since ophthalmologists are required for the glaucoma-detection programs, most of the programs have been held in large or middle-sized cities. Breckenridge is an exception. The program in this town of 6,273 was sponsored by the Texas Society and the Breckenridge Lions Club. The program was coordinated by the Texas Society under the direction of the Tarrant County Ear, Eye, Nose, and Throat Society. In this program, 652 people were screened, eight were referred to physicians, and two were positive glaucoma cases.

"Has it ever occurred to you that I might only look fat because you're a radiologist?"

Information on conducting glaucoma-detection programs and literature on glaucoma may be obtained from Mrs. Pauline Outland, Texas Society for the Prevention of Blindness, 4705 Montrose Boulevard, Room 202, Houston 6.

Information for Authors

Material for the *Texas State Journal of Medicine* may be sent to the Managing Editor, 1801 North Lamar Blvd., Austin 78701. It must be offered solely to this journal. Each paper is reviewed by a consultant specialist and an editorial board, and is accepted or rejected on the basis of its individual merit and the availability of other material.

Manuscripts should be typed double-spaced with ample margins on firm paper. Carbons are unacceptable, but the author should keep a copy.

The *title* should include the words most suitable for indexing the article in "Index Medicus," should stress the main point, and should be brief. Subtitles should not be connected grammatically with the main title.

The *authors' names* and degrees should follow the title, and their mailing addresses should be listed at the end of the article. A glossy picture of the senior author is needed.

The *text* should be narrative with complete sentences, *few abbreviations*, and logical subheadings. For *spelling and usage* the editors follow "Dorland's Illustrated Medical Dictionary," 23rd edition, and "Webster's Third New International Dictionary, Unabridged." *Quotations* must give credit to both author and source. A concise *summary* or conclusion is of professional value.

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Phenothiazine Derivatives

THESE MEDICINAL AGENTS enjoy extensive therapeutic applications in numerous capacities: tranquilizers, antiemetics, and others. Domino[4] has surveyed the pharmacology in his classic review, "Human Pharmacology of Tranquilizing Drugs."

Although phenothiazine per se has been recognized as a urinary antiseptic and anthelmintic for many years, its toxicity has limited its therapeutic utility. However, in 1945 the advent of the antihistaminic, promethazine, and its centrol nervous system depressing properties led many medicinal chemists to explore the field of phenothiazine derivatives. In 1950 Charpenter created the now well-known classic, chlorpromazine. Subsequently, many researchers have examined the field from the structure-activity viewpoint.[4, 8]

Domino has also comprehensively studied the pharmacodynamics on a biochemical basis.[4] Emerson and Miya[5] have reviewed the literature on the metabolism of the phenothiazines. It can be concluded that these compounds undergo a number of metabolic transformations: that is, sulfoxidation, N-demethylation, N-oxide formation, and hydroxylation. The metabolic product arising via sulfoxidation of chlorpromazine, chlorpromazine-sulfoxide exhibits only one-tenth the pharmacologic activity of the parent compound. One the other hand, desmethylchlorpromazine possesses activity that approximates the potency of chlorpromazine.

Gordon's interpretations[8] constitute a very important contribution to the field of phenothiazine derivatives. These interpretations include certain structure-activity correlations which have proved to be useful in predicting the activity of new ring substituents and side chains, as well as guiding the medicinal chemist to the synthesis of new active congeners. Furthermore, these structure-activity studies might ultimately provide the premise for the elucidation of a concise biochemical explanation of the mode of action.

Recently, Bhargava and Chandra[1] have surveyed the antiemetic properties in relation to chemical structure among the phenothiazines. As early as 1953 Friend and Cummins[6] reported that chlorpromazine was effective in suppressing nausea and vomiting caused by a wide variety of clinical conditions such as carcinomatosis, labyrinthitis, lymphomatosis and uremia. In addition, these clinical investigators found this drug to be effective in preventing or controlling the nausea and vomiting caused by disulfiram (Antabuse, Ayerst) administration to patients under the influence of alcohol, and that produced by aureomycin, a folic acid antagonist, codeine, meperidine, methadone, morphine, nitrogen mustard, protoveratrine, oxtetracycline and urethane therapy.

Courvoisier and others[3] also demonstrated the antiemetic activity of chlorpromazine against vomiting induced in dogs by apomorphine. Brand's group[2] studied the efficacy of chlorpromazine in the treatment of vomiting induced by various drugs. With dogs, morphine, ergot, intravenous and oral copper sulfate, lanatoside C and Veriloid were used to bring about emesis. These workers also studied the ability of chlorpromazine to prevent vomiting in cats caused by apomorphine, lanatoside C, Veriloid, nitrogen mustard, pilocarpine, and intravenous copper sulfate. Results showed that in dogs, chlorpromazine was highly effective in antagonizing apomorphine-induced emesis; however, phenergan, an antihistaminic congener of chlorpromazine, demonstrated no protective effect. With this test animal, chlorpromazine was effective also against vomiting invoked by morphine and ergot but was ineffective against intravenous or oral copper sulfate; lanatoside C and Veriloid. The mechanism of its action in dogs was suggested as being a selective depression of the medullary emetic chemoreceptor trigger zone. These investigators also state that the results with cats appear to indicate that there are anatomic and pharmacologic differences in the emetic chemoreceptor trigger zone of cats

and dogs. With cats chlorpromazine did not prevent vomiting induced by apomorphine, lanatoside C, intravenous copper sulfate, nitrogen mustard and Veriloid.

Glaviano and Wang[6] concluded from their experiments with dogs that chlorpromazine may act as an antiemetic by two mechanisms depending on the dose administered. Their results suggest that the primary mechanism is a selective action on the chemoreceptive emetic trigger zone. The more potent effect of larger doses is depression of the reticular vomiting mechanism.

Rosenkilde and Govier[10] compared four phenothiazine derivatives with chlorpromazine on the basis of their inhibitory potencies in apomorphine induced emesis using the dog as the test animal. On intravenous injection, perphenazine (Trilafon, Schering) was approximately 24 times and prochlorperazine three times more effective than chlorpromazine. With the small doses administered no side effects were observed; however, two compounds, mepazine and promazine showed little or no activity in the doses used. Mepazine failed to protect the dogs from vomiting in doses of 1.5, 3 or 6 mg. per kg. while promazine was inactive at 1.5 and 3 mg. per kg. At 6 mg. per kg. the latter drug did suppress vomiting.

When 0.1 mg. per kg. of apomorphine HCl was given to the dogs, Wang[11] found that perphenazine was 16.6 times more potent than chlorpromazine as an antiemetic. However, when the dose of the emetic was increased to 0.5 mg. per kg., the potency ratio was raised to 47.8 times, indicating that perhaps perphenazine gained in relative efficacy as the emetic stimulus was increased. It was stated that perphenazine might be considered a highly versatile antiemetic because it is highly effective against vomiting induced by morphine, ergot and especially lanatoside C. The latter drug is notoriously known to provoke repeated bouts of vomiting lasting three or four hours. This long duration of emetic action indicates that the binding between the receptor site and such emetic agents as morphine and lanatoside C might be so effective that chlorpromazine is unable to displace these drugs. However, in the case of perphenazine, the increased potency and versatility might well be related to its great affinity for the receptors in the trigger zone.

The antiemetic activities in dogs of fluphenazine, perphenazine, trifluprazine and chlorpromazine were determined and the duration of their protective effects against 20 μg. per kg. of apomorphine administered intravenously were established by Laffan and others.[11] Estimations of the relative potency of each drug on the basis of protection-duration indicate that, subcutaneously, fluphenazine is 34 to 124 times as potent as chlorpromazine, and 4.2 to 7.5 and 16.7 to 19.0 times as potent as perphenazine and trifluprazine respectively, in reducing the emetic susceptibility of dogs to apomorphine. None of the phenothiazines afforded significant protection against the emetic effects of lanatoside C, nicotine, veratrum viride or nitrogen mustard. The greater antiemetic activity of fluphenazine as compared with trifluprazine was not paralleled by a correspondingly greater sedative action, since pretreatment with more than 30 times the threshold antiemetic dose of fluphenazine did not alter pentobarbital-induced sleeping times; however, an equivalent dose of trifluprazine extended sleeping time almost two-fold.

In an attempt to resolve some discrepancies in the results of different investigators, Bhargava and Chandra[1] compared the antiemetic activities of 12 phenothiazine derivatives under the same environmental and experimental conditions. These investigators also attempted to correlate the activity of these compounds with chemical structure. Results of the study show that thioproperazine and perphenazine were 300 and 160 times as potent, respectively, as chlorpromazine. The following shows the relative potency of the compounds tested: thioproperazine > perphenazine > fluopromazine > trifluoperazine > thiopropazote > pipamazine > prochlorperazine > methyltrifluoperazine > chlorpromazine > pecazine. Thioridazine and chlorpromazine sulfoxide, a metabolite of chlorpromazine, were found to be inactive.

The antiemetic activity is found only in the parent compounds containing bivalent sulfur (Fig. 1).[1] For a phenothiazine deriva-

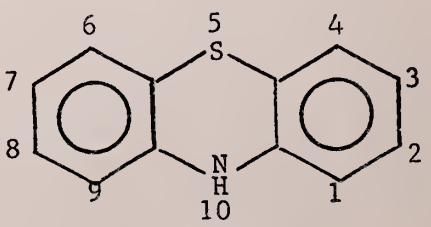

Fig. 1. Phenothiazine.

tive to possess antiemetic activity it is essential that an aminoalkyl group be attached to the nitrogen atom of the phenothiazine nucleus. All active compounds have a three carbon chain between the terminal and phenothiazine ring nitrogen atoms; however, branching in this chain tends to reduce the activity since the methyltrifluoperazine derivative (branched chain) is less active than trifluoperazine, an unbranched chain compound. A reduction in antiemetic activity also occurs when part of the side chain is incorported into a piperidine ring.

Of the derivatives possessing the optimum three carbon atom side chain, the 3-piperazin-1-ylpropyl compound (perphenazine) is more active than the corresponding 3-dimethylaminopropyl derivative (chlorpromazine). Among the 3-piperazinylpropyl phenothiazines, the 4-(2-hydroxyethyl) group of perphenazine gives greater activity than the corresponding 4-methyl group of prochlorperazine. It has been observed that when the free hydroxy group of perphenazine is acetylated, e.g., thioproperazine, there is a ten-fold reduction in the activity of the compound.

The antiemetic activity of the derivatives may be enhanced by replacing the hydrogen atom at position 2 in the phenothiazine nucleus with radicals possessing high electron attracting properties. This is illustrated with the following piperazinyl derivatives listed in increasing order of activity: prochlorperazine, trifluoperazine, and thioproperazine, which possesses respectively as the 2 substituent, a chlorine atom, a trifluormethyl group and dimethyl aminosulfone group.

Fruitful structure-activity studies have been facilitated by research advances in synthetic medicinal chemistry and pharmacodynamics.[4, 8] Now that a multiplicity of phenothiazine derivatives has been made available for pharmacologic evaluation and comparison with related tranquilizers, it is possible to deduce general relationships between chemical structure and pharmacologic action. A brief summary of the major structure-activity relationships may be delineated into two phases: first a consideration of chemical modifications applied to the alkylamino side chain on the 10 position of the phenothiazine cyclic system; secondly, the substituents on the 2 position.

Generally, congeners possessing the β-amino-ethyl side chain in position 10 with two carbon atoms between the two nitrogens exhibit pronounced antihistaminic activity: that is, promethazine and pyrathiazine, which are well known antihistaminics. Analogs with a γ-aminopropyl side chain with a distance of three carbon atoms between these two nitrogen positions exert marked effects in blocking conditioned avoidance behavior, as well as adrenergic blockade. Substitution of a piperazine heterocycle in place of the terminal dimethylamino group leads to an increase in potency: consider prochlorperazine and chlorpromazine. Additionally, replacement of the terminal methyl group on the piperazine cycle with a hydroxyethyl results in a further increase in central activity; this effect can be exemplified with the case of perphenazine vs. prochlorperazine. In this connection it is noteworthy to mention that the introduction of the p-aminophenethyl substituent in place of the terminal methyl on the piperazine moiety leads to a congener[10] with nine-fold the chlorpromazine activity.

Regarding substituents on the 2 position of the phenothiazine system, it is important to summarize the following conclusions: substitution of chlorine for the hydrogen atom generally leads to compounds with higher central potency (that is, chlorpromazine and promazine). In this regard a methoxyl group is almost as effective as the halogen; compare methoxypromazine with chlorpromazine. The trifluoromethyl group in position 2 effects a significant increase in potency as demonstrated with the highly active triflupromazine, trifluperazine, and fluphenazine. Gordon and others[8] have studied novel phenothiazines with trifluoromethylsulfide and trifluoromethyl sulfone groups on the same 2 nuclear position. These compounds are interesting not only because they have proved to be active in effecting blockade of the conditioned escape response, but also because they have led to the revelation of relations between iogenicity and activity. These correlations might account for the difference between negative effect exerted by the methylsulfone group (ionizable) when compared with the positive effect of the trifluoromethylsulfone group (non-ionizable) on activity.

Studies such as this one by Gordon and others[8] are considered major advances in the field of phenothiazine medicinals because such structure-activity relationships have proved their applicability in predicting the effect of chemical modifications on biologi-

cal properties. Gordon's work[8] also exemplifies one of the objectives of medicinal chemistry research; that is, the interpretation of structure-activity relationships as guides to the synthesis of new bioactive compounds.

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American Academy of GPs To Meet in Atlantic City

Family doctors from all parts of the United States will gather in Atlantic City April 13-16 for the 16th annual scientific assembly of the American Academy of General Practice. More than 4,000 doctors, their families, and ancillary medical persons are expected.

Theme of the 1964 Assembly will be "The Family in Medical Perspective," an extension of a comprehensive program called "Perspectives in Medicine," which was inaugurated at Chicago last year.

Additional information may be secured by writing William R. DeLay, The American Academy of General Practice, Volker Blvd. at Brookside, Kansas City 12, Mo.

THE BURNS INSTITUTE of the Shriners Hospital for Crippled Children, Galveston, seen in an architect's rendering, will be the first institute of its kind in the world. The Shrine has allocated $10,000,000 for the construction of three such institutes, in Galveston, Cincinnati, and Boston, in an effort to solve a problem many medical authorities have called the greatest hazard of childhood—severe burns. Interim units at Galveston and Cincinnati have already begun operations, and construction of the Galveston unit will begin this spring.

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1. Appointed October, 1963, to replace Troy A. Shafer, Harlingen, who died on Oct. 9, 1963.
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3. Interim appointment Sept. 27, 1963, to fill vacancy created when Charles W. Castle, Liberty, became Councilor.

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4. Appointed October, 1963, to replace Troy A. Shafer, who died on Oct 9, 1963.
5. Appointed October, 1963, to replace Harvey Renger, Hallettsville, who was appointed delegate to the AMA.

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Vacancy.[8]
Robert Mayo Tenery, Waxahachie.

Committee on Medical History (standing)
William M. Crawford, Fort Worth, Chairman (1965).
G. V. Brindley, Sr., Temple (1965).
W. D. Thames, Jr., Lufkin (1965).
Morris Polsky, Austin (1964).
W. C. Dine, Amarillo (1964).
Gerald A. King, Fort Worth (1966).
Lyman Blair, Houston (1966).
Joe C. Rude, Austin (1966).
Herbert Donnell, Waxahachie (1964).
W. B. Russ, San Antonio (Emeritus Member).
L. H. Reeves, Fort Worth (Emeritus Member).
H. Reid Robinson, Galveston (Emeritus Member).
Pat I. Nixon, San Antonio (Emeritus Member).

Council on Annual Session

Committee on General Arrangements for Annual Session (special)
A. O. Fly, Houston, Chairman.
B. F. Kitchen, Jr., Houston, Vice Chairman.

Committee on Scientific Exhibits (standing)
Jasper H. Arnold, Houston, Chairman (1964).
John A. Welty, Harlingen (1965).
Charles C. Jones, Jr., Kerrville (1965).
J. Frank Beall, Nacogdoches (1965).
Walter Long, Austin (1964).
Nathan Cedars, Stephenville (1966).
David Schrum, Houston (1966).
W. L. Carrington, Mexia (1964).
J. L. Jackson III, Wichita Falls (1966).

6. Appointed November, 1963, to replace N. L. Barker, Paris, who resigned October, 1963, to accept appointment to Board of Trustees.

7. Appointed December, 1963, to replace Courtney M. Townsend, Paris, who resigned November, 1963, to accept appointment to Council on Medical Jurisprudence.

8. Vacancy created by death of George Waldron, Houston, on Feb. 15, 1964.

Council on Medical Education and Hospitals

Committee for American Medical Association Education and Research Foundation (standing)
Herbert A. Bailey, Dallas, Chairman (1964).
Edward S. Crocker, Houston (1965).
E. M. Addison, Huntsville (1965).
Douglas B. Black, Lamesa (1965).
H. E. Whigham, McAllen (1964).
Nelson L. Schiller, Austin (1964).
J. C. Terrell, Stephenville (1966).
P. W. Voltz, Jr., San Antonio (1966).
C. V. Chenoweth, Tyler (1966).

Advisers to Student American Medical Association
Homer A. Taylor, Jr., Houston (Baylor).
Charles LeMaistre, Dallas (Southwestern Medical School).
E. Sinks McLarty, Galveston (University of Texas Medical Branch).

Appointees to Texas Commission on Patient Care
Joseph F. McVeigh, Fort Worth (1964).
G. E. Brereton, Dallas (1964).
A. J. Bankhead, Tyler (1964).
Joseph C. Magliolo, Dickinson (1964).

Committee on Hospital Accreditation (standing)
Ray L. Shepperd, Burnet, Chairman (1966).
Maynard Hart, El Paso (1965).
Dean Dimmitt, Uvalde (1965).
Dan Spivey, Lufkin (1964).
Hugh F. Rives, Jacksonville (1964).
R. E. Speegle, Garland (1964).
William F. Ross, San Benito (1966).
Billy Rual Jones, Amarillo (1965).
Sheldon Joseph, Houston (1966).

Committee on Medical Careers
A. Fletcher Clark, Jr., San Antonio, Chairman (1965).
Harry M. Shytles, Sherman (1965).
John Bonnet, Temple (1964).
John B. Truslow, Galveston (1964).
Walter A. Brooks, Quanah (1964).
Gordon Black, El Paso (1966).
M. D. McCauley, Austin (1966).
Peter Kamin, San Antonio (1966).
John Wensley, Longview (1965).

Committee on Liaison with Medical Schools (standing)
Russell L. Deter, El Paso, Chairman (1966).
Frank Harrison, Dallas (1966).
L. Bonham Jones, San Antonio (1966).
Harry Spence, Dallas (1965).
Stanley Olson, Houston (1965).
May Owen, Fort Worth (1964).
Henry Renfer, Jr., Austin (1964).
Stephen R. Lewis, Galveston (1965).
W. H. Hamrick, Houston (1964).

Committee on Nursing Home Accreditation (special)
Charles B. Dryden, Jr., Wichita Falls, Chairman.
Richard L. Berry, Marshall.
Ralph L. Coffelt, Waco.
W. Kenneth Day, Abilene.
Lloyd L. Gregory, Jr., Houston.
Arvil Ponton, Alpine.
William O. Sweet, Brownwood.
Marshall D. Turnbull, Abilene.[9]

9. Appointed September, 1963.

Council on Medical Jurisprudence

Committee on Military and Veterans Affairs (standing)
Milton V. Davis, Dallas, Chairman (1965).
Dickson K. Boyd, Denton, Vice Chairman (1966).
Philip T. Williams, San Antonio (1965).
James C. Whittington, Fort Worth (1964).
Richard H. Harrison, III, Bryan (1964).
Paul R. Meyer, Port Arthur (1964).
James E. Watson, Jr., Houston (1966).
Ivey E. Lamberth, Tyler (1965).
George W. Tate, Longview (1966).[10]

Council on Medical Service and Insurance

Committee on Association Insurance Programs (standing)
Alford R. Hazzard, Austin, Chairman (1965).
G. J. Pruitt, Lufkin (1965).
A. L. Hewitt, Lubbock (1964).
C. C. Shotts, San Antonio (1964).
Donald S. Gibbs, Fort Worth (1966).[11]
Charles W. Klanke, Houston (1966).
Laurance Nickey, El Paso (1966).

Committee on Health Insurance (standing)
A. Rex Kirkley, Temple, Chairman (1965).
William M. Sherrill, Houston (1965).
Jack L. Eidson, Weatherford (1965).
E. A. Maxwell, San Antonio (1964).
Norman West, Hillsboro (1964).
Mario E. Ramirez, Roma (1964).
George L. Walmsley, Baytown (1966).
John E. Douglas, Austin (1966).[12]
Billy B. Markette, San Antonio (1966).

Subcommittees of Committee on Health Insurance:
Appointees to Hospitals-Insurance-Physicians Joint Advisory Committee
C. D. Bussey, Dallas (1964).
Ernest A. Maxwell, San Antonio (1964).[13]
William M. Sherrill, Houston (1964).[14]

Committee on Liaison with Blue Shield (standing)
Everett C. Fox, Dallas, Chairman (1965).
R. W. Kimbro, Cleburne (1965).
Harvey Renger, Hallettsville (1965).
Olin B. Gober, Temple (1964).
R. H. Bell, Palestine (1964).
J. B. Copeland, Austin (1964).
E. A. Rowley, Amarillo (1966).
Denton Kerr, Houston (1966).
James A. Hallmark, Fort Worth (1966).

Committee on Professional Insurance (standing)
D. O. Johnson, Austin, Chairman (1964).
R. E. Read, Lufkin (1965).
A. W. Bronwell, Lubbock (1965).
Bill Robins, Houston (1964).
Patrick H. Pappas, Lubbock (1966).
Edward G. Naslund, Amarillo (1966).

10. Appointed October, 1963, to replace Prescott Haralson, Amarillo, who resigned October, 1963.
11. Appointed January, 1964, to replace John Douglas, Austin, who resigned to accept appointment to Committee on Health Insurance.
12. Appointed October, 1963, to replace Marvin Schlecte, Plainview, who resigned September, 1963.
13. Appointed in October, 1963, to replace A. Rex Kirkley, Temple, who resigned October, 1963.
14. Appointed in October, 1963, to fill vacancy.

E. A. Cox, Wichita Falls (1966).
Porter Mason, Dallas (1965).[15]
William F. Pipes, San Antonio (1964).[16]

Committee on Workmen's Compensation Insurance (standing)
Edward T. Smith, Houston, Chairman (1965).
J. B. Chester, Dallas (1965).
Richard C. Bellamy, Liberty (1964).
F. C. Rehfeldt, Fort Worth (1964).
Ralph E. Donnell, Jr., Abilene (1966).
James L. Mims, Jr., San Antonio (1966).
Edward F. Thomas, Amarillo (1966).

Council on Public Relations and Public Service

Committee on Aging (standing)
F. G. Dorsey, Houston, Chairman (1964).
C. J. Ruilmann, Austin (1965).
John Dupre, Levelland (1965).
Edward J. Lefeber, Galveston (1965).
Elizabeth Gentry, Austin (1964).
Vance Terrell, Stephenville (1964).
Ernest W. Keil, Austin (1966).
Roy H. Morris, Elgin (1966).
Kenneth R. Flamm, Amarillo (1966).

Committee on Disaster Medical Care (standing)
Ben A. Merrick, Dallas, Chairman (1964).
Joe R. Shaeffer, San Antonio (1965).
T. June Melton, Midland (1965).
W. D. Nicholson, Freeport (1965).
J. L. Johnson, Amarillo (1966).
John C. Allensworth, Mineral Wells (1966).
O. A. Fly, Jr., Houston (1964).
Robert A. Dennison, Jr., Austin (1964).
Joseph Selman, Tyler (1966).

Committee on Industrial Health (standing)
Robert J. Potts, Dallas, Chairman (1966).
Robert A. Wise, Houston (1965).
G. V. Launey, Dallas (1965).
John C. Parsons, San Antonio (1965).
Vacancy (1964).
Jesse G. Burdick, Pasadena (1964).
Kurt Lekisch, Midland (1964).
F. W. Wilson, Port Arthur (1966).
E. E. Baden, Raymondville (1966).

Committee on Medicine and Religion (special)
Edwin Goodall, Breckenridge, Chairman.
H. Frank Carman, Dallas.
Frank Cohen, Fort Worth.
John P. McGraw, Houston.
Howard O. Smith, Marlin.
Robert F. Boverie, El Paso.[17]
M. O. Rouse, Dallas, ex officio.
Travis Smith, Abilene, ex officio.

Committee on Public Health (standing)
Sam A. Nixon, Floresville, Chairman (1966).
Vacancy (1965).[18]
Lorence W. Feller, Fredericksburg (1965).
Charles A. Pigford, Houston (1965).
Ben Primer, Sr., Austin (1964).
W. V. Bradshaw, Jr., Fort Worth (1966).
J. E. Peavy, Austin (1966).
George L. LeBeau, Jr., Wichita Falls (1964).
William H. Wade, El Paso (1964).

15. Appointed September, 1963, to fill vacancy.
16. Appointed November, 1963, to fill vacancy.
17. Appointed January, 1964, to fill vacancy.
18. Vacancy created by death of J. C. Ellington, San Antonio, on Oct. 8, 1963.

Committee on Rural Health (standing)
Curtis R. Haley, San Augustine (1966).
J. G. Sanders, Bremond (1965).
Charles L. Bishop, Ganado (1965).
Sam R. Barnes, Trinity (1965).
Lloyd Sherrill, Ozona (1964).
George V. Pazdral, Somerville (1964).
T. T. Walton, Jr., Bryan (1964).
Dennis W. Short, Commerce (1966).
Erwin G. Pink, Frisco (1966).

Committee on School Health (standing)
Edwin L. Rippy, Dallas, Chairman (1964).
Vacancy (1965).[19]
R. K. Arnett, Lufkin (1965).
H. C. Chancellor, Honey Grove (1964).
M. A. Childers, San Antonio (1964).
A. G. Arrant, Abilene (1965).
John W. Stack, Mesquite (1966).
E. E. Addy, Jr., Cisco (1966).
R. M. Hampton, Pampa (1966).

Committee on Transportation Safety (standing)
Heinrich Lamm, Harlingen, Chairman (1965).
Otto Lippmann, Austin (1965).
William H. Neil, Fort Worth (1965).
D. R. Knapp, Kerrville (1964).
Linwood H. Denman, Lufkin (1964).
Boyd D. Alexander, Waco (1964).
Mario Palafox, El Paso (1966).
Charles H. Wilson, Wichita Falls (1966).
Vernon V. Goss, Tyler (1966).

Council on Scientific Advancement

Committee on Athletics (standing)
Robert G. McCorkle, Houston, Chairman (1966).
Harry W. Slade, Waco (1966).
Charles Sadler, Amarillo (1964).
J. Frank Beall, Nacogdoches (1965).
Louis Levy, Fort Worth (1966).
Alfred E. Voigt, El Paso (1965).
Ray Lawson, Dallas (1965).
William Horn, Fort Worth (1964).
Dan Spivey, Lufkin (1964).

Committee on Cancer (standing)
Howard R. Dudgeon, Waco, Chairman (1965).
Tom B. Bond, Fort Worth (1965).
John F. Thomas, Austin (1965).
R. Lee Clark, Jr., Houston (1964).
John R. Rainey, Austin (1964).
William S. Brumage, Austin (1964).
W. Q. Budd, Amarillo (1966).
Jabez Galt, Dallas (1966).
Gerald Jordan, El Paso (1966).

Committee on Cardiovascular Diseases (standing)
Robert E. Leslie, El Campo, Chairman (1965).
David Miesch, Paris (1965).
S. W. Thorn, Houston (1965).
W. Frank McKinley, Marlin (1964).
William B. Adamson, Abilene (1966).
John R. Derrick, Galveston (1966).
K. C. Sherman, Harlingen (1964).
John M. Verosky, El Paso (1966).
James E. Kreisle, Austin (1964).

Committee on Maternal Mortality (standing)
William J. McGanity, Galveston, Chairman (1966).

19. Vacancy created by resignation of Zenas W. Hutcheson, Jr., Andrews, October, 1963.

J. C. Rucker, Jacksonville (1965).
Donald M. Gready, Houston (1965).
Fred W. Lurting, Big Spring (1965).
Stephen W. Cobb, Dallas (1965).
R. P. McDonald, Fort Worth (1964).
W. H. Jondahl, Harlingen (1964).
R. E. Moon, San Angelo (1964).
Henry C. McGrede, Jr., Longview (1964).
Warren T. Kable, Jr., Wichita Falls (1964).
J. S. Lancaster, Austin (1966).
William E. Strozier, San Antonio (1966).
E. K. Blewett, Austin (1966).
James R. Morgan, El Paso (1964).
Matthew H. Talty, Jr., Houston (1965).
Peggy Joyce Whalley, Dallas, ex officio.
Carl F. Moore, Jr., Austin, ex officio.

Committee on Mental Health (standing)
P. C. Talkington, Dallas, Chairman (1965).
Ivan Bruce, Galveston (1965).
George Constant, Victoria (1965).
A. D. Pattillo, Austin (1964).
Dorothy Wyvell, Midland (1964).
P. C. Palasota, Abilene (1964).
Leroy A. Boriack, Corpus Christi (1966).
Holland C. Mitchell, Waco (1966).
Charles H. Brown, Wichita Falls (1966).

Committee on Nuclear Medicine (standing)
Herbert C. Allen, Jr., Houston, Chairman (1965).
E. E. Anthony, Jr., Fort Worth (1965).
C. C. Shullenberger, Houston (1965).
J. Allen Chamberlin, Houston (1964).
Lloyd R. Hershberger, San Angelo (1964).
Elbert DeCoursey, San Antonio (1966).
J. R. Maxfield, Jr., Dallas (1966).

Committee on Nutrition (standing)
Ralph G. Greenlee, Midland, Chairman (1965).
William Levin, Galveston (1965).
D. J. Sibley, Austin (1965).
Joe D. Nichols, Atlanta (1964).
Robert S. Nelson, Houston (1964).
Andres Goth, Dallas (1964).
Per H. Langsjoen, Temple (1966).
William J. Block, San Antonio (1966).
Charles Robinson, Fort Worth (1966).

Committee on Rehabilitation (standing)
Oscar Selke, Houston, Chairman (1966).
Theodore A. Klecka, San Antonio (1965).
Ridings E. Lee, Dallas (1965).
Edward Krusen, Jr., Dallas (1965).
Ira Budwig, El Paso (1964).
Joe R. Gandy, Houston (1964).
Alfred H. Hill, San Antonio (1964).
D. J. Turell, Houston (1966).
Robert A. Murray, Temple (1966).

Committee on Tuberculosis (standing)
John W. Middleton, Galveston, Chairman (1966).
John S. Chapman, Dallas (1965).
R. B. Morrison, Austin (1965).
James L. Reagan, Beeville (1965).
Samuel Topperman, Tyler (1964).
James E. Dailey, Houston (1964).
Richard E. Allison, Sanatorium (1964).
David McCullough, San Antonio (1966).
Daniel E. Jenkins, Houston (1966).

Scientific Section Officers

Digestive Diseases
John R. Kelsey, Jr., Houston, Chairman.
Marcel Patterson, Galveston, Secretary.

General Practice
Harry A. Tubbs, Post, Chairman.
George A. Burgess, Eagle Pass, Secretary.

Internal Medicine
Raymond D. Pruitt, Houston, Chairman.
Horace Cromer, Austin, Secretary.

Obstetrics and Gynecology
Stephen W. Cobb, Dallas, Chairman.
Wayne F. Baden, Temple, Secretary.

Ophthalmology
Louis Daily, Jr., Houston, Chairman.
E. R. Veirs, Temple, Secretary.

Otolaryngology
Daniel B. Powell, Jr., Austin, Chairman.
Ben T. Withers, Houston, Secretary.

Pathology
Alice Smith, Dallas, Chairman.
John L. Wallace, Jr., Fort Worth, Secretary.

Pediatrics
C. William Daeschner, Jr., Galveston, Chairman.
David M. Mumford, Houston, Secretary.

Public Health
Albert G. Randall, Amarillo, Chairman.
William R. Ross, Galveston, Secretary.

Radiology
Frederick J. Bonte, Dallas, Chairman.
George F. Crawford, Beaumont, Secretary.

Surgery
William D. Seybold, Houston, Chairman.
R. H. Baskin, Waco, Secretary.

Book Notes

Books reviewed in this column are available from TMA Memorial Library.

The Hirsute Female. Robert B. Greenblatt, M.D. 313 pp. Springfield, Ill., Charles C Thomas, 1963. $12.50.

THIS BOOK contains papers presented in a symposium at the University of Georgia on an unspecified date; references as late as November, 1962, are included. Although fifteen participants are listed in this symposium, the book bears strongly the imprint of its editor. The book is profusely illustrated and tabular data is well presented. It is well printed and virtually free of typographical errors. It can be read with pleasure and profit in the space of a long evening. There is inevitably more discussion of virilism than of hirsutism *per se*, and all the known adrenal, gonadal, and genetic disorders are discussed (although virilism of the female infant after treatment of the mother during pregnancy with oral progestins is inadequately described). The Achard-Thiers syndome (diabetes of bearded women) is dignified by a chapter, but the Morgagni-Stewart-Morel syndrome is not mentioned. The final chapter which outlines diagnosis, signally fails to define criteria by which to separate the patient who deserves an extended, expensive, and uncomfortable investigation from the larger number of hirsute women who do not require it. Since electrolysis seems at least tacitly accepted by several authors, it would have been helpful to include a chapter describing this procedure and its practitioners, what may be expected from it, and what are its hazards. Like most symposiums, this one is uneven. The high points for this reviewer were the essentially anthropologic measurements of hirsutism and hair growth by Hamilton and Terada, and the development by Greenblatt and his associates of their concepts of the Stein-Leventhal syndrome as an ovarian virilizing syndrome, presumably due to enzymatic dyshormonogenesis.

—John E. Johnson, Jr., M.D., Cincinnati.

Open-Heart Surgery for Mitral Stenosis. H. T. Nichols, M.D., D. P. Morse, M.D., G. Blanco, M.D., and A. Adam, M.D. 66 pp. Springfield, Ill., Charles C Thomas, 1963. $6.

THIS MONOGRAPH consisting of 59 pages and 17 references describes in some detail the authors' technique of the left thoracic approach to open heart surgery for mitral stenosis. The authors have used the open technique in 230 consecutive operations.

Although the use of the open technique for the treatment of mitral insufficiency has been resolved, since it is apparent that closed methods are inadequate in relieving the regurgitation at the mitral orifice and it is generally agreed that the open technique is better for pure mitral stenosis in patients for whom reoperation is necessary, there appears to be some question of the type of operation preferable for a pure mitral stenosis that has not been operated upon. The authors feel that open operation is preferable in almost all cases of mitral stenosis because it allows a full opening of both commissures of the valve, removal of calcium in the valve or clot from the atrial wall, a complete mobilization of the subvalvular structures, the correction of any insufficiency that may be present, and finally allows replacement of the valve when a totally destroyed calcific valve is encountered.

In the last 100 consecutive open primary operations for mitral stenosis in the authors' clinic there have been only two deaths. There were 13 deaths in the series of 200 consecutive open operations for mitral stenosis for an overall mortality rate of 6.5 per cent. Four of these deaths occurred in the 39 patients undergoing reoperations and represent a death rate of 10.3 per cent in this group. In contrast, there were nine deaths in 161 patients operated on for the first time, for a mortality rate of 5.6 per cent.

The disadvantages of the mechanical dilator with the "closed" technique are pointed out by the authors, particularly in the presence of a heavily calcified valve, or a valve with significant pre-existing insufficiency.

Fifty-six pages are utilized to describe the technique of the left-sided thoracic approach and for those who utilize this approach; much of the information will prove interesting. It needs to be pointed out that there are many surgeons who prefer the right-sided approach for the treatment of mitral valvular disease and it must be pointed out that it is not always possible to be certain that the case involved is one of pure mitral stenosis or mitral valvular disease and that frequently patients will have concomitant aortic valvular disease not suspected by preoperative examination and determination. In such an event, the left-sided approach is extremely restricting.

—LeRoy J. Kleinsasser, M.D., Dallas.

Psychological Techniques in Neurological Diagnosis. Bessie B. Burgemeister, Ph.D. 248 pp. New York, Hoeber Medical Division-Harper & Row, 1962. $7.50.

MANY EMOTIONAL problems may be basically neurological dysfunctions. Psychologists and neurologists both disregard, at times, the uses of psychological tools for diagnosis. This book consolidates the information on neuropsychological tests which are available.

It is recommended that psychologists develop acumen in clinical observation.

Evidences of central nervous system disorders can be listed as follows:

1. Behavioral manifestations without confirmation on testing.
2. Test evidences of behavioral defects, without overt behavior changes.
3. Pathologic evidence on both tests and actions of patients.
4. Pathology not evident on testing or in actions of patients.

Behavioral signs of brain disease may be grouped into production or reductions of perception, attention, consciousness, orientation, memory, judgment, affect, and short and long term behavior. One should remember that psychotic and neurotic symptoms may be a precursor of organic brain disease.

The tools of the psychologist are the tests based on controlled stimuli. Observation is not. The possibilities for reaction are more limited in the test situation and tests estimate discrepancies between the functional and optimal levels in any patient. These should approximate each other in normals. Immediate memory, perception, visual-motor, performance, organizational ability, abstract thinking, and judgment are often upset in brain disease, so these functions should be tested.

The types of tests are reviewed. Deserving mention are intellectual tests which include the Babcock-Levy Mental Efficiency Test, the well known Wechsler-Bellevue Scale, the Shipley-Hartford Scale, and the Hunt-Minnesota Test.

Projective techniques include the Rorschach, Goodenough Draw-A-Man Test, and the Bender Visual-Motor Gestalt Test.

It has been found that the Wechsler-Bellevue test will demonstrate preservation of good performance on information, vocabulary, picture completion, and object assembly in organic brain disease yet digit span, arithmetic, block design, and digit symbol functions are often disturbed. Other signs of organic brain disease are fluctuating efficiency or failing easy problems, whereas complex problems are handled well.

Another simple test that can be done is to test the digit retention span forward and reverse. If the patient does better in reverse, emotional factors are suspected. The Halstead test, in the absence of neurosis or psychosis, is a good localizing sign of frontal lobe disease of the brain. The Goldstein-Scheerer test also measures moderate to severe frontal lobe losses. In matching tests, such as blocks, the behavior must be described. Errors in interpreting these tests arise from considering only the signs instead of the overall Gestalten and personality in general.

In regard to epilepsy, the quest for an epileptic personality has failed. There is no good evidence of mental deterioration in an epileptic. Tests do help evaluate the emotional problems arising as a reaction to the diagnosis of epilepsy, the emotional problems which might precipitate seizures, and the emotional problems contributing to the patterns of seizures.

The author suggests that the clinical psychologist give more observation to behavior in his report, that he be flexible in his choice of test battery, that he use tact and reassurance to get the maximum performance, and that he observe the reaction of the patient.

No one test in itself is adequate to detect organic mental impairment. In mental retardation long range predictions after injury cannot be made. The report should be brief, to the point, and should give the reasons why the psychologist forms his opinions of the patient. The book gathers together much information and many references regarding this interesting field of medicine.

—Marshall G. Henry, M.D., Houston.

Internal Medicine in World War II: "Infectious Diseases." Vol. 2. Ed., Col. John Boyd Coates, Jr., MC, USA. 649 pp. Washington, D. C., 1963. Office of the Surgeon General, Department of the Army, 1963. $6.75.

THIS VALUABLE ADDITION to the medical series in its organization deals with the diagnosis, etiology, pathology, and treatment of diseases encountered. It deals with the effects of climate, methods of control of vectors of diseases, laboratory aids, and meth-

ods of practical management.

One gains an appreciation of the magnitude of the effort when it is realized that over 15,000 drugs were tested for antimalarial control and suppression. Atabrine, which proved to be so useful, was among them.

Epidemic typhus was rampant in the Pacific native populations, but it caused not a single death in our troops due to the skillful management by our medical corps in cooperation with other services. For the first time in World War II there were fewer deaths from disease than there were from injury.

The global scope of our activities brought us into contact with such desert diseases as sandfly fever in North Africa; dengue in the tropics whose control lay in attacking its vector, the mosquito; Q fever due to *Rickettsia burneti* whose control lay in the destruction of ticks that used cattle as the reservoir of the disease. Aureomycin, among other drugs, was found to be effective in its treatment. Typhus with its vector, *Pediculis corporis*, transmitting *R. prowazekii* was found to be widespread in the Pacific islands, China, Burma, India, and North Africa. These are but a few of the many infectious diseases presented with tested methods for their control and eradication.

Other sections deal with diphtheria and its control both in temperate and tropical climates. Emphasis is given to additional clinical aspects to be reckoned with under tropical conditions. Conditions more familiar such as rheumatic fever and tuberculosis receive their proper treatment in accordance with their importance and incidence. The best accepted methods for control of venereal diseases are given. Last, a thorough discussion of the malaria problem is presented. For those with an active interest in the control of infectious diseases, this volume is enjoyable and informative.

—Joe C. Rude, M.D., Austin.

Personnel in World War II. Col. John Boyd Coates, Jr., M.C., USA, and Charles M. Wiltse, Ph.D., Eds. 548 pp. Washington, D. C., Office of the Surgeon General. 1963. $6.

THE PERSONNEL strength of the medical department in World War II was 700,000, some three times as many people as were in the regular Army in 1939. Time did not permit training through officer candidate schools so qualified personnel had to be obtained directly from civilian life.

This book points up the importance for regular officers and men to be ready for enormous, instant expansion with the proper delegation of responsibility and freedom of action to carry out assigned functions. Those from civilian life likewise should adapt themselves to their duties and responsibilities with an eye to doing the things at hand.

It is predicted that as a result of this large effort there will come a better understanding between officers and men in the regular establishment, the reserve and civilians generally, and that there will be better utilization of personnel and a more equitable distribution of effective talent.

—Joe C. Rude, M.D., Austin.

New Books in TMA Library

Best, C. H.: The Living Body, 4th ed., New York, Holt, Rinehart and Winston, 1958.

Bredow, M.: The Medical Assistant, 2nd ed., New York, Blakiston Division, McGraw-Hill Book Co., 1964.

The Dallas Morning News: Texas Almanac 1964-65, Dallas, A. H. Belo Corp., 1963.

Gellis, S. S., and Kazen, B. M.: Current Pediatric Therapy, Philadelphia, W. B. Saunders Co., 1964.

Lowry, O. H., et al.: Harvey Lectures, Series 58, New York, Academic Press, 1963.

New York Heart Association, Inc.: Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels, 6th ed., Boston, Little, Brown & Co., 1964.

Modell, W.: Drugs of Choice, 1964-1965, Saint Louis, C. V. Mosby Co., 1964.

Shilling, C. W.: Atomic Energy Encyclopedia in the Life Sciences, Philadelphia, W. B. Saunders Co., 1964.

Slobody, L. B., and Wasserman, E.: Survey of Clinical Pediatrics, 4th ed., New York, McGraw-Hill Book Co., Inc., 1963.

Sokolov, Y. N.: Perception and the Conditioned Reflex., London, Pergamon Press, 1963.

Yahr, M. D., ed.: Transactions of the American Neurological Association, New York, Springer Publishing Co., 1963. Vol. 88.

Gudger, J. R., ed.: Transactions of the Association of Life Insurance Medical Directors of America, New York, Recordings and Statistical Corporation, vol. 38, 1954; vol. 40, 1956; vol. 43, 1959; vol. 44, 1960; vol. 46, 1962.

Pack, G. T., and Ariel, I. M., Eds.: Treatment of Cancer and Allied Diseases, 2nd ed., New York, Paul B. Hoeber, Inc., 1962, vols. 6-8.

Holt, J. F., and Whitehouse, W. M., Eds.: Year Book of Radiology, Chicago, Year Book Medical Publishers, 1964.

Recent Deaths

DR. O. N. MAYO

DR. O. N. MAYO

Dr. Oscar Newton Mayo, a Brownwood radiologist, died Dec. 26 in a Brownwood hospital. He was 79.

He had belonged to Texas Medical Association since 1908 and had been an honorary member since 1962. He was a former Councilor from the 4th District and was a past president and secretary of Brown-Comanche-Mills-San Saba Counties Medical Society. He was a member of the American Medical Association.

Dr. Mayo was born Oct. 22, 1884, in Caldwell County, the son of James A. and Julia Jefferson Camp Mayo. He attended school in Lockhart and at Bastrop Normal College. He went to Tennessee for his medical education at the University of the South in Sewanee where he received his degree in 1906.

A number of years of his life were spent in the Army medical corps. He served as a first lieutenant during World War I and because of World War II, he returned to the service for the period 1941-1946, during which time he rose from major to lieutenant colonel.

His medical career included many years spent first in general practice and for the past 29 years in radiology. He had practiced in Elgin, Belton, and Brownwood. In 1946 he was certified by the American Board of Radiology.

He owned and operated a dairy and was one time vice president of a bank.

He married Susie D. Anderson of Milford, Tex., on Dec. 23, 1906. The ceremony took place in Italy, Tex. Mrs. Mayo died on Nov. 16, 1950. On July 5, 1951, in Los Angeles, Dr. Mayo was remarried to Ann Langsford, the widow of CDR. R. B. Langsford of Waxahachie.

Dr. Mayo was a member of the First Christian Church of Brownwood where he was an elder for many years. He belonged to the Lions Club, was a Mason, and was a member of Suez Temple Shrine in San Angelo. Other organizations to which he belonged include the Knights of Pythias and the Woodmen of the World.

Survivors include his wife; three daughters, Mrs. E. B. Grady of Borger, Mrs. George R. Roland, and Mrs. J. E. Bohannon of Dallas; one sister, Mrs. Smith Robbins of Mathis; and eight grandchildren.

DR. E. N. SCHAFFER

Dr. Elliott Nathan Schaffer of Houston died Jan. 4 of ventricular arrhythmia associated with arteriosclerotic heart disease. He had been in the hospital since Aug. 22, 1963, under treatment for multiform glioblastoma.

DR. E. N. SCHAFFER

His heart condition had made it necessary for him to limit his work to halftime office practice for the past 12 years.

A native of New York, he was brought to Houston when he was a small child. He attended Houston public schools, Rice Institute, and received a bachelor of science degree from New York City College. His medical degree was earned at the University of Texas Medical Branch. He interned at Kings County Hospital in Brooklyn, N. Y.

During World War II he served in the Army Air Corps.

Dr. Schaffer was a member of Congregation Beth Yeshurun, Holland Masonic Lodge No. 1, AF&AM, and Arabia Temple Shrine and Consistory.

He belonged to the American Medical Association, Texas Medical Association, and Harris County Medical Society.

He is survived by his wife, the former Della Shapiro; a son, Martin Schaffer; a sister, Mrs. David Jaffe; and a brother, Dr. Samuel S. Schaffer, all of Houston.

DR. M. P. CHIASSON

Dr. Mary Pauline Chiasson of Beaumont died on Nov. 2 of cancer of the cervix.

She was born Oct. 21, 1911, in Scott, La. Her parents are Dr. John Leon Chiasson of Port Neches and Mrs. Katie Paxton Chiasson of Port Neches. She attended public school in Port Neches and Lamar Technological College in Beaumont. She received her B.A. degree from H. Sophie Newcomb Memorial College for Women in New Orleans and her M.D. degree from Tulane University School of Medicine.

She interned at Edward W. Sparrow Hospital in Lansing, Mich., and served residencies at New Orleans Hospital and Dispensary for Women and Children and St. Therese Hospital in Beaumont. She had done postgraduate work at Charity Hospital in New Orleans.

In 1939 she began a general practice in Beaumont. She left for a year as an anesthesiologist in New Orleans and returned to Beaumont in 1943 to practice that specialty for the rest of her life.

She was a member of the Jefferson County Medical Society, Texas Medical Association, American Medical Association, American Society of Anesthesiologists, and the American Medical Women's Association.

DR. M. P. Chiasson

Dr. Chiasson was a Baptist.

In addition to her parents, she is survived by a sister, Dr. Chaille Chiasson (Mrs. John J. King) of Columbia, Mo.

Coming Meetings

American Medical Association, Annual Meeting, San Francisco, Calif., June 21-25, 1964. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Houston, Annual Session, April 23-26, 1964; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

MARCH

American College of Allergists, Bal Harbour, Fla., March 1-6, 1964. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

South Central Association of Blood Banks, Oklahoma City, March 27-28, 1964. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southwestern Society of Nuclear Medicine, Houston, March 6-8, 1964. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Texas Tuberculosis Association, Austin, March 19-21, 1964. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

United States-Mexico Border Public Health Association, Monterrey, Mexico, March 2-5, 1964. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

Ninth District Society, Huntsville, March 19, 1964. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Dallas Southern Clinical Society Conference, Dallas, March 15-18, 1964. Dr. W. O. Tschumy, 1616 Medical Arts Bldg., Dallas, Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 2-4, 1964. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

APRIL

American Academy of General Practice, Atlantic City, April 11-17, 1964. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 12, Exec. Sec.

American Academy of Pediatrics, Philadelphia, April 20-22, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association for Thoracic Surgery, Montreal, Canada, April 27-29, 1964. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American College of Physicians, Atlantic City, April 6-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American Gastroenterological Association, Dallas, April 24-25, 1964. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C.

American Surgical Association, Hot Springs, Va., April 1-3, 1964. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., Chicago, April 3-4, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Southwest Allergy Forum, Hot Springs, Ark., April 5-7, 1964. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwestern Surgical Congress, San Antonio, April 26-29, 1964. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

Texas Air-Medics Association, Houston, April 25-26, 1964. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association of Public Health Physicians, Houston, April 23, 1964. Dr. W. S. Brumage, 2600 Westover Rd., Austin, Sec.

Texas Chapter, American Academy of Pediatrics, Houston, April 24, 1964. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, Houston, April 26, 1964. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Houston, April 25, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, Houston, April 26, 1964. Dr. John W. Chriss, 2436 Morgan, Corpus Christi, Sec.

Texas Industrial Medical Association, Houston, April 23, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, Houston, April 26, 1964. Dr. E. I. Bruce, Jr., 1014 Strand, Galveston, Sec.

Texas Ophthalmological Association, Houston, April 25-26, 1964. Dr. Harold Hunt, 150 Eighth St., S.E., Paris, Sec.

Texas Orthopaedic Association, Houston April 25-26, 1964. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, Houston, April 24-25, 1964. Dr. J. P. Moran, 3154 Reid Dr., Corpus Christi, Sec.

Texas Physical Medicine and Rehabilitation Society, Houston, April 23-26, 1964. Dr. D. Hoehn, Doctor's Bldg., Fort Worth, Sec.

Texas Proctologic Society, Houston, April 23, 1964. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Society of Anesthesiologists, Houston, April 24-26, 1964. Dr. L. Duflot, 173 San Marino, Galveston, Sec.

Texas Society of Athletic Team Physicians, Houston, April 23, 1964. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section. Houston, April 26, 1964. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Pathologists, Inc., Houston, April 26, 1964. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, Houston, April 23, 1964. Dr. W. C. Sellman, Jr., 610 Medical Arts Bldg., Dallas, Sec.

Texas Surgical Society, Waco, April 5-7, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Houston, April 23, 1964. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

National and Regional

American Academy of Allergy. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Association of Plastic Surgeons, Chicago, May 13-16, 1964. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N.Y., Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Chest Physicians, San Francisco, Calif., June 18-22, 1964. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Obstetricians and Gynecologists, Miami Beach, Fla., May 17-22, 1964. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Radiology, Minneapolis, Minn., Oct. 2-3, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Hawaii, June 27-July 2, 1964. Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Sec.

American Gynecological Society, Hot Springs, Va., May 25-27, 1964. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Medical Women's Association, Roanoke, Va., May 31-June 2, 1964. Dr. Jessie Brodie, 1790 Broadway, New York 19, Exec. Sec.

American Neurological Association, Atlantic City, June 15-17, 1964. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Philadelphia, May 10-14, 1964. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

International College of Surgeons, Vienna, Austria, May 11-16, 1964. Dr. W. F. James, 1615 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico. Dr. C. Kalil, 200 East Monterey Way, Phoenix, Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Regional Cancer Conference. Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., October, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

State

South Texas District, American College of Surgeons, Corpus Christi, May 15-16, 1964. Dr. Sanford Glanz, 2425 Medical Center, Corpus Christi, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists. Dr. H. W. Savage, 815 Fifth Ave., Fort Worth, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Public Health Association. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas State Board of Examiners in Basic Sciences, April, 1964. Mrs. Maria P. Hathaway, 1012 State Office Bldg., Austin, Exec Sec.

Texas State Board of Medical Examiners. Fort Worth, June 22-24, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Urological Society, Fort Worth, February, 1965. Dr. B. C. Bates, 1108 Nueces, Austin, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea. Scott and White Clinic, Temple, Sec.

Thirteenth District Society. Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 412 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals. Mr. R. L. Heath, Malone-Hogan Clinic, Big Spring, Sec.

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A Look at Research In Medical Schools

MUCH HAS BEEN written recently, and a lot more has been said, about the emphasis on research in medical schools today.

How great is this emphasis really, and to what purpose? What are the activities in a medical school which are considered in the category of research?

These questions are very important, but their simplicity is deceptive; for they are asked in the atmosphere of an increasing persuasion in the medical profession that modern medical education is emphasizing "the theoretical" at the expense of "the practical," and "the science" without reference to "the art" of medicine—as though these were titratable and distinguishable as black and white or evil and virtue. And now the alleged subsidization of medical education in recent years by the National Institutes of Health seems to be the clincher.

Let us focus the microscope on the field in question before we start dissecting, or reporting, or publishing! What were those questions again?

What are the activities in medical school which are considered in the category of research, and how great an emphasis is put on them?

Research in its broadest sense is a habit of inquiry, characterized by intelligent skepticism and impatience with the realization that the body of medical knowledge which has multiplied astronomically in the past few decades is still characterized far more by assumptions, theories, hypotheses, and pragmatisms than by scientific facts.

This habit of mind is constantly emphasized in the selection of faculty, in the teaching of students, and in the care of patients in a university medical center today. Such emphasis can never become overemphasis, because the habit of inquiry is as intrinsic as human sympathy to the vitality of the art and the science of modern medicine.

Research in its more precise definition is merely one manifestation of this habit of mind. In any worthy body of faculty and students there are exciting members who have a drive to pursue inquiry *personally* plus the knack and talent to learn how to

do it, and then—to do it. Emphasis upon fostering research efforts in this definition varies somewhat from school to school, and within every school, from department to department; but attempted overemphasis, no matter how seductive in terms of departmental glamour or N.I.H. support, runs into a stone wall. For, while the capacity for achieving the habit of inquiry is almost universal in a population as carefully selected as medical school faculty and students, only the relatively rare individual has the capacity for (or can be coaxed into) a full career devoted to creative research.

Accordingly, our concern in the medical profession today should be directed toward encouragement of the habit of inquiry in the *practice* of medicine. Using the critical disciplines of science, practitioners thus may really learn more with experience and fortify their service to patients.

The fountainhead of knowledge, which is creative research in a medical center, must not be allowed to dribble or dry up for want of professional thirst. No student-physician in this day of medical miracles *can* be overexposed to the atmosphere of inquiry. The Art of Medicine has no shape and substance without the Science of Medicine, and the Science of Medicine requires the Art of Medicine for full diagnosis of human need. Neither can truly flourish without the other.

The facts of life are simply that as time goes on and medical knowledge increases, the translation of this knowledge to the benefit of patients must depend upon our capacity for critical analysis and judgment. One wonders whether in all probability the practicing physician is not already doing a better job in this area than his sense of distance from university campuses can allow him to admit!*

—JOHN B. TRUSLOW, M.D., Galveston,
Executive Dean and Director,
University of Texas Medical Branch.

A Moment in the Patient's Shoes

THE PATIENT-DOCTOR relationship begins as an exquisitely fragile thing, requiring great care by both parties to be brought to full maturity. Because of the ethics of our profession, this important interpersonal relationship is always born in the mind of the patient. Its growth and development, however, are uniquely dependent on the physician.

Why does the patient seek the help and understanding of the physician? What changes a person to a patient?

In the simplest terms, the birth of the patient-physician relationship has its conception at the moment a personality decompensates. This decompensation results from the occurrence of incidents, usually uncomfortable, in the person's physical, mental, or emotional environment. Thus the patient feels he can no longer "go it alone." This feeling is fundamental and must be consciously recognized by the physician.

Responsive emotional values from the physician are the immediate essential ingredients in nurturing this relationship. If this response to the patient's needs is accepted as a necessity before the first meeting, the physician can enter into the meeting mentally and emotionally prepared to give a little of himself in understanding and interest in the patient's problem from the patient's point of view.

The biggest handicap the physician must overcome to respond to the patient's needs is the physician's natural tendency to evaluate the seriousness of the patient's complaint by his own standards rather than by the patient's.

By the patient's standards some problems are a life threat but by the physician's standards are foolish. A person did not become a patient, however, by the physician's standards but by his own. Therefore, the "life threat" is a real problem.

All person-to-patient transformations are made by patient standards and all are equally important to the patient. If the physician can accept these premises, which are really ego-building for him, he will discover that all patients have a real and urgent need for his knowledge and understanding.

—SILAS W. GRANT, M.D., Hillsboro.

*The thesis of this editorial will be demonstrated in Galveston on May 8, when the Fifth Annual Student Research Forum will be held at the Medical Branch. Inspired, organized, and promoted by students themselves, the Forum will include more than 60 student papers, including over 30 by students from out-of-state medical schools. Members of TMA are invited to attend; they will discover that these 15-minute papers demonstrate a measure of depth and comprehension their elders would do well to emulate.

Minister Needed for Total Care of Man

THE MEDICAL PROFESSION in this country is making steady progress in establishing a closer relationship between minister and physician in the total care of man. This program was initiated by the American Medical Association and is progressing at the state and county medical society levels. More than a few county societies in Texas have functioning committees on medicine and religion and have held meetings between doctors and ministers which have been accepted with tremendous enthusiasm.

The patient stands to gain the most from this program. Ordinarily, the minister can obtain from the physician a few facts about the patient's illness which help to make his calls more timely and more beneficial. In turn, the physician can lean on this better informed minister and suggest that he be called to administer spiritual guidance (therapy, if you please) when sedatives and tranquilizers are no longer of real value. Certainly it is not necessary for the physician to become an evangelist. There is evidence however, that a potentially large army in the clergy awaits a chance to be shown how to be more helpful in caring for the patient and for the family of the patient when needed.

Often the physician turns from the bedside of a critically ill or injured patient and finds the clergyman present. Perhaps at times he may think, "What is he doing here? Probably expects me to give him a firsthand report on the case right now when I don't have any time to spare." Of course an instant of reflection dissolves such thoughts; the minister, like the doctor, has been called because he is needed and wanted. He is there to help if he can, and it would seem logical to give him a chance to help in such situations. He is a student by nature of his profession, and is willing to learn more about sick people if we will but take a little time to teach him.

No formal period of instruction is necessary or even intimated, but physician-sponsored meetings with the ministers have established a congenial atmosphere for stimulating discussions. Some county societies have held as many as five programs with increasing enthusiasm each time.

Such meetings will help us understand that a degree of rapport with the clergy helps to assure better care and treatment for the individual patient, and encourages more consideration for the needs of patients' families.

—EDWIN GOODALL, M.D., Breckenridge,
Chairman, Committee on
Medicine and Religion.

AID TO "DEVELOPING" COUNTRIES

Health Is Most Firmly Grasped Blessing

MANY MEDICAL MISSIONS connected with church groups, foundations, pharmaceutical companies, or government (such as the Peace Corps) are currently active in the field of medical care and education throughout the world.

The American Medical Association has recognized the importance of strengthening the international health effort and has organized a Department of International Health. Through this department, I was put in touch with the Christian Medical Council for Overseas Work of the Lutheran Church—Missouri Synod, and was accepted for a one-year program of voluntary medical assignment at the Bethesda Lutheran Hospital in Ambur, North Arcot District, Madras State, South India.

The service of American doctors offers a proved, effective way by which the sick, blind, and crippled of the world may understand, in a personal way, the value placed on human dignity by Western medicine and Western physicians. Schweitzer, Seagraves, and Dooley have become symbolic names. Their efforts present a favorable and lasting image in the underdeveloped countries —however, the term "developing" countries should be used. It seems likely that, as time passes, history will record the physician-to-physician, physician-to-people, and people-to-people approaches as among the most ef-

fective adjuncts to the American foreign aid program.

Diseases that have yielded to modern medical science, such as smallpox, tuberculosis, cholera, and malaria, still cause millions of preventable deaths annually in these developing countries. My work was mainly with the tuberculosis control program. It has been estimated that 2 per cent of the entire population of India is afflicted with active pulmonary tuberculosis. Seven million Indians require chemotherapy. Only 30,000 beds are available in India for this malady; therefore, domiciliary treatment for tuberculosis has been evaluated intensively at the Tuberculosis Chemotherapy Center in Madras.

Public statistics in India state that in 1953, 75,000,000 people developed malarial fever annually. This figure has dropped to less than 100,000 under the impact of the well-organized nationwide campaign financially sponsored by the Rockefeller Foundation and put into action by the World Health Organization. In 1962 in India, 4,000 medical students received degrees. This is in contrast to 1947 when there were only 16 medical schools, providing a few hundred graduates yearly.

The Ford Foundation operating in India is deeply interested in the massive expanding population amounting to 10,000,000 people each year. Family planning and planned parenthood clinics have been developed in large cities, but the result of this undertaking is sporadic. Mr. J. Kenneth Galbraith, the previous United States Ambassador to India, stated on June 28, 1963, in Bombay, "The thing which I find more alarming than the prospect of security along the northern frontier of India is the problem of population explosion, when one considers that some 9,000,000 to 10,000,000 people are added each year. This is a measure of the handicap which Indian planning suffers under, or one might say, staggers under."

Agricultural output of the land currently under cultivation is not adequate to feed 480,000,000 people; actually the utilization of the land, because of outdated methods, is about one-third that of Japan. American-sponsored agricultural projects and equipment are offered and accepted. However, the long-range effectiveness of this aid is somewhat uncertain, largely because of discrepancies between American ways and Asian ways of doing things, and because neither side can easily understand and communicate with the other. The construction of dams has produced 20,000,000 additional acres of land for cultivation during the last ten years, but this still is not adequate. On the average, one shipload of American farm products has left United States ports for India every day during the past ten years.

The Indian people are tolerant but deeply committed to traditions and customs, and have retained a basic respect for religion which communism aims to destroy. The Indians dislike regimentation; there is freedom of the press in India. Indian leaders realize that communism will bring regimentation, and it appears likely that India, with the assistance of the Western powers, will oppose communism. This, indeed, holds out hope for the future. Gandhi said, shortly before his death, "If India fails, Asia dies."

Asia today is a scene of intense and rapid change. People who have now learned of modern antibiotics are not willing to go back to herb medicine. The mountain villager who showed me his new kerosene lantern will never go back to using pine torches. The man or woman who once attended a modern school will not be the same again, nor will be his village.

In conclusion, of all the varied and somewhat doubtful blessings the West can confer, better health is perhaps the most easily understood, the most immediately wanted, and most firmly grasped.

Whatever work you do, do it as perfectly as you can. That, I believe, is the best service to the Divine in man.

—KURT LEKISCH, M.D., Midland.

personal

Opinions and Views

NARCOTICS ADDICTION And the "American System"

THE WHITE HOUSE CONFERENCE on Narcotics and Drug Abuse in September, 1962, managed—if nothing else—to draw attention to the magnitude of the problem of narcotics addiction in the United States. At the Conference, the battle lines between the experts who favor the present punitive approach and treatment of addicts by the federal government, and the experts who prefer more leniency and local (ambulatory) medical treatment, were redefined and recorded for posterity.[2] There was no meeting of the minds since the panel on "Law Enforcement and Controls" and the panel on "Current and Experimental Methods of Treatment" were held at the same time in different rooms. These panels epitomized the two different approaches, and in spirit were worlds apart.

That almost uniform concern about the problem was expressed attests to the failure during the past 38 years of the federal government to either treat successfully, contain, or prevent narcotics addiction. The treatment centers at Fort Worth and Lexington, Ky., are public health centers primarily intended for addicted federal prisoners. Special admitting procedures are required for state prisoners and for voluntary patients.

There are 2,000 beds in these institutions. Since there are more than 4,000 addicted federal prisoners and many more addicted state prisoners, and the number of addicts in America is conservatively estimated at 60,000; and since treatment takes anywhere from 6 to 18 months, it follows that this federally sponsored treatment accomplishes next to nothing in solving the problem for the entire country.

The research done under the same institutional auspices probably should be relegated the same importance as the treatment. The scarcity of beds makes it next to impossible to have a patient admitted for the third time on a voluntary basis. But isn't addiction the disease in which the patient may relapse again and again and again . . . ? Even the additional treatment facilities in the District of Columbia, the State of California, and the State and City of New York are woefully inadequate to meet the demands. The statement that the average narcotics addict, for want of doctors, must turn to the peddler or to the police for treatment is as true today as it was 25 years ago.

The reluctance to accept ambulatory treatment, with drugs if necessary, as sound medical practice for narcotics addiction has resulted in today's excesses. It is ironic that a 1924 resolution of the American Medical Association—that ambulatory treatment of narcotics addiction was impossible—resulted in the establishment of the purest form of socialized medicine ever invented or practiced. History repeats itself, and in 1962 the AMA, to the "gratification" of the Federal Bureau of Narcotics, again voiced opposition to the principle of ambulatory treatment. Although this opposition was later

somewhat ameliorated by ambiguous clarifications, the AMA has not given a clear mandate to individual doctors, nor has it encouraged them to apply their skills to the problem of the addicted patient.*

This, then, is the "American system," a medical house divided in itself. Compare it to the "British system," which is no system at all, but represents the freedom of doctors to treat addicts on the basis of sound medical principles. What are these principles? In England, in 1926, the Rolleston Committee on Morphine and Heroin Addiction concluded that: ". . . morphine or heroin may be legitimately administered to addicts when the patient while capable of leading a useful and fairly normal life so long as he takes a certain non-progressive quantity, usually small, of the drug of addiction, ceases to be able to do so when the regular allowance is withdrawn." The advice of the Rolleston Committee was reviewed and confirmed by another interdepartmental committee, which reported in 1961: "The continued provision of supplies to patient addicts depends solely on the individual decision made by the medical practitioner professionally responsible for each case. We are strongly opposed to any suggestion that "registration" would be either desirable or helpful. Furthermore, we are impressed that the right of doctors in Great Britain to continue at their own professional discretion the provision of dangerous drugs to known addicts has not contributed to any increase in the total number of patients receiving regular supplies in this way."

The problem in the United States, because of the sheer number of patients involved, requires drastic measures. But to intimate that the national climate, cultural differences, racial origins, and so forth, are such that sound medical principles of ambulatory treatment cannot operate here is begging the question.

Hospitalization—on a voluntary basis, and frequently applied—is a very useful adjunct to ambulatory treatment, but because of the nature of addiction has to remain a supportive mode of treatment. To substitute "civil hospital commitment" procedures for ambulatory or voluntary institutional treatment is to call incarceration euphemistically hospitalization. To compare civil commitment for narcotics addiction to the forced hospitalization employed in certain infectious diseases lacks a basis in scientific medical reasoning. The so-called "failure of clinic treatment" which is constantly brandished as an argument by laymen is drawn from a distorted evaluation of the hastily organized medical clinics before 1923, which were unable to cope with the problems posed by the regulations of the Harrison Narcotics Act of 1914.[1]

The muddle following the first legal explorations of the Act caused the AMA to withhold endorsement of the clinics and of the individual practitioner treating addicts. Clinic treatment never had a fair chance in the USA.

In England the duties and rights of the doctor to treat a narcotics addict according to the dictates of his conscience have been kept intact and are defended by the medical societies against any and all critics, be they homegrown law enforcement agencies, governmental busybodies, or foreign snoopers and snipers. We could learn from their example.

—ROBERT HAUSMAN, M.D., San Antonio.*

*Ed. Note.—Since this article was written, the AMA approved for wide dissemination a joint statement of the AMA Council on Mental Health and the National Academy of Sciences—National Research Council regarding continued research on problems of addiction. The statement advocated modification of many of the restrictions on the use of narcotics since research creates special requirements. It added, however, that such activities must be done within limits of ethical medical practice.

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*Dr. Hausman is presently in Surabaja, Indonesia, under the sponsorship of the State Department.

Latent Image

The view has been wonderful! This year as President has provided me the opportunity to see the complete panorama of the Association. Little did I realize one year ago the magnitude of the services that are provided for over 9,000 members. More than 350 dedicated practicing physicians are serving on 52 councils and committees and 11 scientific sections. It has been an education and a pleasure to work with them and attend many of their meetings.

In traveling over the state I have enjoyed the hospitality and friendship of physicians in county and district medical societies where many more physicians are earnestly seeking and finding the solutions to a multitude of local problems. Freedom in the practice of our art still hangs in the balance—there is no sign of relief in the near future. As some of us move out of active duty, replacements are needed in all echelons of the Association, and it has been reassuring to meet these young physicians who will respond to the call. Component societies are obligated to help them to find their places in the service of organized medicine.

The picture would be only half complete without recognition of the full measure of service and devotion which is tirelessly given us individually and collectively by the Woman's Auxiliary. We would be lost without them!

Beyond our own ranks I have enjoyed the fellowship with paramedical organizations that are working with us to provide the best health care in the world for the people of Texas, and they are standing beside us in the front line of the battle to preserve the private practice of medicine. The Texas Medical Association is unique and fortunate to have the complete cooperation of all of these groups.

I thank all of you for allowing me the privilege of serving as your President—every minute will remain in my memory as a precious treasure for the remainder of my days.

Robert Mayo Jenny

President
Texas Medical Association

Surgical Treatment Of Parkinson's Syndrome

CLINICAL MEDICINE

IT HAS BEEN estimated that 34,000 new cases of parkinsonism occur every year in the United States. The incidence is four times higher in persons age 50 or over than in the younger individuals.[3]

The severe social and economic disability caused by the disease is further aggravated by a mortality rate which in parkinsonism appears to be at least one-third higher than in the normal population of the same age group.[3]

The disease affects both sexes, but is slightly more common in males than females.

The disease, which would better be called Parkinson's syndrome, has different etiologies. The common assumption that it is a postencephalitic disease has not been confirmed in recent studies; one large recent series shows that only 10 per cent of all cases studied could be ascribed to past encephalitis. More than three times as many cases were attributable to, or associated with, arteriosclerosis. More than half of all the cases in the series, however, could not be attributed to any particular etiologic factor. The possibility of an unrecognized or forgotten viral infection, not necessarily a clinically recognized encephalitis, must of course be considered.[3]

Another factor brought out recently by Mjones[4] was the remarkably frequent family history of dyskinesia of a parkinsonian type. In a careful study of the families of many patients, he found that in almost 40 per cent of the cases some family history of similar disease could be found, and he therefore concluded that a hereditary susceptibility of the extrapyramidal system may be a determining factor in the development of Parkinson's syndrome.

Clinical Features

Parkinsonism is a disease of middle and later life characterized by slowly progressive rigidity of the limbs, trunk, and face, a constant rapid tremor of the hands and to a lesser degree of the arms and legs. This tremor is present at rest and usually, but not always, diminishes on movement. The disability may often be greater in one limb in the early stages, or on one side, but eventually it progresses and both sides of the body are usually involved. Either the tremor or the rigidity may be the more troublesome and obvious feature. The characteristic part of the rigidity is the "cogwheel" sensation in which the limb moves in short and jerky steps whenever the affected limb is moved passively. Difficulties in phonation and swallowing are frequent and speech may become unintelligible. Rarely oculogyric crises are described.

Pathology

The pathology of Parkinson's syndrome is characterized by widespread lesions of the brain and brain stem, with more involvement of the nerve cells than fiber tracts. The pigmented nuclei of the brain, particularly the substantia nigra, the globus pallidus and substantia innominata of Reichert are affected, but lesions are also seen in the hypothalamus, the reticular formation, and other parts of the brain stem, the cerebellum, the thalamus, and the cerebral cortex. Lesions were also described in the locus caeruleus and the pigmented cells of the dorsal nucleus of the vagus. The most important pathological abnormalities summarized by Greenfield,[2] are in the melanin-bear-

When medical treatment of Parkinson's syndrome fails, the author advocates surgical treatment, noting that age, cardiac disease, and severe symptoms of bilateral parkinsonism are no bar to successful treatment. A method of exact placement of stereotactic lesions is described.

ALEXANDER GOL, M.D.

ing cells of the brain stem and the globus pallidus. It is possible that the atrophic changes of the nerve cells of the globus pallidus may be secondary to the lesions in the substantia nigra, since the substantia nigra has an important projection on this part of the basal ganglia.

Treatment

The treatment of Parkinson's syndrome can be divided into two phases. Phase one is entirely medical. During this period, medications such as Cogentin up to 14 mg. per day are administered—increasing the dosage gradually. Other effective agents are Disipal, Parsidol, Phenoxene, Kemadrin, and Artane. These can be tried alone and in combination up to the limit of tolerance. Other medications are of course available for the treatment of parkinsonism, but those named are the ones most frequently used. The most common side effects are difficulty of focusing and excessive dryness of the mouth. If the patient receives adequate relief from medication, without too severe side effects, he can continue for a period of many years on medication with a satisfactory range of activities.

It is our practice, on first seeing the patient, to ascertain whether he has had an adequate medication trial. If such has not been the case, then medications are tested in an intensive course, lasting perhaps two weeks depending on the response and medications that have been tried in the past. Prior to starting out on the trial of medication, the patient is taken off medication completely. Films are made during this stage to show maximum disability. Following an intensive trial of medication, the maximum benefit is compared with the status prior to medication and the decision is then made whether surgery is justified.

Many contraindications to surgery have been described; however, with increasing experience the contraindications have shrunk

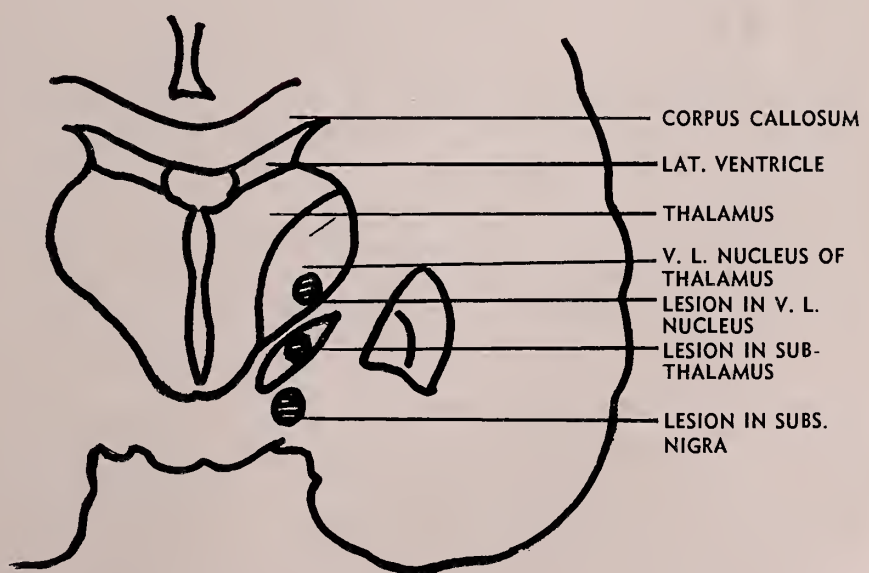

Fig. 1. Illustration shows the three nuclear masses mentioned in the text and their relation to the globus pallidus, an earlier site of stereotactic lesions. The actual lesion is placed in the more posterior parts of the ventrolateral nucleus, subthalamus and substantia nigra. (Fig. 5.)

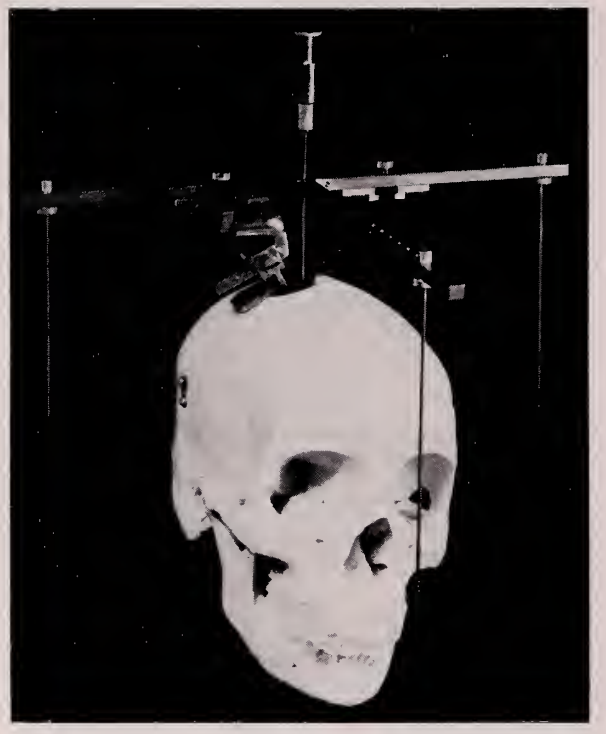

Fig. 2.

in multitude and importance. Age is no bar to surgical relief of the disease, since the operation itself is not a severe one and most patients are ambulant within a few days following the operations. Unilateral or bilateral disease is equally amenable to treatment. In bilateral disease, the side most severely affected is done initially; this is followed by a period of rest and reevaluation of about three months. If at the end of this time the patient feels that the amount of benefit obtained by having one side treated is not adequate for his daily needs, the other side is then treated. Frequently, patients with a minor disability on one side find themselves able to cope when the more severely affected side has been relieved by surgery.

Hypertension and cardiac disease have often been quoted as being an absolute contraindication, but we do not find this to be the case. The operation is done under local anesthesia. The patient is not required to perform any strenuous exercises. The procedure is relatively non-shocking, only mildly uncomfortable, and not associated with any blood loss of consequence.

The only absolute contraindication which we have carefully observed is one of severe mental disease. Under these conditions physical improvement hardly contributes to the patient's well being and therefore the result usually does not justify the effort.

We have operated successfully on patients up to the age of 75, and on several patients with serious cardiac disease without any ill effects.

The surgical procedure currently in use consists of placing a discrete small lesion into the lateral-ventral nucleus of the thalamus or sometimes the subthalamus and occasionally in the substantia nigra, under radiographic control. The site of the lesions is illustrated in Fig. 1.

The instrument used is one of two types; one developed by Dr. George Ehni of Houston and the other one by the author. The instrument is placed into the skull as seen in Fig. 2. Then roentgenograms are taken in the anterior and lateral planes while the apparatus is adjusted to superimpose the external pins of the instrument on the area of the desired lesion. When the pins have been aligned on the target area, an electrode is introduced through the center of the stereotactic machine. Since the anterior and lateral pins have been carefully superimposed on the target area, the electrode must enter the target area. When this happens, a noticeable alteration of tremor and rigidity is usually noted, showing that even the minute lesion created by the tip of the electrode affects the symptoms of the disease.

Stimulation is then carried out with a square wave generator up to a voltage of approximately five volts to obtain exact physiologic as well as anatomic localization of the target area. Stimulation will produce slight twitching of the muscles of the opposite limbs. This is the maximum motor effect when the electrode is correctly placed. Sensations of tingling or numbness may also occasionally be produced by stimulation. The desirable effect of the stimulation is an obvious change in tremor or rigidity. Two effects have been observed: either an increase in tremor or rigidity on stimulation, denoting that the area under test contributes to the production of the tremor and rigidity; or occasionally the reverse is observed with higher voltages, when stimulation produces a blocking effect diminishing tremor or rigidity.

If inadequate responses are obtained from stimulation by means of an ordinary straight electrode, occasionally a special electrode is introduced which contains a curved stylet that can be extruded radially and thus an

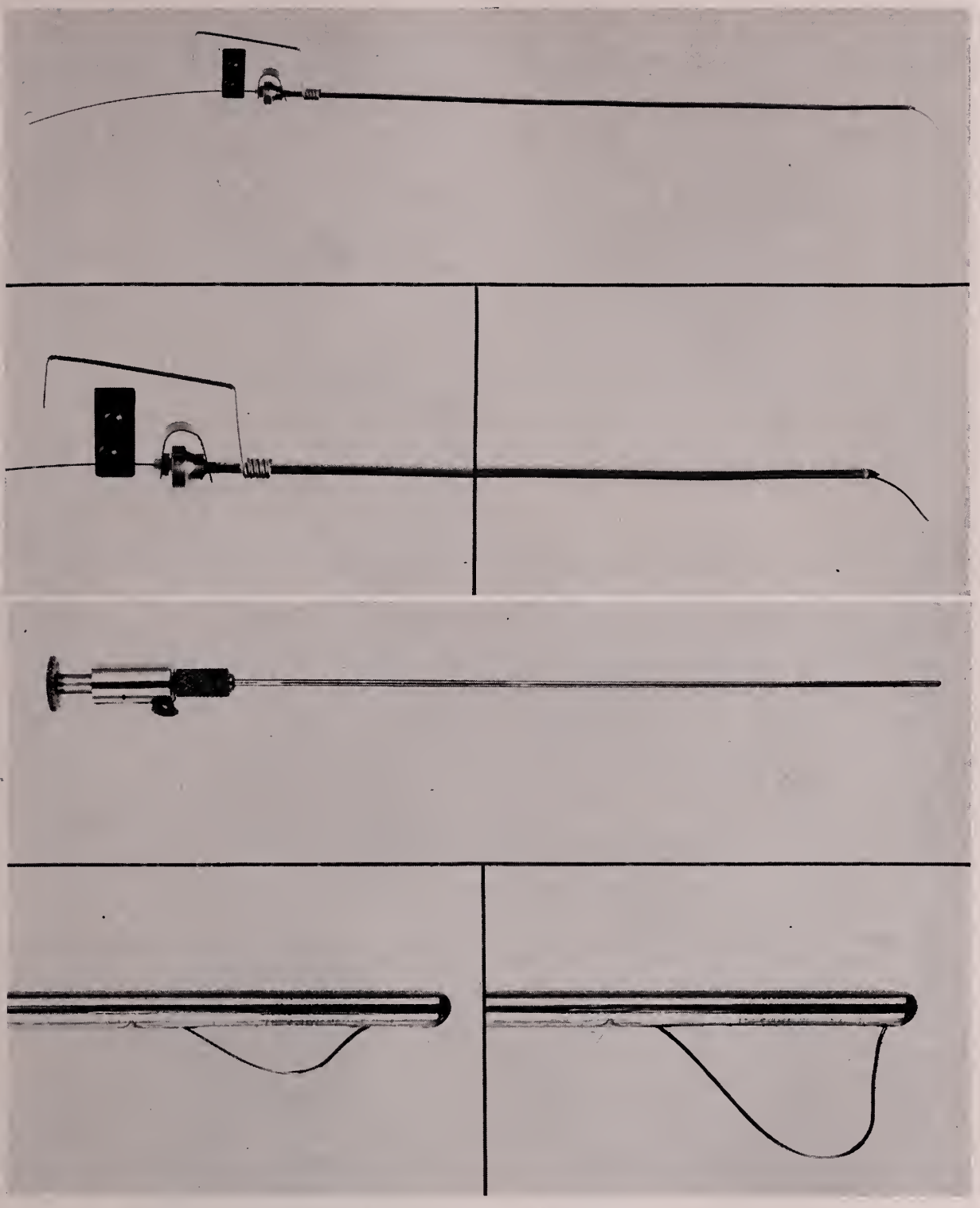

Fig. 3. (top) and Fig. 4 (bottom).

area of 2 cm. in diameter can be explored from a single puncture wound. This electrode is illustrated in Fig. 3. The area of maximal desirable effect of electrical stimulation is thus defined. The electrode is then withdrawn and a leukotome is introduced. The leukotome (Fig. 4) consists essentially of a long needle from which a stiff stainless steel loop can be extruded through a notch near the tip of the needle. This acts as a cutting loop when the needle is rotated.

After introduction of the leukotome, the

loop is extruded and small serial cuts are made with a small loop, and the patient is continuously tested to ascertain which quadrant of the lesion produces maximum effect. The quadrant or segment which produces the maximal effect is then enlarged until rigidity and tremor have been abolished. We find that rarely will a globular lesion be required since the structures which are to be destroyed are not globular themselves and the degree of anatomic variation is significant. The leukotome appears to be the most accurate method of producing a lesion for the relief of parkinsonism and is preferable to devices generating heat or cold which produce a globular lesion without directional control.

Results

The surgical control of parkinsonism has proved to be satisfactory. Patients leave the operating room showing complete relief of tremor and rigidity in the upper limb. A lesser degree of relief is obtained in the lower limb since there is a considerable amount of bilateral cerebral control over the lower limbs. Maximum improvement in the lower limb, in cases of bilateral Parkinson's syndrome, can only be expected after both sides have been treated.

Most patients get out of bed the day after thalamotomy and are then treated actively in the physical medicine department. Most patients can be discharged from the hospital within a week and often sooner.

The immediate excellent result of the operation endures in approximately 80 per cent of cases. In the remainder there is some recurrence of tremor or rigidity over the next few months. It has been our experience that if the patient has maintained his or her improvement for approximately four months, then no deterioration would occur for the whole follow-up period, which now amounts to three or four years in some cases.

Whenever deterioration in the original degree of relief is noted, a re-operation is usually advised to enlarge the original lesion. The result is usually good and the explanation may be that the lesion was in the correct site, but not large enough. As mentioned before, the lesions are of varying sizes and shapes, depending on the degree of improvement while the lesion is being created. It should be mentioned that the second operation, if such should become necessary, is even less stressing than the first one, and the patient is usually discharged from the hospital within a few days.

Complications

Complications in this form of surgery, though not frequent, can be severe. Only one patient died in our series of over 50 procedures, a mortality of less than 2 per cent. This patient died after a successful operation from which he recovered within a few days, being fully ambulant and active and showing no signs of parkinsonism. However, on the tenth day, he suddenly collapsed and died of a pulmonary embolus. Serial sections of the brain showed the operative lesion to be exactly where intended—in the posterior part of the ventrolateral nucleus of the thalamus (Fig. 5).

The histology in this case shows that a very small lesion in the correct site can control the clinical picture of Parkinson's syndome with virtually no damage to other closely related structures.

Of other complications, we have had two patients with transient severe hemiparesis who recovered completely, and one patient with severe hemiparesis who has a severe disability, although he is able to walk well.

In conclusion, it should be added that both surgical and medical treatment of parkinsonism, although frequently providing satisfactory control of the disease, may not do

Fig. 5. Shows lesion in the posterior part of the ventrolateral nucleus just above the subthalamus.

so permanently. This is easily understood if it is realized that diseases such as arteriosclerosis are progressive and therefore a patient so afflicted gradually deteriorates. All that surgery and medication can accomplish is to provide a temporary remission, although a remission of several years is undoubtedly desirable. In postencephalitic parkinsonism or parkinsonism of undetermined etiology, the pathology may not be progressive and the outlook for treatment is, of course, much more permanent.

Summary and Conclusions

Parkinson's syndrome is an important and relatively frequent disease with an estimated annual incidence of 34,000 new cases in the United States alone. Many etiologic factors have been recognized in the pathogenesis of the disease. The disease tends to be progressive, severely disabling, and leading to complete and enduring disability. It deserves, therefore, the most intensive efforts aimed at alleviating the suffering of the patient. Medical treatment with maximum doses of Cogentin, Phenoxene, Disipal, Parsidal, Kemadrin, and Artane is desirable in the first instance, but when all chemical measures have been exhausted, the patient should be offered the benefit of surgical treatment.

Dr. Alexander Gol is a radiological surgeon in Houston.

Age, cardiac disease, and severe symptoms of bilateral parkinsonism are no bar to successful surgical treatment.

A method of exact placement of stereotatic lesions has been described. This method takes account of anatomic variability and the actual shape of the structures in which the lesion is made.

The surgical treatment of Parkinson's syndrome has reached a point where it is one of the most precise procedures known to modern surgery. The mortality rate is remarkably low and the ratio of undesirable complications is acceptable.

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Dr. Gol, Hermann Professional Building, Houston 25.

Fatal "Blood Poisoning"—Remember?

Because of combined efforts of pharmaceutical concerns and physicians, some of the greatest killers exist no longer.

"Several decades ago we stood helplessly by as we watched people die of meningitis, typhoid fever, influenza, polio, tuberculosis, diphtheria, streptococcus, and many other infections. Hundreds of thousands of children died or were maimed with blindness, deafness, and paralysis from such ailments as diphtheria, scarlet fever, and polio. Pneumonia was a great killer. In recent years, we do not hear any more of anyone dying of a ruptured appendix. It is now most unusual, although very frequent before. . . . Infections were untamed without sulfas and antibiotics. A simple scratch could cause blood poisoning and death. President Coolidge lost a teenage son as the result of an infection of a little blister on his foot," comments Dr. William L. Gould in *New York State General Practice News*, Nov.-Dec. 1963.

Metabolic Role of Neutral Fats (Triglycerides) In Coronary Arteriosclerosis

REVIEW ARTICLES

CORONARY ATHEROSCLEROSIS is characterized by lesions with a high cholesterol content localized in the subendothelial stratum of the arterial intima. Phagocytic cells collect at this site. These cells contain cholesterol crystals in both fluid and solid forms. Stimulated by the excess cholesterol in the phagocytic cells, the lesion progresses to a proliferation of connective tissue. Later, deeper strata of the intima are invaded. Necrosis occurs here because plasma diffusion is insufficient to nourish the intima. With ultimate repair by connective tissue, vascularization and calcification result.[17, 26]

Formation of these lesions has been related to a disturbance in cholesterol metabolism.[18,25] The importance of the relationship of other blood lipids to cholesterol as a factor in atherosclerosis also has been emphasized.[18] The initial stage in the development of human arteriosclerosis, however, consists of local alterations which are responsible for the subsequent precipitation of lipoid in the affected areas. These localizing factors and the deposition of lipoids in the interstitial structures of the intima are merely an index of other more important alterations which have occurred in this stratum. The deposit contains not only lipoid but protein substances and the subsequent alterations which occur in the artery are not the result of lipoid-cholesterol substances, for when these *per se* are injected into the tissues these alterations do not occur.[22]

No one has seriously questioned the basic concept,[17] but there are other factors that enter into the pathogenesis of atherosclerosis, including mechanical, physical, other local vascular and metabolic disturbances, and coagulation and fibrinolytic disorders. The development and progression of atherosclerosis is the result of the interrelation of several local and systemic factors, the relative importance of which may vary among individuals and in the same individual at different times.

Physiology

Natural fats and oils consist almost entirely of neutral fat or triglycerides (fatty acid esters of glycerol). Separated natural fats consist of approximately 99 per cent triglycerides as mixtures and are the most prevalent group of lipids in both plant and animal tissues. The normal serum concentration of triglycerides in persons in the third decade is 100±, approximately 25 mg. per 100 ml. (standard deviation). The range for 95 per cent of the normal United States population would be between approximately 25 and 175 mg. per 100 ml. Values in excess of 250 mg. per 100 ml. must be considered indicative of hypertriglyceridemia, and 150 to 200 mg. per 100 ml. should be considered the range of normal. Since lipids are insoluble in water they require special vehicles to effect their transport in serum. Lipid being transported is combined with protein and usually with other lipids to produce a variety of lipoprotein complexes. These have been studied with several techniques to effect separation; for example, by electrophoresis, fractionation, and ultracentrifugation. The alpha are designated high density and the beta low density lipoproteins. The lipid transported is mostly triglyceride in the chylomicrons and in low density beta lipoproteins.

When fat is ingested, triglycerides are either completely or partially hydrolyzed.

The author reviews the literature and studies the present concepts of the metabolic role of triglycerides in coronary arteriosclerosis. He notes that one of the important factors to be observed now is recognition of persons who are susceptible to coronary artery disease early enough to initiate a program of prevention.

CARL F. SHAFFER, M.D.

These are mixed with other lipids, resynthesized, and absorbed with small amounts of protein to form chylomicrons, the least soluble of the lipoproteins, which produce the visible lactescence in the plasma. Chylomicrons are present normally only when large amounts of absorbed fat, principally triglycerides, are entering the blood. They are rapidly removed from normal subjects but the mechanism has not been completely elucidated.[11]

The serum lipids circulate as four major classes of compounds: the high and low density lipoproteins, the chylomicrons, and the free fatty acids. The latter, although of physiologic importance, constitute a negligibly small fraction of the total serum lipids. The high density, or alpha lipoproteins, are not implicated in coronary artery disease except insofar as they may be relatively or absolutely decreased. Increased lipid concentration of serum can usually be attributed to an increase in one or both the remaining categories, the low density, or beta lipoproteins, and the chylomicrons.[14] The latter do not constitute a distinct entity and their route through the circulation and the mechanism of their removal vary considerably with the nutritional state.

Pathophysiology

The cause of increased serum triglycerides apparently lies in the defective removal of exogenous or endogenously synthesized triglycerides from the blood. Evidence available from a variety of studies indicates that interference with an enzymatic lipemia-reducing factor is the main cause of hypertriglyceridemia. The cause of essential (familial) hypertriglyceridemia is unknown.[15]

The lipid materials usually found in blood plasma and tissue fluids are cholesterol, cholesterol esters, phospholipids, glycerides (mainly triglycerides), and unesterified fatty acids. The present evidence of the metabolism of lipids supports the concept that, *in vivo*, lipoprotein lipases have a major role in the post-absorptive removal of chylomicra from the plasma.[24] These enzymes are finely emulsified fat globules and consist mainly of triglycerides but also contain cholesterol, phospholipid, and protein. Phospholipid may be considered relatively unimportant. Various properties of lipids are combined with proteins to form lipoproteins with different physicochemical characteristics. The lipoproteins may be separated into a number of fractions according to their separate composition and other physicochemical properties.[13]

The disappearance of the chylomicra appearing in plasma after the absorption of ingestive fats provides one of the most intriguing problems in the field of fat metabolism.[30] It is probably for this reason that investigation of the role of glycerides (mainly triglycerides) has assumed major importance in the relationship to the development of coronary artery disease.[2] The enzyme lipoprotein lipase specifically catalyzes the hydrolysis of triglycerides to fatty acids and glycerol.[10] This releases unesterified fatty acids and desaturates both lipemic plasma[23] and triglyceride emulsion.[14]

The lipolytic enzyme lipase has an important role in the reduction of the concentration of serum triglycerides. However, proteolytic enzymes, particularly vasculokinase, may eventually prove to have an equally important role in the pathophysiology related to atherosclerosis.[31]

Various investigators have evaluated the different lipoprotein categories and serum cholesterol in coronary artery disease, but only recently have studies appeared in which triglycerides also were measured.[2, 9] In each instance the triglycerides were found more frequently elevated than the cholesterol. In a recent study, a serum triglyceride concentration of 5.4 mEq. per liter (expressed as triglyceride fatty acid) best separated the normal from the coronary population.[1] There has been no extensive study of the serum triglyceride concentration but an analysis of various studies indicates that triglycerides compare favorably with other factors in separating the normal from the coronary artery disease population. The serum cholesterol concentration is more often normal than not in coronary artery disease. This was evident in a recent study of approximately 2,000 men in the coronary age group; those persons who developed coronary disease had a lower average cholesterol level than those who did not develop coronary artery disease.[32] In contrast, in the study measuring triglycerides the concentration exceeded the previously stated figure in more than 85 per cent of coronary patients.[12] This included all age groups and those over age 50 had serum triglyceride levels of 5.5 mEq. per liter or higher. A similar association has been noted between high serum triglyceride levels in normal persons and a family history of coronary artery disease.[8] The use of triglyceride determinations as an indicator of coronary prone individuals is also facilitated by the ease of technical procedure and interpretation.[27]

The frequent occurrence of fasting hypertriglyceridemia in coronary artery disease is also correlated with an increase in chylomicrons.[29] Observations in the literature support the concept of a rapid phase of triglyceride removal pertaining mainly to chylomicrons, and a second slower phase concerned with the disposition of the low density lipoprotein subsequently derived from chylomicrons. A metabolic error in one or the other of these two processes would thus produce an accumulation either exclusively of the elementary chylomicrons or of the low density lipoproteins, in either event resulting in an accumulation of triglyceride in the serum.[16] Elevations of serum triglycerides occur in 85 to 90 per cent of patients with coronary disease.[1]

Diet and Cholesterol

From a brief summary of cholesterol balance it is apparent that the control of cholesterol remains to be attempted by at least several methods: inhibition of cholesterol biosynthesis; inhibition of absorption or reabsorption of cholesterol; promotion of cholesterol oxidation and increased excretion of its degradation products; and increased removal by tissues of cholesterol.[6] The current hypotheses are inclined toward the concept that any agent which raises serum fat for prolonged periods of time promotes the development of atherosclerosis. The prolonged elevation of neutral fat (hypertriglyceridemia) involves the coronary artery as contrasted with the other arteries while hypercholesterolemia appears more liable to favor the development of peripheral arteriosclerosis.[28] The American Medical Association Council on Foods and Nutrition believes a causal relationship between low cholesterol values and low mortality rates for coronary artery disease has not been proved and furthermore that there is insufficient evidence available to warrant a change in the American diet aimed at preventing heart disease in the general population.[4] The value of an antiatherogenic diet is to allow the recognition of persons, by at least their third decade of life, who are susceptible to coronary artery disease because of hereditary or endocrine (metabolic) factors.[34]

The diet, with an ideal polyunsaturated to saturated (PS/S) ratio and restricted cholesterol, could achieve but a maximum average of 20 per cent reduction in circulating blood cholesterol. This is due to the continual endogenous synthesis of cholesterol. Nutritionists agree that severe restriction of dietary fat is undesirable and such restriction is usually required (ingestion of 20 gm. per day or lower) to reduce cholesterol levels in hypercholesterolemic persons.[20]

Dr. Carl Shaffer practices internal medicine in San Antonio. This paper is a follow-up report to an article he wrote in the May, 1963, issue of the *Journal*.

Diet and Triglycerides

The concept that excessive total caloric intake is deleterious to health and associated with increased incidence of coronary artery disease has been implicated for years by life insurance and other statistics. The hyperlipemic effect of dietary fat is restricted to saturated or animal fat. Unsaturated fat or vegetable oils have the opposite effect of lowering serum lipid concentration.[21] The lipid lowering effect of the substitution of vegetable for animal fat has not been entirely explained. There is no certainty whether this effect is due only to the removal of some cholesterol-raising substance in animal fat or to some specific cholesterol-lowering action of the vegetable oil itself. Post-absorptive serum triglycerides are not as susceptible as cholesterol to the amount and type of dietary fat *per se* but are greatly susceptible to dietary carbohydrates and to total calories. A number of investigators have observed the present high serum triglyceride level associated with high dietary carbohydrate.[1, 5]

In the more common carbohydrate-induced lipemia (as compared to animal fat-induced) the triglycerides are probably derived from endogenous fat synthesized from carbohydrate. It is notable that the glycerides constitute approximately 99 per cent of adipose tissue in man.[11] The sensitivity of triglycerides to carbohydrate and total calories probably accounts for the low triglyceride level in the serum of ethnic groups consuming high carbohydrate, low fat, and low caloric diets.[19] This sensitivity may have prevented elevation of serum triglyceride caused by low fat diets, commonly recommended today. The significance of the opposing action of dietary fat and carbohydrate on post-absorptive concentration of serum triglyceride in the etiology of hypertriglyceridemia in coronary atherosclerosis remains unsolved.[7]

Comment

Hyperlipidemia is a nonspecific reference to all conditions in which one or more lipid constituents of the serum are present in abnormally large amounts. Hyperlipemia is used to describe the condition in which serum lactescence is associated with an increased triglyceride concentration.[11] There have been studies of diets in relation to the mortality rates of degenerative heart disease which have shown the incidence of coronary arteriosclerosis in direct proportion to the population consumption of diets high in animal fat and total calories.[7, 19] Another group of studies have contradicted this.[8, 28] Both of these studies used serum cholesterol measurements as an index of circulating blood lipids. The former study determined that the level was higher than normal (250 mg. per ml. for adult men) and the latter determined that it was lower than normal. The former group assumed that because the level of blood cholesterol was high, this implied that hypercholesterolemia was an etiologic factor in coronary atherosclerosis. The results of the latter group contradicted this implication. Based on the results of clinical research published earlier,[33] the subject of the dietary role of cholesterol in the etiology of coronary atherosclerosis is reviewed.[34] Relative to the effect of lipid substances which involve the pathogenesis of atherosclerosis, it was concluded that available evidence does not support a dietary factor of increased cholesterol as a cause of coronary atherosclerosis, nor does evidence show that dietary restriction of cholesterol affects the disease favorably in coronary atherosclerosis.

Heretofore, the emphasis on the estimation of the concentration of blood lipids has been virtually on the determination of cholesterol, but current knowledge indicates that emphasis should be equally placed on determination of glycerides in the plasma. Interpretation of lipid measurements in terms of (1) what constitutes normalcy; (2) differentiation between genetically and environmentally caused lipid elevation; and (3) the use of drugs and diet to counteract hyperlipidemia are important factors. Glycerides should be measured routinely in all men over 30, and in certain other high-risk groups, notably children whose histories suggest the possibility of genetically determined lipid abnormalities. Measurements of glycerides and cholesterol may complement each other.

Studies (as that in Framingham, Mass.)[17a] which have demonstrated increased coronary heart disease in men with serum cholesterol in excess of 250 mg. per 100 ml., indicate the value of cholesterol measurements, especially in selecting persons with high coronary predisposition. However, measurement of plasma triglycerides is more valuable, with the greatest potential coronary disease in those

whose triglyceride levels tend to rise after age 25. This rise can be correlated with a weight gain of more than 10 pounds after that age, even in individuals who become only moderately obese.[3] Morever, triglyceride elevation may occur in the presence of a normal or nearly normal cholesterol. This is true because cholesterol can be found both in the alpha and beta lipoprotein fractions, and a deficit in the alpha lipoproteins may disguise an elevation in cholesterol in the beta lipoproteins, where most triglycerides occur. The measurement of lipoproteins is therefore also of secondary importance. The biochemical disturbance that appears to be one of the most predominant is in carbohydrate metabolism, secondarily affecting endogenous glyceride metabolism. A number of observations implicate an association between coronary artery disease, elevation of serum triglyceride, and abnormal glucose tolerance. A diabetic type of glucose tolerance is common in atherosclerosis.[35]

The ideal in measuring blood lipids is attempting to establish hyperlipidemia which is genetically determined. Since there are no direct studies capable of proving genetic determination, this must be done on the basis of plasma triglyceride levels of the original subject and on blood relatives available for study. The glycerides give valuable information on metabolism, not only of fats but also of carbohydrates. Probably the most common reason for increased plasma triglyceride content is an excessive demand for fat calories because of impaired carbohydrate metabolism. Under such circumstances, fats are mobilized from the adipose tissues and these fats are returned to the circulation from the liver in the form of glycerides. During periods of caloric excess, or when carbohydrate intake is extremely high, the plasma triglyceride content may rise significantly. A comprehension of these basic metabolic patterns is of major importance in designing diets and medical therapy aimed at combating hyperlipidemia. In such diets, it is necessary to avoid reducing fats to a level at which caloric needs are met by excess carbohydrate.

In attempting to treat most cases of hyperlipidemia today, one can design a diet with enough fat—preferably of an unsaturated type—to counteract unreasonable carbohydrate excess. Such a diet typically includes a caloric content established to bring weight toward the "ideal," a fat ratio amounting to 25 to 35 per cent of calories, and avoidance of excessive carbohydrates.

Measurement of a circulating blood lipid has a vital role in the increasing effort to diagnose and prevent atherosclerotic heart disease, and for the reasons heretofore described, triglyceride measurements should be relied upon primarily, with cholesterol and lipoprotein measurements of secondary importance.

Summary

The following may be stated regarding the effects of lipid substances which involve the pathogenesis of coronary atherosclerosis:

1. There is a high lipid content of the lesions in human atherosclerosis.
2. The disturbance of the biochemical mechanism in essential familial hypertriglyceridemia appears to be predominantly in carbohydrate metabolism, secondarily affecting glyceride metabolism.
3. There is a uniform hypertriglyceridemia in patients with coronary arteriosclerosis, apparently due to a defective removal of exogenous or endogenously synthesized triglycerides from the blood. A concomitant hypercholesterolemia is apparently a secondary physicochemical effect.
4. There is an increased prevalence of atherosclerosis in persons who have abnormalities in lipid metabolism, such as in diabetes mellitus. This, together with a diabetic type of glucose tolerance curve in persons with atherosclerosis, suggests that impaired carbohydrate metabolism may be as important a factor as impaired lipid metabolism in the pathogenesis of atherosclerosis.
5. Any mechanism which increases serum lipids for prolonged times promotes coronary atherosclerosis. Prolonged hypertriglyceridemia primarily involves the coronary arteries in contrast to hypercholesterolemia which is more related to peripheral arteriosclerosis.
6. The measurement of serum triglycerides is apparently a more important index of both potential and existing coronary arteriosclerosis as compared to measurement of cholesterol and lipoprotein levels.

Conclusions

Lipids are related to the pathogenesis of human atherosclerosis, but whether the im-

portance of factors in lipid metabolism are primary, contribute to, or only promote by participating in a different basic process remains to be elucidated.

The identification of susceptible persons (to coronary artery disease) in early adult life, by measurement of plasma glycerides, appears to be one of the most important factors at the present time in the program of prevention of coronary arteriosclerosis. A serum triglyceride level above 250 mg. per 100 ml. is significant and demands continued observation.

A modification of the diet as related to fat content probably benefits only certain persons: those with hypertriglyceridemia associated with a genetic inheritance, endocrine (metabolic) disturbances, and possibly obesity.

A modification of the diet as related to total calories, particularly from carbohydrates, appears to be more important in order to benefit the "susceptible" and "nonsusceptible" persons in early adult life in the program of prevention of coronary arteriosclerosis.

Atherosclerotic development and progression is influenced by other factors, which emphasizes the complexity of the pathogenesis of coronary arteriosclerosis.

Addendum

Since this article was submitted for publication an editorial in the *Annals of Internal Medicine* (Rationale for Treatment of Hyperglyceridemia, Ann. Intern. Med. 59:581 (Oct.) 1963) reviews more recent publications not contained in the present article and states that "the appearance of these papers . . . provides an opportunity to review the evidence implicating serum lipids in the etiology of atherosclerosis and particularly to emphasize the importance of serum triglycerides", concluding that, with the exception of the uncommon familial hypercholesterolemia, triglycerides are the more reasonable object of therapy than cholesterol.

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Isolated Myocarditis

CASE REPORTS

FIEDLER,[4] IN 1899, described an acute diffuse interstitial myocarditis in which the inflammatory process was isolated to the myocardium. Later, a focal or diffuse granulomatous type of isolated myocarditis and advanced interstitial fibrosis were considered components of this entity.[8,10,12,17] Clinical and pathologic characteristics of isolated myocarditis which justify its retention as a nosologic entity are: (1) an acute, subacute, or chronic clinical course of progressive myocardial insufficiency characterized by refractory congestive heart failure, embolic episodes, arrhythmias, and sudden death; (2) absence of a history of ischemic, hypertensive, or rheumatic heart disease; (3) an obscure etiology; (4) absence of a primary systemic infection or disease elsewhere in the body to which the myocarditis could be secondary; and (5) diffuse or circumscribed nonspecific inflammatory, necrotic, and/or fibrotic lesions confined to the myocardium in the absence of significant pericardial, endocardial, valvular, or coronary disease at necropsy. Further investigation will probably resolve this condition into several distinct etiologic entities with similar clinical and pathologic features.

Isolated myocarditis, a diagnosis formerly saved for the pathologist, is now made with a high degree of accuracy by clinicians. Since this form of myocarditis is not too well appreciated, particularly the acute type, the writers' recent experiences and a brief review of the subject may be of interest to other clinicians.

During the past five years, six cases of isolated myocarditis have been observed at Hermann Hospital in Houston. The clinical course was acute in four cases, subacute in one, and chronic in one.

Case 1.—Clinical findings: A 47-year-old Negro woman was admitted to Hermann Hospital July 29, 1960, unconscious and in shock. According to the referring physician she had always been in excellent health and had never experienced angina, dyspnea, nor edema. There was no history of rheumatic fever nor hypertension. On July 25, 1960, she developed mild upper abdominal pain and, subsequently, nausea and vomiting. On July 29, 1960, she became febrile with a temperature of 102 F., went into shock, and lapsed into coma.

At physical examination on admission the patient was unresponsive, the skin pale and moist, and the pulse and blood pressure unobtainable. The cardiac sounds were distant and of poor quality, and the rate was regular at 120 beats per minute. There was no cardiomegaly. Rhonchi were noted in both lung bases. Hepatomegaly was the only significant finding on abdominal examination.

Laboratory data: Urinalysis showed specific gravity, 1.031; protein, 2 plus; sugar, negative; and formed elements, leukocytes, 6-8, erythrocytes, 4-6, and hyalin casts, 10-15 per high power field. Complete blood count included hemoglobin, 17 gm. per 100 cc.; leukocytes, 15,750 per cu. mm., differentiated as polymorphonuclear neutrophils, 72 per cent; stab forms, 8 per cent; lymphocytes, 14 per cent;

Fig. 1. *Case 1.*—Sinus tachycardia; rate 125 beats per min.; QRS interval, .06 sec.; P-R interval, .14 sec.; and segmental shifts indicative of recent anterior and diaphragmatic injury.

Case 2.—Sinus rhythm; rate 88 beats per min.; P-R interval, .10 sec.; QRS interval, .07 sec.; and minor S-T segment changes. Electrocardiogram was considered to be within normal limits.

Case 3.—Sinus rhythm; rate 114 beats per min.; frequent ventricular extrasystoles; P-R interval, .14 sec.; QRS interval, .14 sec.; and S-T segment and T-wave changes compatible with complete left bundle-branch block.

Case 4.—Sinus rhythm; rate 120 beats per min.; P-R interval, .16 sec.; QRS interval, .10 sec.; intraventricular conduction defect; and low voltage.

Case 5.—Atrial fibrillation; rate 140 per min.; QRS interval, .12 sec.; Q-T interval, .28 sec.; left axis deviation; and left bundle-branch block.

Case 6.—Complete heart block; atrial rate, 96 per min.; and ventricular rate, 32 per min.

Considerable clinical confusion frequently attends the recognition of isolated myocarditis (Fiedler's). Although the symptoms, signs, and laboratory findings in this entity are nonspecific, if suspected, it can be diagnosed clinically.

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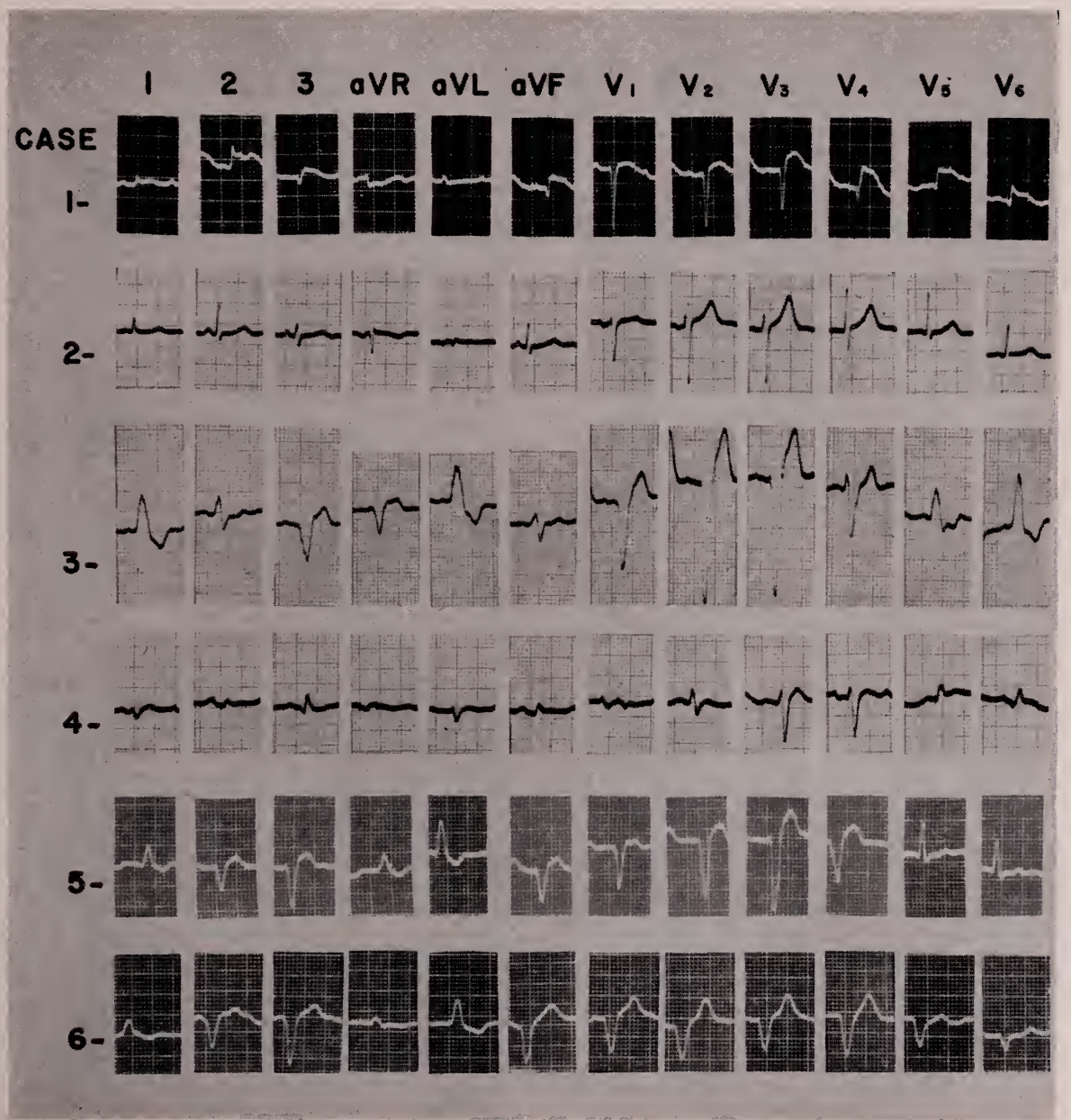

and monocytes, 6 per cent. Blood urea nitrogen was 98 mg. per 100 cc. Carbon dioxide combining power was 19.9 mEq per liter. Serum sodium, potassium, chlorides, and amylase were normal. Kolmer's test was negative. Spinal fluid was normal. Electrocardiographic findings (Fig. 1) were interpreted as indicative of acute anterior and diaphragmatic infarction. An electrocardiogram taken the next day showed no change.

The clinical course was one of progressive deterioration, despite administration of vasopressors, antibiotics, intravenous fluids, and supportive treatment. On July 30, 1960, a tracheostomy was done. After transitory stabilization, hypotension, tachycardia, and pyrexia progressed and the patient died of congestive failure on July 31, 1960.

Autopsy findings: Minimal left pleural effusion, congestive hepatomegaly, and ascites were present. The lungs were congested, and terminal bronchopneumonia was evident. The heart was hypertrophied, weighing 440 gm. The endocardium and epicardium were smooth and translucent, and the coronary vessels were free of thrombi and atheromata. The myocardium was ecchymotic, particularly in the papillary muscle and the subendocardium.

In microscopic sections of the myocardium (Fig. 2) there was an interstitial infiltration of polymorphonuclear leukocytes, lymphocytes, and a disproportionate number of eosinophils. Hemorrhagic areas were numerous, particularly in the subendocardium. Muscle fibers were swollen and occasionally necrotic, with fragmentation of the nuclei. Interstitial fibrosis was present, and all sections of myocardium contained focal bundles of loose, faintly basophilic fibrous connective tissues. The pathologic diagnosis was acute isolated myocarditis.

Case 2.—Clinical findings: A 51-year-old white man had been in excellent health until 11:30 a.m. Oct. 21, 1957, when severe retrosternal pain developed, radiating across the anterior chest and into both arms. There was no past history of similar pain, dyspnea, rheumatic fever, hypertension, nor recent infection.

At the time of physical examination the patient was acutely ill, with respirations, 24 per minute; pulse, 110 per minute; and blood pressure, 110/80 mm. of mercury. The heart was not enlarged and no murmurs were noted. Heart sounds were of poor quality, and a diastolic gallop was discernible. The lungs were clear to auscultation and percussion. Abdominal examination was negative.

Laboratory data: Urinalysis and complete blood count were uninformative. All findings were normal, as was the electrocardiogram (Fig. 1).

Anginal pain continued. The patient developed tachycardia, hypotension, and pulmonary edema and died of congestive heart failure at 3:05 p.m. Vasopressors, oxygen, and digitalis were ineffective.

Autopsy findings: The lungs and liver were congested, and the pericardial sac contained 100 cc. of serous fluid. The heart appeared normal and weighed 360 gm. The endocardium and pericardium were smooth and translucent and the coronary vessels were patent.

In microscopic sections of the myocardium (Fig. 3) there was sparse interstitial infiltration of lymphocytes, polymorphonuclear neutrophils, and eosinophils. In some areas the sarcoplasm of the myocardial fibers was lost. Focal deposition of interstitial connective tissue at the expense of myocardial fibers was observed. The pathologic diagnosis was acute isolated myocarditis.

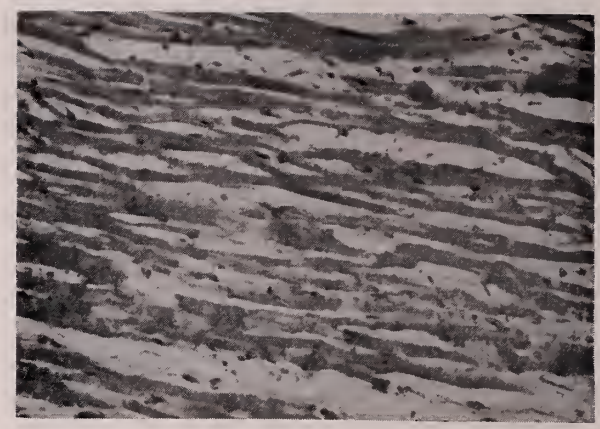

Fig. 2. *Case 1.*—Photomicrograph (x 69) of myocardium.

Case 3.—Clinical findings: A 78-year-old white woman previously in good health, experienced cough and dyspnea for three weeks prior to hospital admission. She developed severe substernal pain with profuse perspiration and a rapid irregular pulse. Digitalis and rest restored the rhythm to normal; however, frequent extrasystoles continued. Subsequently, with another episode of severe substernal pain, dyspnea, cough, and sweating, the patient was admitted to Hermann Hospital. There was no history of rheumatic fever, hypertension, nor recent infection.

This senile patient appeared acutely ill at physical examination; she was pale, cyanotic, and sweating profusely. Temperature was 101 F.; the pulse was totally irregular at a rate of 140 per minute; and blood pressure was 190/130 mm. of mercury. The heart was not enlarged. Heart sounds were distant and an apical diastolic gallop was present. No murmurs were audible. The lungs were filled with moist rales and the liver was palpable. Peripheral edema was absent.

Laboratory data: The urinalysis was normal. Complete blood count revealed a hemoglobin of 12.9 gm. per 100 cc.; erythrocytes, 4,300,000 per cu. mm.; and leukocytes 16,200 per cu. mm., differentiated as polymorphonuclear neutrophils, 94 per cent, stab forms, 1 per cent, and lymphocytes, 5 per cent. An electrocardiogram revealed left bundle-branch block (Fig. 1).

Pulmonary edema, tachycardia, and hypotension progressed, despite oxygen, Mercuhydrin, digitalis, aminophylline, and vasopressors, and the patient died from congestive heart failure 18 hours after admission.

Autopsy findings: The lungs and liver were congested. The heart was hypertrophied and weighed 445 gm. Endocardium and pericardium were smooth and translucent. Minimal calcification and fibrosis of the aortic valve were observed; however no stenosis was present and the coronary ostia were widely patent. Serial transections of the coronary arteries exposed only slight to moderate atherosclerotic changes and no occlusion nor thrombosis. The myocardium appeared normal, but on microscopic examination (Fig. 4) an intense interstitial infil-

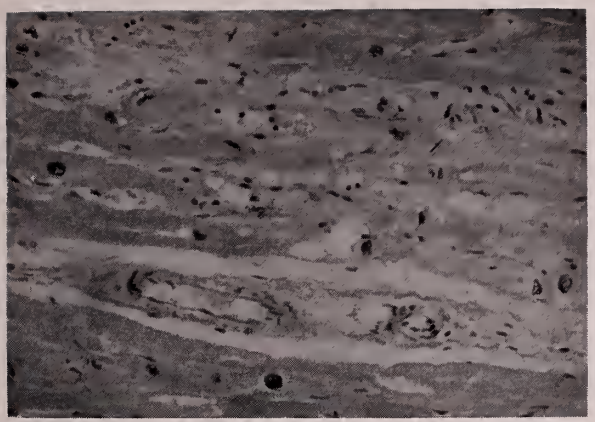

Fig. 3. Case 2.—Photomicragraph (x 143) of myacardium illustrates sparse leukocytic infiltratian with lass af fiber sarcaplasm.

tration by polymorphonuclear leukocytes and lymphocytes, moderate interstitial fibrosis, and hypertrophied myocardial fibers were observed. The pathologic diagnosis was acute isolated myocarditis.

Case 4.—Clinical findings: A 21-year-old white woman had been healthy until two months before hospital admission when she noticed increasing fatigue. Three weeks before admission, ascites, ankle edema, and dyspnea developed. During the next two weeks anasarca, cough, orthopnea, and, finally, total incapacitation occurred. The only significant past illness was diphtheria when the patient was five years old.

At physical examination the patient was acutely ill, dyspneic, orthopneic, and had distended neck veins. Temperature was 98.4 F. The pulse rate was 120 per minute, blood pressure 80/50 mm. of mercury. The heart was enlarged, the pulmonary component of the second heart sound was accentuated, and an apical diastolic gallop was present. There were no cardiac murmurs. The lungs were filled with moist rales. The liver was enlarged, and ascites and peripheral edema were conspicuous.

Laboratory data: The urinalysis revealed a specific gravity of 1.015, protein 1 plus, and was otherwise normal. The complete blood count was also normal. Erythrocyte sedimentation rate was 5 mm. per hr. Blood urea nitrogen was 22 mg. per 100 cc. The hepatic battery was characteristic of chronic congestion of the liver. Cardiomegaly, prominence of the left ventricle and pulmonary artery segment, and bilateral pulmonary congestion were evident in roentgenograms of the chest. The electrocardiogram (Fig. 1) showed an intraventricular conduction defect and low voltage. A preterminal electrocardiogram showed atrial fibrillation and right axis deviation.

The patient received digitalis, oxygen, Mercuhydrin, Levophed, and intravenous cortisone. Despite these measures congestive heart failure progressed and death occurred 24 hours after admission.

Autopsy findings: Passive congestion of the lungs, liver, and spleen were observed. Bilateral pleural effusions and minimal pericardial effusion and ascites were present. The heart was dilated and hypertrophied, and weighed 400 gm. Valves, endocardium, and pericardium were normal, and the coronary vessels patent. Apical subendocardial hemorrhage was noted. Microscopically, an intense interstitial leukocytic infiltration was observed in the myocardium (Fig. 5), and the muscle fibers were hypertrophied and occasionally necrotic. The pathologic diagnosis was subacute isolated myocarditis.

Case 5.—Clinical findings: A 55-year-old colored man was admitted to Hermann Hospital on Aug. 24, 1960, with increasing weakness, dyspnea, orthopnea, abdominal pain, nausea, vomiting, and pedal edema. These signs and symptoms had been progressive for one week before admission. The patient had first experienced dyspnea on exertion in 1956. In 1957, he was told that his heart was enlarged, and, in 1958, he was seen in the Outpatient Department with orthopnea, paroxysmal nocturnal dyspnea, cough, and weakness. On this occasion the pulse was 110 per minute and blood pressure 120/96 mm. of mercury. The electrocardiogram revealed left axis deviation, left ventricular strain, and left ventricular hypertrophy, with frequent ventricular extrasystoles. The chest roentgenogram revealed cardiomegaly-cardiothoracic ratio 14/28.5, a prominent left ventricle, and pulmonary congestion. The patient was placed on digitalis and Diuril, and his symptoms abated temporarily. He was seen again in 1959, with chronic left ventricular failure.

On admission the patient was acutely ill, with

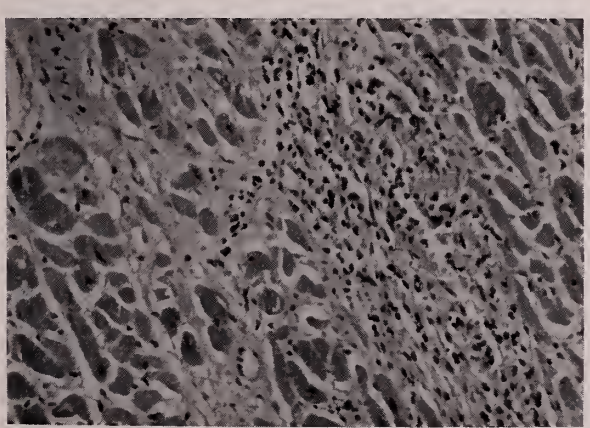

Fig. 4. Case 3.—Photomicragraph (x 69) af myacardium with intense leukacytic infiltratian. Nate absence af interstitial fibrasis.

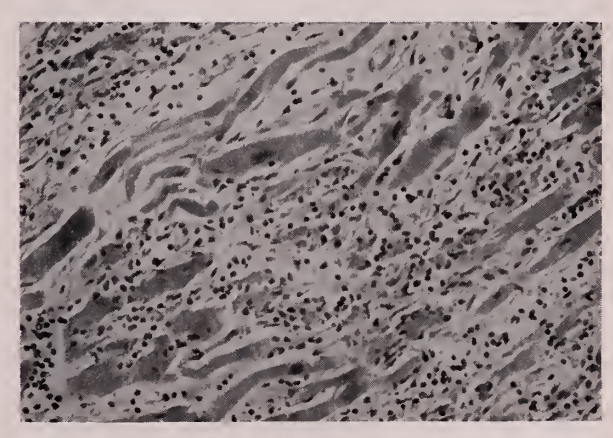

Fig. 5. Case 4.—Phatamicrograph (x 69) af myacardium illustrates intense leukacytic infiltration with muscle fiber necrasis and hypertraphy.

distended neck veins, atrial fibrillation at a rate of 140 to 160 per minute, and no obtainable blood pressure. The heart was definitely enlarged, sounds were distant and of poor quality, and a diastolic gallop was present. A grade 2/4 holosystolic murmur transmitted to the left axilla was heard at the apex. The lungs were filled with moist rales. Hepatomegaly and pedal edema were prominent.

Laboratory findings: Urinalysis revealed a specific gravity of 1.020, protein 2 plus, and was otherwise normal. Complete blood count revealed a hemoglobin of 14.2 gm. per 100 cc.; a leukocyte count of 9,400 per cu. mm., differentiated as polymorphonuclear neutrophils, 86 per cent; stab forms, 1 per cent; lymphocytes, 8 per cent; and monocytes, 5 per cent. Carbon dioxide combining power was 21.2 mEq. per liter. Prothrombin time revealed 38 per cent prothrombin activity. Serum bilirubin was 1.4 mg. per 100 cc. Serum cholesterol was 170 mg. per 100 cc. Serum glutamic oxaloacetic transaminase was 640 units. The electrocardiogram (Fig. 1) revealed atrial fibrillation, left axis deviation, and left bundle branch block.

The patient was placed in oxygen and given Cedilanid and vasopressors. Blood pressure rose to 115/80 mm. of mercury, and the pulse slowed to 110 per minute temporarily. Congestive heart failure persisted. Atrial fibrillation continued with frequent ventricular extrasystoles, and subsequently hypotension returned despite administration of Levophed. The SGOT rose to 1,260 units and serum glutamic pyruvic transaminase to 1,510 units. The patient's condition deteriorated, and death resulted from profound congestive heart failure four days after admission.

Autopsy: Bilateral pleural effusions and ascites were present, and the lungs, liver, and spleen were congested. The heart was hypertrophied, weighing 600 gm., and all chambers were dilated. Mural thrombi were found in both atrial appendages. The pericardium, endocardium, and coronary arteries were normal.

Microscopic examination of the myocardium (Fig. 6) revealed conspicuous interstitial fibrosis, with hypertrophy of the remaining myocardial cells. The muscle fibers were separated by bands of collagenous tissue. There was no infarction. Pathologic diagnosis was chronic isolated myocarditis.

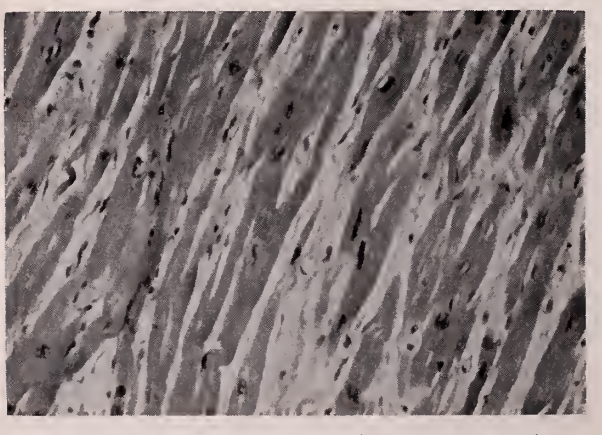

Fig. 6. *Case 5.*—Photomicrograph (x 143) of myocardium exemplifies diffuse interstitial fibrosis (confirmed by Masson trichrome connective tissue stain) and muscle fiber hypertrophy.

Case 6.—Clinical findings: A 48-year-old white woman who had previously been well was admitted to Hermann Hospital on Nov. 25, 1961. The patient had been awakened at 2 a.m. on the day of admission by nausea and vomiting and subsequently experienced two brief syncopal episodes. There was no history of angina, edema, hypertension, rheumatic heart disease, nor diabetes mellitus. The patient had recently had cystitis and had received penicillin and a sulfa drug.

At physical examination, the patient, in a convulsive seizure, was pale and cyanotic. Blood pressure was 100/60 mm. of mercury, and temperature, 98 F. Subsequently, the pulse was 50 per minute. The first heart sound was of poor quality and variable intensity, and a diastolic gallop was discernible. Occasionally, sounds of atrial contraction were audible. The lungs were filled with moist rales. The liver was tender and the edge palpable four fingers' breadth beneath the costal margin.

Laboratory data: Urinalysis revealed a specific gravity of 1.020, protein 1 plus, and was otherwise normal. The complete blood count revealed the following: hemoglobin 12.0 gm. per 100 cc.; leukocytes 15,680 per cu. mm., differentiated as polymorphonuclear leukocytes, 73 per cent; stab forms, 9 per cent; lymphocytes, 11 per cent; and monocytes, 7 per cent. The SGOT was 670 units and SGPT, 27 units. Blood cultures were negative. A chest roentgenogram displayed generalized cardiomegaly and pulmonary congestion. Electrocardiogram (Fig. 1) revealed complete heart block with an atrial rate of 96 and a ventricular rate of 32 per minute. Bed rest, oxygen, Isuprel, Aramine, hydrocortisone, Mercuhydrin, Crysticillin, and Demerol resulted in a ventricular rate of 70 beats per min.; however, despite therapy, the temperature rose to 102 F. rectally and congestive failure increased. The patient died of congestive heart failure 32 hours after admission.

Autopsy findings: The lungs and liver were congested. The hypertrophied heart weighed 512 gm. The endocardium and pericardium were smooth and translucent, the valves pliant, and the coronary arteries patent. Microscopically there was a predominantly mononuclear leukocytic interstitial cellular infiltrate and moderate interstitial fibrosis, with muscle fiber hypertrophy. The pathologic diagnosis was acute isolated myocarditis.

Discussion

Obviously each of the cases presented here was inconsistent with a recognized specific myocarditis—rheumatic, septic embolic, Chagas', trichinous, syphilitic, sarcoid, tuberculous, and so forth. In each case the etiology was obscure; however, it was impossible to exclude a virus even though no evidence for such was detected in any of the other organs at necropsy. Although coronary injection studies[13] were not done, failure to observe narrowing of the lumina of

the coronary vessels or thrombi on careful serial transection mitigates against the presence of significant ischemic heart disease. It is also inconceivable that coronary atherosclerosis could result in such a uniform or diffuse pathologic process as was observed in the many sections of myocardium taken from each heart. No discrete scars, localizing boundaries of injury, or repair were observed in the myocardium of any of these hearts. Absence of a primary systemic disease or infection in each case excludes the possibility of a secondary myocarditis. From what is known today, these cases are all best classified as examples of isolated myocarditis and illustrate the clinical and pathologic range of this heterogenous entity.

Isolated myocarditis occurs in all age groups. In these patients, age at onset of symptoms ranged from 21 to 78 years. No sex or race predominance was apparent; however, the seeming rarity of the disease practically precludes demonstration of significant statistical trends. The clinical course may be acute, subacute, or chronic. In five of these patients, the entire clinical course lasted from three and one-half hours to eight weeks, and in one patient, four years. Except for those who die suddenly and unexpectedly from an arrhythmic or embolic episode, the clinical course in all cases varies from that of an acute fulminant heart failure to chronic progressive congestive failure.

Symptoms of isolated myocarditis are predominantly those of myocardial insufficiency. In acute cases, weakness, easy fatigability, dyspnea on exertion, and palpitation are common. Occasionally, anginal pain, indistinguishable from that of ischemic heart disease, is present. Subsequently orthopnea, cough, abdominal pain, nausea, vomiting, edema, and ascites occur. In subacute and chronic cases, symptoms and signs of refractory congestive failure dominate the clinical picture. Syncope, paroxysmal arrhythmias, embolic episodes, and sudden death are not unusual.[9, 11, 12, 16] Physical manifestations include tachycardia, hypotension, frequent extrasystoles, pulsus alternans, a narrow pulse pressure, distant heart sounds of poor quality, and a diastolic gallop. Frequently there are accompanying signs of congestive heart failure and shock. In acute fulminant cases or terminally, patients may be febrile. Cardiomegaly does not always exist, but in subacute and chronic cases, the heart is usually enlarged and ejection and regurgitant murmurs may be present.

In acute and fulminant disease, moderate leukocytosis may be present. In subacute and chronic cases, urine and liver profiles are indicative of severe congestive heart failure. Roentgenographic studies may show pulmonary congestion, pulmonary edema, cardiomegaly, and pleural effusions. Left ventricular prominence may appear before generalized cardiomegaly. In a subacute or chronic case with a large dilated heart, the cardiac silhouette may resemble, and at times be indistinguishable from, that of a large pericardial effusion. Electrocardiographic abnormalities are nonspecific. Conduction defects (A-V block, left or right bundle-branch block, intraventricular conduction defects, accelerated A-V conduction) and arrhythmias (extrasystoles, tachycardia, and atrial fibrillation) are common. Left axis deviation and left ventricular hypertrophy are usual findings in subacute and chronic cases. T-wave abnormalities, Q-waves, and segmental shifts are frequently observed, and changes usually occur in all leads because of the diffuse nature of the myocardial process —injury or fibrosis. Such changes may be misinterpreted as acute myocardial infarction. Low voltage in all leads is occasionally observed. Transient conduction defects and paroxysmal arrhythmias characterize myocarditis, and hemodynamic studies reveal impaired myocardial function with a narrow pulse pressure, reduced and relatively fixed cardiac output, increased arteriovenous oxygen difference, and diastolic elevation of right atrial, right ventricular, and wedge pressures.[1] This pattern may be identical with that of other conditions associated with markedly impaired myocardial function.

The etiology of isolated myocarditis remains obscure. Virus infection,[14] metabolic deficiency states,[2] and allergic response,[3, 18] have all been incriminated, but each lacks proof. Pathologic findings are variable, ranging from an advanced interstitial inflammatory process with muscle fiber necrosis in acute cases to advanced interstitial fibrosis with muscle fiber hypertrophy in chronic cases. The interstitial infiltration may be composed predominantly of polymorphonuclear leukocytes or lymphocytes and occasionally eosinophilic leukocytes. Transitions from a severe inflammatory process to scar tissue are frequently observed. In subacute cases, the degree of interstitial inflam-

mation, fibrosis, muscle fiber degeneration and hypertrophy are variable. Interstitial fibrosis with hypertrophy of the remaining muscle fibers and minimal cellular infiltrate is to be expected in chronic cases. Some cases of advanced interstitial fibrosis probably result from a chronic smoldering myocarditis of undetermined etiology,[8, 17] while others undoubtedly represent the fibrotic stage of an earlier specific myocarditis. The cardiac dilatation, hypertrophy, and fibrosis observed at necropsy correlate well with the clinical duration of disease. Congestive changes in lungs, liver, spleen, and so forth, are secondary to heart failure. Terminal pulmonary infections are frequent in these patients. The pathologic changes in isolated myocarditis may resemble changes that result from known etiologic agents; however, in isolated myocarditis these changes are always limited to the myocardium. Isolated myocarditis may also occur in patients with ischemic or congenital disease, and under these circumstances recognition is extremely difficult.

In only one of the cases reported here was the correct diagnosis made prior to necropsy; in one it was suspected, and included in the differential diagnosis; and in five an erroneous clinical diagnosis of ischemic heart disease was made. The similarity of isolated myocarditis to ischemic disease is intensified when anginal pain, fever, leukocytosis, elevated SGOT, and electrocardiographic changes indicative of myocardial injury are evident.[5] This is understandable since myocardial damage is the common denominator in both. Isolated myocarditis should be strongly suspected in instances of sudden and unexpected death,[7, 11] and instances of chronic progressive heart failure of obscure etiology. Painless coronary atherosclerotic heart disease is usually suspected in chronic cases, and a diagnosis of ischemic heart disease made on a statistical basis. In suspected cases, embolic phenomena, paroxysmal arrhythmias, transient conduction defects, and diffuse electrocardiographic changes should intensify suspicion. This disease is probably more common than is generally appreciated, particularly the acute type.

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When isolated myocarditis occurs in the acute or fulminant form, ischemic heart disease is the major disease to be excluded. However, in subacute and chronic forms with cardiomegaly and progressive refractory congestive failure, several conditions are to be differentiated: (1) painless ischemic heart disease, (2) pulmonary hypertension, (3) rheumatic heart disease with "silent" mitral stenosis, (4) endocardial fibroelastosis, (5) constrictive pericarditis, (6) chronic pericardial effusion, (7) myocardial amyloidosis, (8) metastatic malignancy of the myocardium, and (9) familial, congenital, or idiopathic cardiomegaly. The modern cardiologist tends to classify most cases of chronic progressive myocardial insufficiency with cardiomegaly and an obscure etiology as "cardiomyopathy"[6] indicating noncoronary primary myocardial disease.

In some cases of isolated myocarditis, asymmetrical myocardial hypertrophy results in altered hemodynamics producing aortic ejection and mitral regurgitant murmurs. This can lead to the erroneous diagnosis of valvular heart disease with subsequent unnecessary surgical exploration.[8] In chronic cases with murmurs, right heart catheterization, even though it involves some risk, allows the clinician to exclude congenital defects, high cardiac output states caused by metabolic deficiencies, and pulmonary hypertension. Left heart catheterization is justifiable when aortic or mitral valve surgery is being considered. Patients with acute isolated myocarditis usually become too ill to study, and die within a few hours or days. Actually, subacute and chronic cases occasion most of the clinical confusion. In such instances, needle biopsy[15] of the myocardium is perhaps justifiable to obtain tissue for virus and enzyme studies, establish a diagnosis, and afford more knowledge of the natural history of the disease. Exact diagnosis is always desirable for therapeutic and prognostic reasons.

Summary

Six cases of isolated myocarditis have been presented and the disease discussed. The

clinical course may be acute, subacute, or chronic, and is characterized by symptoms of primary myocardial insufficiency terminating in progressive, refractory congestive heart failure or sudden and unexpected death from arrhythmias or thrombo-embolism. The etiology is obscure, and symptoms, signs, laboratory findings, hemodynamics, and pathology are nonspecific. The common characteristic is isolation of the primary pathologic process to the myocardium.

The therapeutic and prognostic implications of isolated myocarditis accentuate the importance of clinical recognition. Although treatment is symptomatic, bed rest, digitalis, steroids, anticoagulants, and antiarrhythmic drugs are indicated. Surgical exploration for valvular heart disease and constrictive pericarditis should be avoided.

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Ruptured Aortic Valve Surgically Reparable

Surgical implantation of an artificial aortic valve has been performed successfully in a woman whose valve was torn in an automobile accident, two Houston surgeons have reported.

Drs. Arthur C. Beall, Jr., and Albert L. Shirkey said the operation indicates that present techniques of total aortic valve replacement now allow successful surgical correction of this problem. In only one previous operation under these circumstances has the patient survived, they pointed out.

The injury to the Houston woman led to aortic valve regurgitation and congestive heart failure. Injuries are a rare cause of aortic valve regurgitation, the authors said, but with improvements in overall care of patients sustaining nonpenetrating chest injuries, this problem probably will be recognized with increasing frequency.

Physiological improvement of this patient following implantation of a Starr-Edwards artificial valve was "truly impressive," the surgeons commented.

Topical Cooling

An Adjunct to Intracranial Surgery

CASE REPORTS

COOLING OF the surface of the brain with topical irrigation of cold normal saline solution, either alone or in combination with total body hypothermia, has been suggested by several authors as an agent in reducing the size and vascularity of the brain. Most patients in whom this method has been used have undergone either extirpation of gliomas or temporal lobectomy for temporal lobe seizures. The authors claim no originality for this technique but have adopted the general principles and used them in two cases; one, a metastatic adenocarcinoma in the frontoparietal area and the other, an extensive arteriovenous malformation of the left parietal area convexity. To date, the possibilities of this method with particular reference to cerebral vascular surgery have not been investigated nor reported.

Method

Gravity-feed sterile normal saline, previously cooled in ice, is attached to run through a copper coil which is immersed in an ice-filled container. The outflow from this coil leads directly to the operative field where, through one or two orifices, the cooling solution is allowed to flow directly upon the cerebral cortical surface. The average temperature of the cooling solution is 5 to 7 C.

Case Reports

Case 1.—A 67-year-old white male was apparently well until September, 1962, when he had a nonlocalized major seizure. Since that time he had four major seizures and developed a slight central left facial weakness as well as slight weakness of the left arm and left leg. The patient had also shown some loss of memory for recent events. Preoperative neurological examination showed a profound motor weakness involving both the arm and leg on the left side with little sensory impairment. The patient had no papilledema and no abnormalities of the cranial nerves other than a mild left central facial weakness. The patient's speech and general activity were slow but he was oriented and able to carry on conversation. Carotid arteriography showed a shift of the anterior cerebral artery to the left in a fashion consistent with a right posterior frontoparietal lesion.

On Jan. 10, 1963, a right frontoparietal craniotomy was performed. On opening the dura the brain was tight, and grayish tumor material was seen on the cortical surface in the posterior-superior frontal region. Frozen section from the tumor was consistent with metastatic adenocarcinoma. At this juncture topical cooling was instituted for a one-hour period while resection of the tumor and the right frontal lobe was performed. At the beginning of topical cooling (Fig. 1), brain temperature monitored by thermocouple implant on the cortical surface was 94 F. and at the end of topical cooling the surface temperature was 80 F. Temperature inside the right ventricle after cooling registered 85 F. During the one-hour period of topical cooling, body temperature dropped from 96 F. to 94 F. The tumor mass and right frontal lobe were resected with no difficulty. The topical cooling produced a pale avascular brain (Fig. 2) (reduced in size) which made resection exceedingly easy. There was minimal blood loss from this procedure. The patient had an uneventful postoperative course and on discharge had regained a fair amount of use of his left upper and lower extremities.

Case 2.—A 36-year-old white female, for four and one-half years, had focal seizures involving the right arm, leg, face, and hand. These were only moderately controlled with anticonvulsants. In the year prior to admission she had begun to experience a definite motor weakness of the right arm and right leg as well as a moderate expressive aphasia. Neurological examination revealed a moderate weakness

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Excision of two intracranial lesions carried out with the aid of cortical topical cooling are reported. Reduction in cerebral vascularity and bulk made the excision much easier.

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of the right arm and right hand, a right facial weakness, moderate weakness of the right leg, and a moderate expressive aphasia. Comprehension seemed unaffected. The patient was right-handed. Carotid arteriography revealed a large arteriovenous malformation over the left parietal convexity with an extremely dilated left anterior cerebral artery and its branches feeding the malformation (Fig. 3). There was also secondary feeding from branches of the left middle cerebral artery. Right carotid arteriograms revealed filling of the left anterior cerebral artery but the malformation did not seem to feed from other vessels over the right hemisphere. On June 3, 1963, a left frontoparietal craniotomy was performed under general hypothermia. Upon opening the dura, the use of 15 minutes of topical cooling reduced the vascularity of the lesion. We were then able to retract the left frontal lobe exposing the left anterior cerebral artery clearly (Fig. 4). The venous circulation was exceedingly diminished by this cooling which made securing of the very large veins leading into the sagittal sinus much easier (Fig. 5). During the 15 minutes of topical cooling, body temperature dropped from 93 F. to 91 F. With the reduction of vascularity there was also a general reduction in the bulk of the brain. This extensive lesion was resected without undue difficulty. Postoperatively, the patient was able to walk and the preoperative aphasia was essentially unchanged. The weakness of the right arm was considerably worse, however. She has since been discharged ambulatory to her home.

The simplicity of topical cooling and the avascular field which it readily affords make

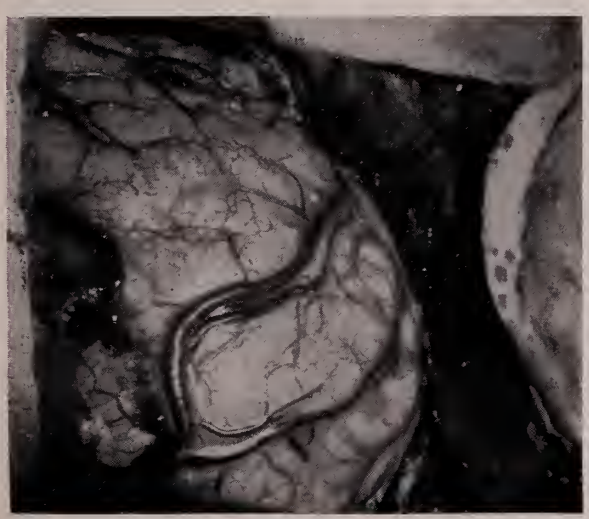

Fig. 1. Operative field prior to cooling. Tumor in the left margin following biopsy.

Fig. 2. Operative field undergoing topical cooling. Tumor and right frontal lobe partially resected.

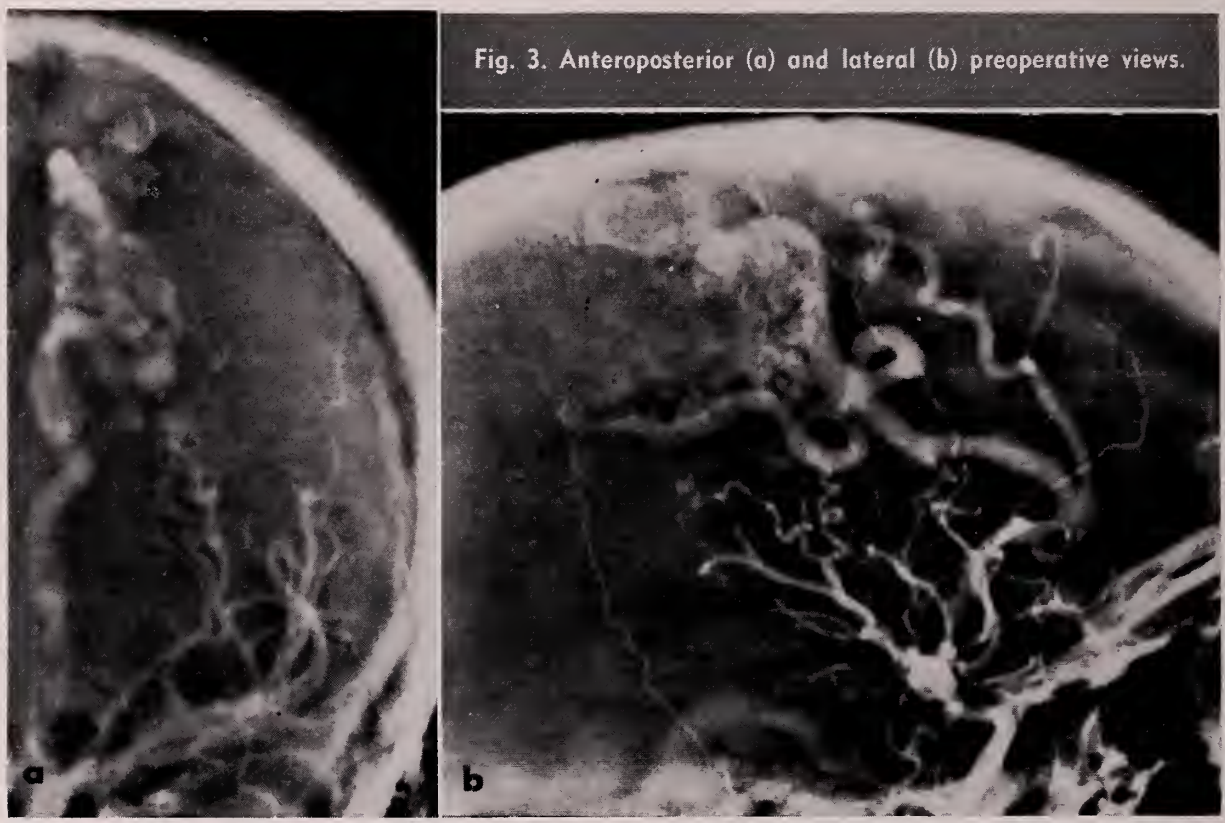

Fig. 3. Anteroposterior (a) and lateral (b) preoperative views.

the procedure an attractive aid to consider, particularly in cerebrovascular surgery. Since only the organ being operated on is cooled and not the body in general, it would seem that the dangers of sludge formation, increased viscosity and possible resultant thromboses in the intracranial vessels can be more readily avoided. The degree of protection of the human cerebral cortex to various periods of temporary vascular occlusion during this type of cooling needs still to be worked out. There is some evidence to indicate that there is a time limit to which topical cooling can be applied without protein breakdown. Whether this blood-brain

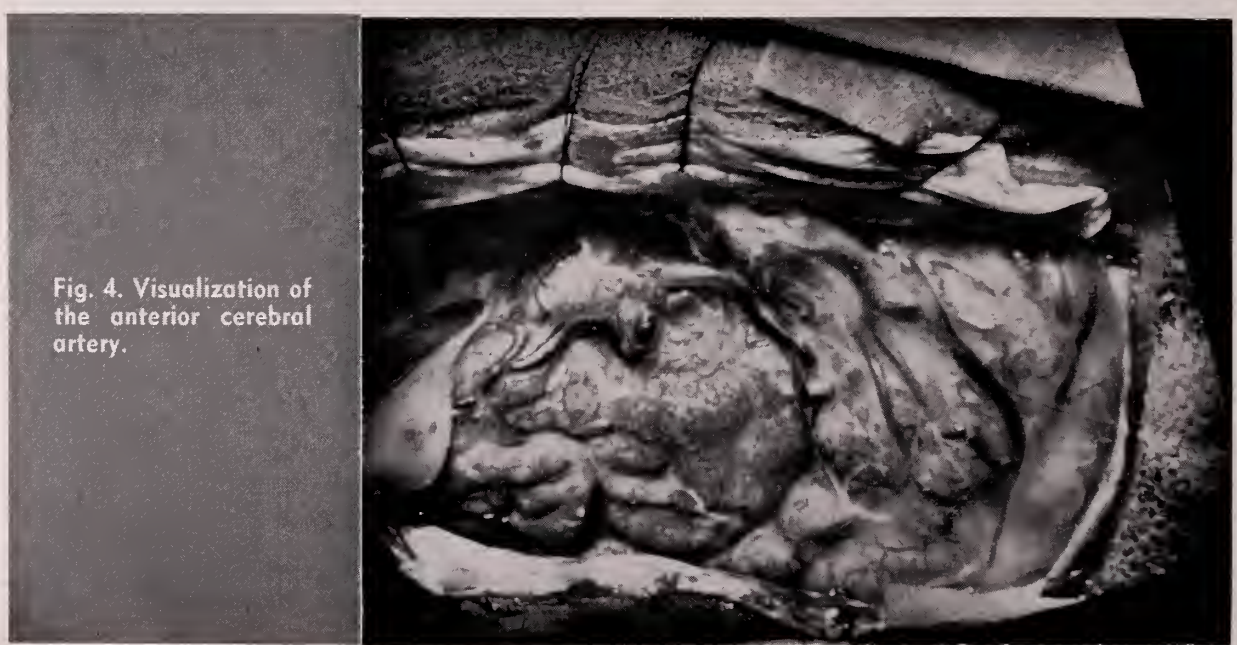

Fig. 4. Visualization of the anterior cerebral artery.

Fig. 5. Additional reduction in venous filling with anterior cerebral clipping and additional cooling.

barrier breakdown results in any demonstrable organic difficulties has not been shown to date. We have been impressed with the ease, simplicity, and results of this method, and are continuing to evaluate it both clinically and in the laboratory.

Summary

Two cases of intracranial space-occupying lesions are reported, excision of which was carried out with the aid of cortical topical cooling. Reduction in both cerebral vascularity and bulk was observed, and excision of both lesions was facilitated by this method.

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Ocular Complications From Chloroquine Therapy

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CASE REPORTS

CHLOROQUINE (ARALEN) was developed during World War II as an antimalarial drug. Compared to the antimalarial drugs previously in use it is of relatively low toxicity and high antimalarial activity.[5] Other antimalarials developed during this time are quinacrine (Atabrine) and hydroxychloroquine (Plaquenil).

Since these drugs were first marketed, other valuable therapeutic properties have been discovered—antiactinic, antiarthritic, and amebicidal. Dame[2] observed a high incidence of paracentral scotomas in a controlled group taking quinacrine. Quinacrine has been discontinued to a large extent because it discolors the skin and has caused aplastic anemia. Chloroquine is widely used in disseminated lupus erythematosus, discoid lupus erythematosus, polymorphic light sensitivity eruptions, sarcoid, asthma, skin tumors, rheumatoid arthritis, and other collagen diseases. Treatment of these chronic dermatologic and rheumatoid diseases has greatly increased the use of chloroquine beyond that prescribed for malarial suppression.

Corneal changes were noted soon after the introduction of these drugs and have been well documented in the literature.[1,6,7] Hobbs[9] stated that corneal changes occur in approximately one-third of patients on prolonged chloroquine therapy. These corneal changes have been described as edema and deposition of whitish to yellowish fine discrete granules confined to the epithelium and Bowman's membrane. These are similar to corneal changes previously reported to occur from quinacrine.[15] Hobbs and Calnan[7] have differentiated between the acute onset of corneal edema and later development of corneal infiltration, which they postulated to represent a loading phenomena. Both the edema and depositions are reversible and disappear after cessation of the drug.

Of much more serious concern are the retinal changes which, once established, do not regress. Goldman and Preston[4] first reported a serious problem of permanent visual disturbance associated with chloroquine therapy in 1957. Hobbs and associates[8] published a more detailed report on three cases with visual loss associated with chloroquine therapy in 1958. Ellsworth and Zeller reported three cases with retinopathy in 1961,[3] and since that time several articles have appeared in the ophthalmic literature with case reports totaling 27.

It is difficult to assess the incidence of retinopathy in patients on chloroquine therapy. Hobbs[9] reported five cases in 170 patients on chloroquine therapy. This seems unusually high when compared to the total number of cases reported and the number of patients taking chloroquine. Mills and Moyer[11] reported that in a series of over 1,500 patients taking an antimalarial compound for periods varying from a few months to five years, all drug reactions were completely reversible following discontinuation of medication. Roth[14] states that chloroquine has been given in hundreds of millions of doses and used for long-term administration in hundreds of thousands of patients. The medical literature contains approximately 1,500 published reports on chloroquine treatment of collagen diseases alone. Although the exact incidence of retinopathy

Chloroquine (Aralen) is often prescribed for dermatologic and collagen diseases, and treatment may be lengthy. This drug can cause changes in both the retina and cornea. Retinal changes are permanent, but corneal changes are reversible. Periodic eye examinations are recommended for patients on chloroquine therapy.

isn't known, some things seem evident from the reported cases. Retinopathy has occurred only in patients undergoing long-term treatment. All of the patients thus far reported have had at least one year of therapy and a minimum of 200 gm. of the drug prior to the onset of retinopathy. Most have had considerably more than this minimum, with the average being over 500 gm. of chloroquine.

There are several typical findings in the reported cases. The retinopathy is bilateral and nearly symmetrical in the two eyes. A constant early feature is a pericentral scotoma with the central acuity remaining normal, or almost normal, despite a residual central field of as little as 1°. As the retinopathy progresses the pericentral scotoma spreads further centrally and peripherally and there is constriction of the peripheral field.[12] The earliest change occurs in the macula where there is an increased granularity or patchy depigmentation. As this is well established, a central pigmented area is seen surrounded by a clear zone and then another concentric ring of pigment. This has been described by the terms "doughnut" or "bull's-eye" macula, and this can usually be seen in fundus photographs.[16] There appears to be no absolute correlation between the amount of pigment seen and the level of visual reduction. Narrowing of the retinal arteries and arterioles has been reported in some of the more advanced cases. Another finding of the advanced cases is pigment disturbance in the periphery resembling retinitis pigmentosa.

Okun and associates[12] reported on eight cases of chloroquine retinopathy in which they did studies on color charts, dark adaptation, and electroretinogram (ERG) tracings. They report that dark adaptation thresholds are affected either very slightly or not at all in the early cases and only moderately in the advanced ones. The ERG shows only slight reduction in the early cases of retinopathy. In advanced cases photopic responses are not detectable, but some scotopic function remains. This is in contrast to retinitis pigmentosa, where dark adaptation thresholds are much more elevated and the ERG is correspondingly more reduced. These authors report that the results of color vision testing are of particular interest because of high sensitivity of the Hardy-Rand-Rittler (H-R-R) pseudo-isochromatic plates. They believe that the sensitivity of the pseudo-isochromatic plates for detecting this retinopathy is due to the presence of the central and pericentral scotomas which occur as an early sign of this abnormality and not to an actual color vision defect. A color vision deficit does occur later. The simplicity of this test with color plates makes it a very useful screening device for incipient chloroquine retinopathy.

Subjectively, patients notice reading difficulties, with letters or words missing, photophobia, and or halos, blurred vision from interference with accommodation, misty or foggy vision, peripheral field defects, and floaters and flashes of light.

The mechanism of the toxic action of chloroquine on the retina is not understood. Hobbs and others[8] tried to produce a retinopathy by acute intravenous administration of the drug in rabbits, but were unable to do so. The absence of a specific effect of chloroquine on the rod and pigment layer of the retina in rabbits led the researchers to suggest that a vascular mechanism was responsible for the human retinopathy. Okun was unable to produce a retinopathy in rats by long-term oral administration of chloroquine, though the animals developed keratopathies and cataracts. In these animals both the ERG and histologic studies of the retina failed to show any evidence of retinal damage, which might be expected if chloroquine had a specific effect on either the pigment epithelium or the external layers of the retina.

Though the exact mechanism is not understood, the visual loss from the retinopathy is permanent. Improvement does not occur either objectively or subjectively after the

drug is stopped. In several reported cases there has been further loss of vision after cessation of chloroquine. In one reported case the vision was 20/40 at the time chloroquine was discontinued, and this decreased to 15/400 over the next few months, with the pericentral scotomas becoming central.[12] A few cases have been reported in which scotomas were present without fundus changes after a few months of chloroquine therapy.[13,15]

Though it is unlikely that the ophthalmologist will prescribe chloroquine, he will see patients who are taking this medication. It would be desirable for an ophthalmologic examination, including visual field, to be done on patients at the beginning of therapy with chloroquine in order to establish a base line for future assessment of the patient's vision. Periodic examinations should be done on all those on long-term chloroquine therapy, and the drug should be stopped immediately if scotomas, or any evidences of retinopathy, occur.

Case Report

This 47-year-old white woman was seen Sept. 7, 1961. She complained of some difficulty in reading and of multiple small floaters noticeable in each eye (O. U.). The uncorrected vision in each eye was 20/20-1, correcting to 20/20+ with —.25 + .50 x 180. The patient wore glasses (+1.00 O. U.) for reading and the near point of accommodation was 30 cm. Intraocular pressure was 17 O. U. and the visual field was normal. The fundi appeared normal and no vitreous floaters could be seen objectively. A stronger prescription was given for reading, and she was told to return if the spots persisted.

She was next seen on Nov. 29, 1961, complaining of more difficulty in reading and of blind spots in her vision. She had noticed at football games that she lost the ball in the air; when reading, part of the words were missing; and when looking at people she could not see their eyes. Questioning revealed that she had been taking two Aralen tablets daily for about five years to control dermatitis caused by photosensitivity. Two years earlier a second dermatologist had confirmed the diagnosis and doubled her dose of Aralen. Following this her vision blurred; she cut back to her previous dose and her vision cleared. Since taking Aralen there had been depigmentation of some of her hair. The patient smoked about 15 cigarettes daily. Vision of each eye without correction was 20/25, correctable to 20/20—.

Corneae showed some haze and fine deposits in the epithelium with fine punctate staining of the inferior one-third with fluorescein. The fundi showed absence of the foveal reflex of an increased redness and granular appearance of the maculae with a slight increase in pigmentation just inferior to the maculae. Visual fields showed a horseshoe-shaped pericentral scotoma O. U. This was an absolute scotoma and the sides were very steep and clear-cut to any size target from 1 to 10 mm.

Chloroquine was stopped and the patient was put on ammonium chloride, 8 gm. daily, because this hastens excretion of chloroquine. Carbon dioxide inhalations were used for several weeks for vasodilatation. The use of tobacco was stopped, but not the use of alcohol. The patient was also given a corticosteroid and put on Arlidin, 6 mg. three times a day, for vasodilatation.

There was no appreciable change in the vision or visual fields during the ensuing months.

This patient had been an avid and rapid reader, and the development of the pericentral scotomas slowed her reading greatly and frustrated her. Though the scotomas have not become any smaller, she has been able to regain part of her previous reading skill.

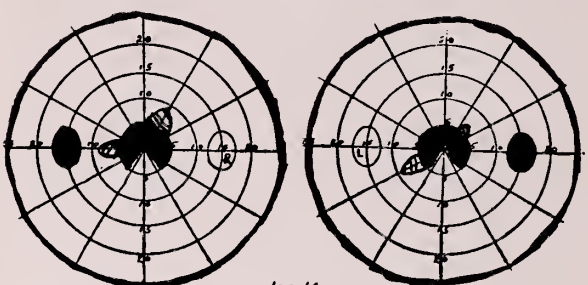

Fig. 1. Visual field, Nov. 29, 1961. It is possible that the scotoma did not come quite as close to fixation as is shown by this field. The examination was done following other tests; the patient was fatigued, and fixation may not have been as good as it was when other fields were taken.

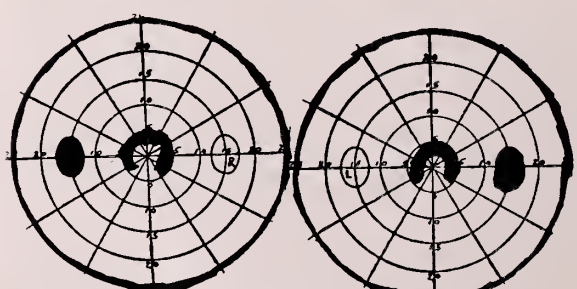

Fig. 2. Visual field, Dec. 6, 1961.

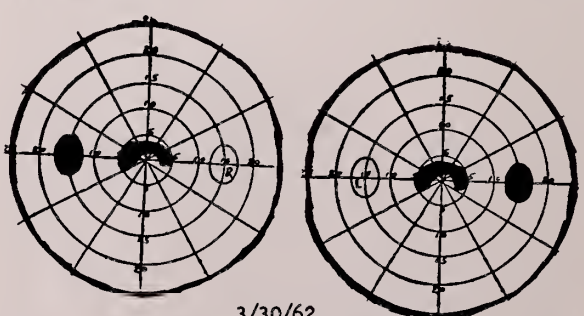

Fig. 3. Visual field, March 30, 1962.

Summary

Chloroquine was developed 20 years ago as an antimalarial but has since gained wide use in a variety of collagen and dermatologic diseases. Approximately a third of those on prolonged therapy will have some blur of vision from interference with accommodation, or keratopathy. These changes are reversible. A small percentage of patients on prolonged therapy develop a retinopathy which is not reversible. The number of cases reported has increased greatly in the last year. It is felt that those on prolonged chloroquine therapy should have periodic eye examinations, including visual fields, and should be told to report any suspicious symptoms immediately. The H-R-R pseudo-isochromatic plates are a sensitive test for these early scotomas, and this test could be employed regularly by the physician prescribing the chloroquine.

Dr. James H. Scruggs, a Waco ophthalmologist, presented this paper at the 1963 meeting of the Texas Ophthalmological Association in Dallas.

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Physicians' Smoking Habits Changing

More than 60,000 physicians have made it clear in a nationwide survey that U. S. medical opinion is nearly unanimous on one subject: cigarette smoking is dangerous.

Many physicians report they have changed their own smoking habits by stopping, cutting down, or switching away from cigarettes.

The survey was conducted by the medical journal, *Modern Medicine*. A questionnaire was sent to the 192,000 physicians in active practice, and was returned by 60,202—nearly a third.

Commenting on their own habits, 80 per cent said they have smoked at some time in their lives. At present, however, 52 per cent do *not* smoke, and many who continue to smoke use cigars and pipes. Only 22.5 per cent are now smoking cigarettes, and 89 per cent of these consider the habit a health hazard. Significant, too, is the fact that thoracic surgeons and pathologists, specialists most likely to get frequent, good looks at the lungs, rank at the very bottom of cigarette smokers.

Diversities Of Adrenal Cortical Hyperfunction

V. WILLIAM COLE, M.D.; BYONG S. MIN, M.D.; RYOSO KAWATE, M.D.; RAYMOND G. ROSE, M.D.; AND CLIFTON D. HOWE, M.D.

CASE REPORTS

Cases representing three common varieties of adrenal cortical abnormalities are presented and discussed. Several diagnostic tests are evaluated, and the importance of early and correct diagnosis is emphasized.

THREE PATIENTS with adrenal cortical hyperfunction of diverse etiologies, accompanied by strikingly increased urinary 17-ketosteroid excretion, are reported. The response to adrenocortical stimulative and suppressive agents employed in diagnosis is illustrated for each patient. The patient with adrenocortical carcinoma was treated with o,p'DDD.* In the patient with congenital adrenal hyperplasia, the adrenal status was left unaltered; in the patient with acquired bilateral adrenal hyperplasia, subtotal adrenalectomy successfully ameliorated the signs and symptoms of Cushing's syndrome.

During the past 12 months, three patients displaying adrenocortical hyperfunction and striking elevation of urinary 17-ketosteroid excretion were admitted to the University of Texas M. D. Anderson Hospital and Tumor Institute. Although many types of adrenal cortical hyperfunction are known, the three case studies presented herein illustrate the most common varieties; for completeness, additional categories are outlined in Table 1. The etiologies of the adrenal cortical hyperfunctions were (1) adrenal cortical carcinoma, (2) congenital adrenal hyperplasia, and (3) acquired bilateral adrenal hyperplasia. Diagnostic studies and treatment employed for each patient are discussed.

Tests of adrenal cortical function performed on these patients included a standard ACTH test (administration of 25 units of ACTH via intravenous infusion extending over an eight-hour period, 8 a.m. to 4 p.m.), and the dexamethasone suppression test (.5 mg. every six hours and 2 mg. orally every six hours for 48 hours). Pituitary ACTH reserve was studied by the use of intravenous SU 4885,† 30 mg. per kg. of body weight administered over a four-hour period, 8 a.m. to 12 noon.[10] Ancillary glucose tolerance tests, insulin tolerance tests, and glucose-

TABLE 1.—*Classification of Diseases Associated with Increased 17-Ketosteroid Excretion.*

Adrenal
Acquired adrenal hyperplasia.
Congenital adrenal hyperplasia.
Adrenocortical carcinoma and adenoma.
Idiopathic hirsutism.
Feminizing hyperadrenocorticalism.
Ovary
Arrhenoblastoma.
Adrenal-like tumor.
Stein-Leventhal syndrome.
Testes
Leydig's cell tumor.
Pituitary or Hypothalamus
Basophilic adenoma.
Sexual precocity of constitutional type.
Malignant Conditions Associated with Increased ACTH-like Hormone
Oat cell carcinoma of lung.
Pancreatic carcinoma.
Parotid gland carcinoma.

*2,2-bis (2-chlorophenyl, 4-chlorophenyl) 1, 1-dichloroethane.

†Metopirone (2-methyl-1,2 bis-(3-pyridyl)-1-propanone).

insulin tolerance tests, which were administered, are listed in Table 2. The total urinary 17-ketosteroids were measured in triplicate by a modification of Drekter's method[6] in which 10 ml. of urine were hydrolyzed with concentrated hydrochloric acid, extracted with ether, and washed with sodium hydroxide. Chromogen removal was accomplished with sodium hydroxide pellets. The Zimmerman color reaction was employed using 5 normal potassium hydroxide and 2 per cent metadinitrobenzene. Twenty, 40, 60, and 80 gammas of dehydroepiandrosterone were utilized for standard reference. Spectroscopy at a wave length of 520 mμ was recorded on a Coleman Junior Spectrophotometer. The urinary 17-hydroxycorticosteroids (Porter-Silber chromogens) were measured in duplicate using the Glenn-Nelson method.[9] In the case of congenital adrenal hyperplasia, Bongiovanni's method of determining pregnanetriol was utilized.[2]

Case Reports

Case 1.—This 54-year-old woman was referred to M. D. Anderson Hospital in December, 1961, for management of an adrenal cortical carcinoma. Included in the patient's history were intermittent headaches which had occurred over a period of two years prior to admission. She had a painless mass in the right upper quadrant, and mild hypertension, both of one year's duration. An exploratory laparotomy, at another hospital, had revealed a large, firm, retroperitoneal mass associated with the right adrenal gland. Operative removal had been attempted, but because the tumor had invaded the inferior vena cava, complete removal was impossible. The postoperative course was complicated by uremia associated with hypokalemic alkalosis and hypotension. Following stabilization of these conditions, radiotherapy of 4,000 rads had been given to the tumor area. Subsequent to irradiation, progressive hirsutism was noted.

At admission to this institution, the patient's blood pressure was 154/102 mm. of mercury; pulse, 84; and respiration, 12. General body build and muscle development were good (Fig. 1). Temporal recession was noted as was hirsutism of the nasolabial surface. Heart, lungs, and breasts were normal. A large, firm, nontender mass was easily palpable in the right upper quadrant, extending down well into the right lower quadrant. The pelvic examination revealed only slight clitoral enlargement.

The results of the urinary steroid excretion studies are illustrated in Fig. 2 and Table 3.

TABLE 2.—*Carbohydrate Studies.*

	Case 1 Carcinoma		Case 2 Congenital hyperplasia		Case 3 Acquired hyperplasia (Intravenous Test)	
	Time	Blood Glucose mg./100 ml.	Time	Blood Glucose mg./100 ml.	Time	Blood Glucose mg./100 ml.
Oral Glucose Tolerance Test	Fasting	82	Fasting	82	Fasting	95
	1 hr.	184	1 hr.	121	2 min.	232
	2 hrs.	214	2 hrs.	131	5 min.	206
	3 hrs.	228	3 hrs.	100	10 min.	191
	4 hrs.	184	4 hrs.	64	25 min.	169
	5 hrs.	64	5 hrs.	81	40 min.	147
Intravenous Insulin Tolerance Test	Fasting	80	Fasting	86	Fasting	99
	10 min.	61	10 min.	65	10 min.	90
	20 min.	53	20 min.	55	20 min.	69
	30 min.	58	30 min.	53	30 min.	59
	45 min.	71	45 min.	65	45 min.	68
	60 min.	79	60 min.	75	60 min.	74
	90 min.		90 min.	90	90 min.	109
	120 min.		120 min.	109	120 min.	129
Glucose-Insulin Tolerance Test	Fasting	78			Fasting	94
	15 min.	108			15 min.	96
	30 min.	122			30 min.	104
	45 min.	134			45 min.	119
	60 min.	150			60 min.	139
	75 min.	182			75 min.	182
	120 min.	212			120 min.	234
	150 min.	246			150 min.	230
	180 min.	250			180 min.	193

TABLE 3.—*Adrenal Function Studies in Hypersecretory States in Mg./24 Hours.*

Day	Carcinoma		Congenital Adrenal Hyperplasia		Acquired Adrenal Hyperplasia	
	17-KS	17-OHCS	17-KS	17-OHCS	17-KS	17-OHCS
Resting State						
1	45.7	17.6	71.1	1.4	45.9	24.4
2	76.4	18.2	70.1	1.6	21.3	20.4
3	62.7	16.5	70.0	2.5	25.8	14.7
ACTH						
1	74.5	13.8	43.6	0.29	62.8	73.6
2	67.9	17.2	113.4	2.3	69.6	90.0
3	52.8	14.8	54.3	1.9	67.4	46.1
Dexamethasone Suppression*						
1	79.7	12.2	14.2	0.0	29.7	24.3
2	64.4	13.6	5.2	0.2	18.9	17.3
Dexamethasone Suppression**						
1	92.7	12.8	6.1	0.4	12.2	9.5
2	93.6	11.2	6.0	0.8		
SU 4885	62.1		40.6	2.9	16.3	17.4
Day after SU 4885	55.8	12.7	67.8	2.4	39.1	33.8

*Dexamethasone suppression, 0.5 mg. per 6 hours.

**Dexamethasone suppression, 2.0 mg. per 6 hours.

Abbreviations: 17-KS — 17-ketosteroids, normal level at this institution: 5-20 mg./24 hrs. 17-OHCS — 17-hydroxycorticosteroids (Porter-Silber chromogens), normal level at this institution: 2-8 mg./24 hrs.

The mean resting total urinary 17-ketosteroid level was 60 mg. per 24 hours; for this laboratory, the normal female's level ranges from 5 to 15 mg. per 24 hours. No significant response was noted following infusion with SU 4885. Likewise, no appreciable alteration in steroid excretion occurred following administration of ACTH during a three-day period. Dexamethasone failed to suppress steroid excretion. An experimental adrenocortical inhibitor, o,p'DDD,[1] was administered in varying dosages (Fig. 2). Following the use of this agent, a significant reduction in excretion of urinary 17-ketosteroid and 17-hydroxycorticosteroid was noted. However, despite the fall in steroid production, the tumor gradually enlarged. ACTH studies conducted while the patient was receiving o,p'DDD failed to induce any change in steroid excretion. Despite the administration of large amounts of o,p'DDD, an increase in urinary steroid metabolites occurred near the end of February, 1962. The last two weeks of the patient's life were characterized by vomiting, hyponatremia, decreased urinary output, and cachexia. Postmortem examination revealed the presence of a large necrotic retroperitoneal adrenal carcinoma which occupied the entire right side of the abdomen and involved the liver, kidney, lungs, and lumbar vertebrae.

Fig. 1. Case 1, adrenal cortical carcinoma. On admission, this patient had hirsutism of the upper lip. Tracings over the abdomen outline the large adrenal cortical tumor.

Despite the elevated steroid excretion found in this patient, few of the clinical manifestations of Cushing's syndrome were present. The minimal increase in blood pressure and the late appearance of hirsutism with slight clitoral enlargement were clinical manifestations of the disease evident only during the last 12 months of life. Abnormalities of body fluids, electrolytes, and protein wasting were not observed until the disease was far advanced. Characteristically, adrenocortical carcinomatous tissue produces excessive quantities of steroids which are reflected in the urinary excretory metabolites.

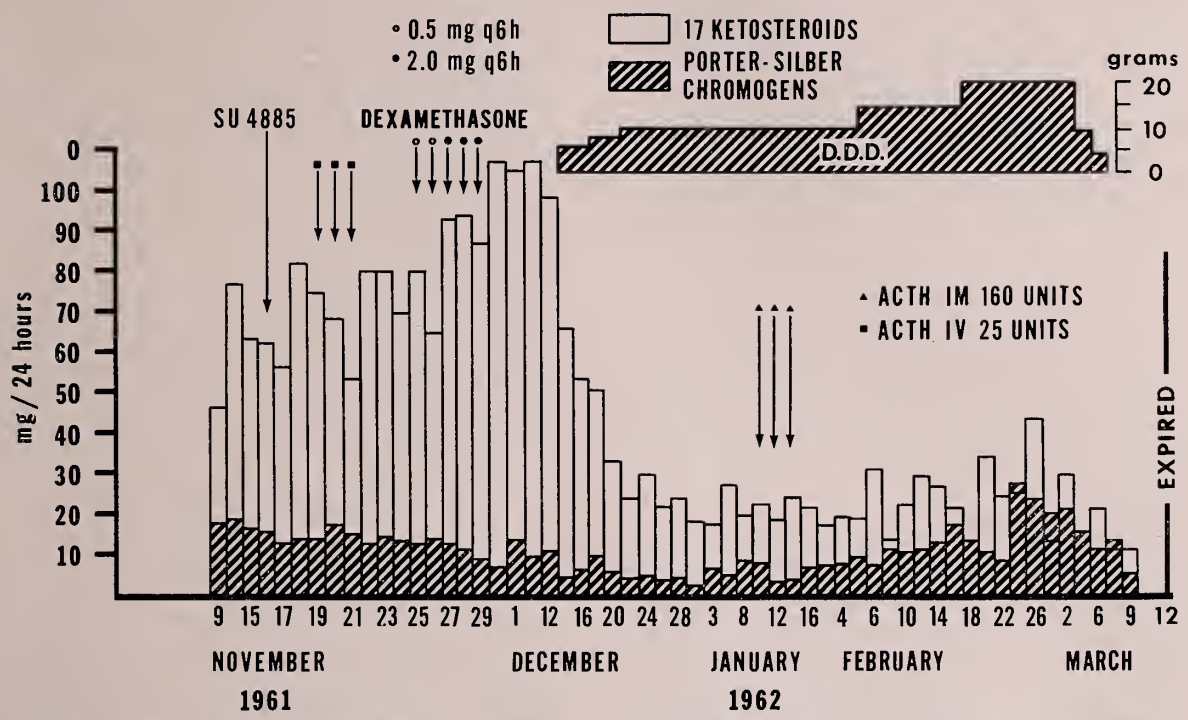


Fig. 2. Studies of urinary steroid excretion in case 1, adrenal cortical carcinoma. Note the relative unresponsiveness of adrenocortical excretion following testing with either stimulative or suppressive agents, the striking inhibitory effect of o,p'DDD on steroidogenesis, and the preterminal elevation of steroid synthesis despite the administration of increased amounts of o,p'DDD.

The carcinomatous tissue is autonomous and, as this case demonstrates, fails to respond to either exogenous ACTH or endogenously stimulated ACTH after the administration of SU 4885. Further substantiation of this unregulated secretion of adrenal cortical steroids is shown by the failure of relatively large amounts of dexamethasone, which are usually effective in diminishing endogenous ACTH release and, thereby, indirectly lowering adrenocortical function, to suppress secretion of adrenal steroids.[13] In contrast, by using a potent adrenocortical blocking agent, steroid production was rapidly diminished and was maintained at low levels for two or three months. Nevertheless, the progressive nature of the disease was not significantly altered by this blocking agent, for despite administration of relatively large amounts of this substance, the patient ultimately died.

Case 2.—A 38-year-old service station attendant was admitted in December, 1962, with a diagnosis of pseudohermaphroditism and ovarian carcinoma. At birth, the patient had shown ambiguous external genitalia and was reared as a male. Until the age of 14, growth and development were normal; thereafter, his height remained below average. Throughout his adult life, his occupation had been of a nature which required only moderate physical exertion. Attempts at matrimonial adjustment failed in less than a year on two occasions. However, the patient maintained that his sexual relationship with each partner had been satisfactory and had included weekly intercourse accompanied by ejaculation. Two months prior to admission, the patient developed intermittent pain of the left lower quadrant accompanied by weakness, vomiting, darkening of the skin, and dysuria. One month prior to admission, exploratory laparotomy performed at another institution disclosed a rudimentary uterus, tubes, and ovaries. At biopsy, the diagnosis of possible ovarian carcinoma was made, for which the patient was transferred to this institution.

At admission, the patient's blood pressure was 130/90 mm. of mercury; height, 4 ft. 9 in. (142.5 cm.); and weight, 92 pounds (41.8 kg.). The body proportions were normal (Fig. 3). The patient appeared masculine and showed temporal recession. He had abundant axillary and pubic hair with a male escutcheon. Muscular development and strength were excellent. The phallus measured 8.5 cm. in length and 4 cm. in diameter. A hypospadiac cleft extended from the glans to the base of the phallus, below which was a urogenital sinus. No testicles were palpable. Laboratory data on this patient is summarized in Tables 2 and 3 and Fig. 4. While on a sodium intake of 170 mg. per day, the patient exhibited normal salt conservation which was documented by appropriate balance studies. Chromosome analysis, by peripheral blood culture technique, demonstrated a normal female XX karyotype.

Fig. 4 graphically demonstrates the results of the pituitary-adrenal function studies. On the second day

of intravenous ACTH infusion, 17-ketosteroid excretion increased from 45 to 113 mg.; the administration of 2 mg. of dexamethasone per day resulted in a striking fall in 17-ketosteroid excretion. Following ancillary function studies with SU 4885, a fall in 17-ketosteroid secretion was recorded. Low urinary 17-hydroxycorticosteroid excretion was not altered either by ACTH or by SU 4885. Dexamethasone administration caused a reduction of these metabolites to undetectable levels. An elevated level of urinary pregnanetriol of 4.4 mg. per 24 hours was noteworthy.

At exploratory laparotomy, an infantile uterus, ovaries, and tubes associated with chronic pelvic inflammatory disease were disclosed. Prominent bilateral adrenal enlargement was noted by palpation. Total hysterectomy, salpingectomy, and oophorectomy were performed. Pathologically, no carcinoma was found. Throughout the operative period, hormonal replacement therapy was used without complications.

The diagnosis of female pseudohermaphroditism was verified by the presence of ovaries, uterus, and tubes, and of ambiguous external genitalia, a female chromosome pattern, bilateral adrenal hyperplasia, greatly increased urinary 17-ketosteroids with low 17-hydroxycorticosteroids, and increased urinary pregnanetriol levels. Three types of congenital adrenogenital syndrome are well recognized:

1. 3-Beta-hydroxysteroid dehydrogenase deficiency, characterized by adrenocortical insufficiency, sodium loss, and an accumulation of steroids which are antecedents of this enzymatic conversion.[3]

2. 11-Beta-hydroxylase deficiency, characterized by an increase in 11-desoxycortisol and 11-desoxycorticosterone; the latter is a potent hypertensive mineralocorticosteroid.[4]

3. 21-Hydroxylase deficiency, the most common form of the adrenogenital syndrome, which leads to an accumulation of 17-alpha-OH-progesterone and an increase in its urinary metabolite, pregnanetriol.[15]

This patient represents a case of partial 21-hydroxylase deficiency, which was diagnosed by the absence of salt wasting and hypertension and confirmed by increased urinary pregnanetriol.

If the diagnosis of this enzymatic defect is made in the infant, the treatment of choice is glucocorticoid administration in an amount adequate to suppress endogenous ACTH.[15, 16] Normal growth and development ensue and successful pregnancies have been reported for some individuals.[8] Hormonal replacement must be instituted during infancy; otherwise, serious psychiatric maladjustment may follow sex reversal.[12] As a result of the relatively advanced age at which this patient was diagnosed, little consideration was given to changing the phenotype. Because of the recurrent inflammatory process, salpingo-oophorectomy, and total hysterectomy were done. The patient was discharged without hormonal replacement therapy, and was instructed as to the possible complications of his disease, should stressful situations arise.

Case 3.—A 28-year-old white man was referred to the Hematology Clinic in January, 1963, because of plethora and back pain of five years' duration. Hirsutism of the chest and abdomen had been noted for two years. In the year preceding admission, the patient gained 60 lbs. His past history included a short episode of unconsciousness following head trauma without apparent residual effect. Physical examination (Fig. 5) revealed an obese, plethoric, hirsute young male. Blood pressure was 158/80 mm. of mercury; pulse, 78; and weight, 217 lbs. (98.6 kg.). Heart and lungs were normal. Several bilateral, purplish striae were noted on the lower abdomen; pyoderma of the buttocks was present. Percussion of the lumbar spine revealed minimal tenderness. The laboratory data are summarized in Tables 2 and 3. Minimal decrease in carbohydrate tolerance was revealed by the intravenous glucose tolerance test. Likewise, the glucose-insulin tolerance test demonstrated the decreased insulin sensitivity which is characteristically observed in individuals with excessive circulating glucocorticoids. Results of pituitary-adrenal function studies which demonstrate the elevated values of baseline urinary 17-ketosteroids (average 37.1 mg.) and 17-hydroxycor-

Fig. 3. Case 2, congenital adrenal hyperplasia. This early postoperative photograph demonstrates excellent muscular development and male appearance. The phallus is bound down convex anteriorly.

ticoids (average 22.6 mg.) are reproduced in Fig. 6. In the dosage employed, the administration of SU 4885 resulted in a fall in steroid excretion. Of more diagnostic significance was an increase in steroid production following ACTH administration and the suppression of steroid excretion following the larger dosage (8 mg. per day) of dexamethasone.

A normal cardiac silhouette and sella turcica were revealed by chest and skull roentgenograms. In studies of the dorsolumbar spine, osteoporosis was noted. Bilateral enlargement of the adrenal glands was suggested from examination of retroperitoneal pneumograms.

Total left adrenalectomy and subtotal right adrenalectomy were performed; approximately 25 per cent of the right adrenal gland was left intact. The combined weight of the excised tissue was 25 gm. Microscopic examination confirmed the previous diagnosis of bilateral adrenocortical hyperplasia. Steroid replacement therapy was gradually withdrawn postoperatively, and by two weeks after operation, the urinary excretion of 17-ketosteroids and 17-hydroxycorticosteroids fell to relatively normal levels (Fig. 6).

The clinical diagnosis of Cushing's syndrome was suggested by findings of increased blood pressure, obesity, purple striae, hirsutism, pyoderma, and plethora. Radiological studies showing osteoporosis and bilateral adrenal enlargement supported the clinical impression. The diagnosis was strengthened by results of the urinary steroid excretion studies relating to pituitary-adrenal function tests. These tests are critical in arriving at a correct preoperative diagnosis. The most informative test in this case was the suppression of steroid secretion by the administration of 8 mg. of dexamethasone per day as first suggested by Liddle.[13] Over-responsiveness following stimulation by ACTH was noted, but this response was not diagnostic in itself.

Discussion

Examples of adrenal cortical carcinoma, congenital adrenal hyperplasia with pseudohermaphroditism, and acquired bilateral adrenal cortical hyperplasia with Cushing's syndrome have been presented to illustrate the diversity of adrenal abnormalities which may occur in association with substantial elevation of the total urinary 17-ketosteroid secretion. Wide differences in diagnostic procedures, etiology, treatment, and prognosis are clearly evident among the three cases.

When measured by the Zimmerman color reaction, the total urinary 17-ketosteroids

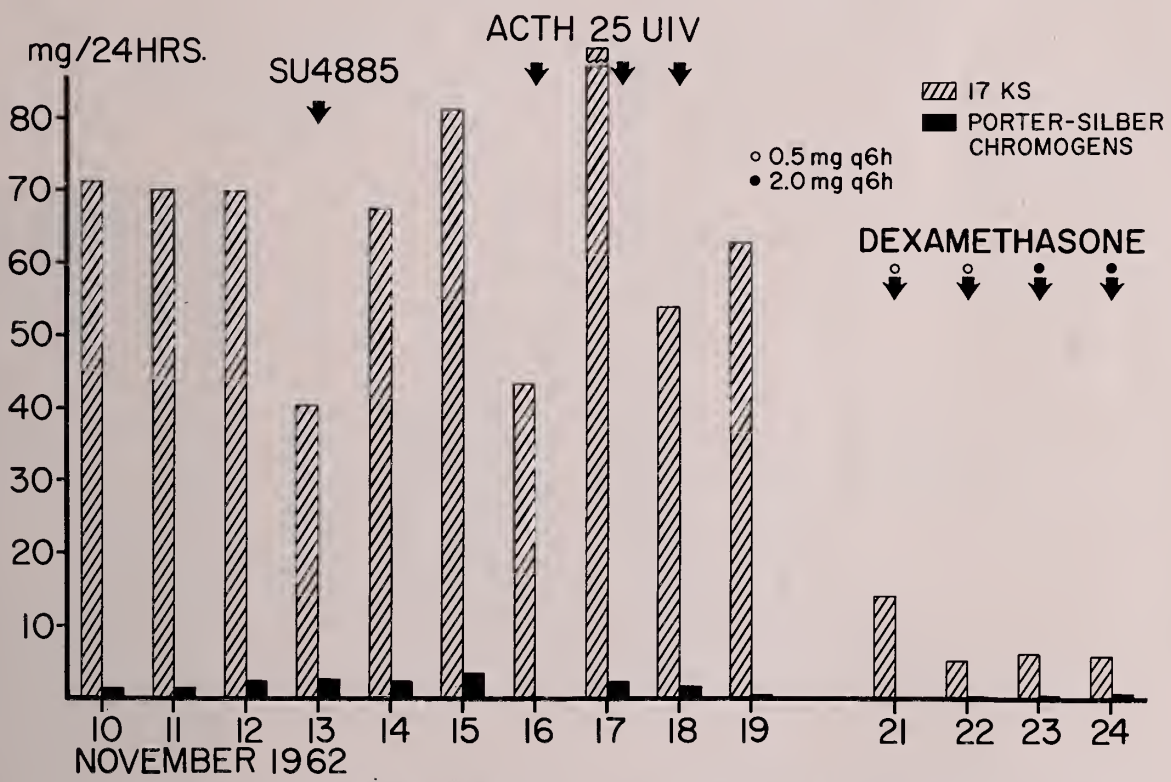


Fig. 4. Pituitary adrenal function studies in case 2, congenital adrenal hyperplasia. Note the striking elevation of the urinary 17-ketosteroids with greatly depressed urinary Porter-Silber chromogens, the failure of the Porter-Silber chromogens to respond to ACTH, and prompt fall in 17-ketosteroid excretion following suppression with dexamethasone.

represent a heterogenous group of weakly androgenic compounds arising mainly from the adrenals and testes. These steroids all possess 19 carbon atoms as well as a keto group at position 17 which is responsible for the development of the characteristic blue "Zimmerman" color when incubated with metadinitrobenzene under alkaline conditions. As these examples demonstrate, abnormalities of adrenocortical function may result in a greatly increased excretion of 17-ketosteroids which may be of diagnostic significance. Following the administration of ACTH, SU 4885, and adrenocortical suppressive agents to a patient, the comparison of 17-ketosteroids with 17-hydroxycorticosteroids is a useful diagnostic procedure.

In man, a known important factor in ACTH release and storage is the "free" hydrocortisone concentration of the blood. Reflecting this important function is the fact that approximately 80 per cent of the adrenal vein Porter-Silber chromogen secretion is hydrocortisone. According to the generally accepted principle of negative feed-back systems, a reduction in circulating hydrocortisone stimulates ACTH release and thereby increases adrenal cortical hydrocortisone secretion. Diagnostic advantage may be taken of this relationship in normal and disease states through employment of ACTH and SU 4885 stimulation as well as dexamethasone suppression tests.

The ACTH test is a measure of adrenal responsiveness to exogenously administered ACTH; results of this test are in the form of limited normal, or excessive adrenal cortical steroidogenesis following the administration of this agent. SU 4885 blocks 11-beta hydroxylation of the steroid molecule; this action depresses circulating hydrocortisone and, therefore, stimulates endogenous ACTH release. Characteristically, a rise in urinary 17-ketosteroids and 11-deoxygenated corticosteroids follows. In the dexamethasone suppression test, small amounts of this compound (either 2 or 8 mg. per day) are administered to the patient. This synthetic compound, which is about 30 times as potent as hydrocortisone, inhibits ACTH release. Following the inhibition of endogenous ACTH and adrenal cortical secretion, the presence of urinary steroid metabolites is depressed to a low level.

Case 1 illustrated failure of the endocrine system's physiological homeostatic mechanism in a case of adrenal carcinoma. During the period of repeated tests with ACTH, SU 4885, and dexamethasone, the relatively similar day-to-day excretion of urinary metabolites indicated an autonomous adrenal cortical synthesis of steroids.

In the patient with congenital adrenal hyperplasia in which hydrocortisone synthesis was impaired (Case 2), an increase in endogenous ACTH resulted, with the continual overproduction of precursors of hydrocortisone and associated urinary metabolites, and of 17-ketosteroids and pregnanetriol. Following administration of small amounts of dexamethasone, the suppression of these urinary metabolites was evident.

Case 3 (acquired bilateral adrenal hyperplasia) typifies a partially responsive pituitary-adrenal relationship. Excessive responsiveness to exogenously administered ACTH was evident. On testing with dexamethasone, steroidogenesis was suppressed with the large dose (8 mg. per day) but not with the small dose (2 mg. per day), as occurs in normal persons and in patients with simple obesity.

Glucocorticoid production was increased in Case 1 and Case 3, resulting in the expected abnormalities in carbohydrate metabolism (Table 2). Mild carbohydrate intolerance was noted in each glucose tolerance test employed and a decreased insulin sensitivity was revealed in the results of the glucose-

Fig. 5. Case 3, acquired bilateral adrenal hyperplasia. Preoperatively, obesity, fullness of the cervical fat pads, increased hirsutism, and acne are evident. Not visible are the pyoderma of the buttocks and flattened gluteal folds.

insulin tolerance tests. As expected, no abnormality in carbohydrate metabolism was detected in Case 2 when studied by these standard procedures.[7]

Of additional diagnostic value are abdominal roentgenograms which may disclose abnormalities in the size and shape of the adrenals and films of the spine which may document the presence or absence of osteoporosis. Final proof must be surgical exploration followed by histopathological study. In all three of the cases reported here, testing with SU 4885 was of ancillary value. This agent is helpful in diagnosis in situations where pituitary ACTH reserve is of major concern, as, for example, in suspected hypopituitarism.

Etiology

As is the case with any cancer, the ultimate etiology of adrenal cortical carcinoma is unknown. The tumor is characterized by an autonomous, slowly progressive, metastasizing mass of neoplastic cells which have a variable pattern of steroid secretion. Etiological possibilities include: chromosomal mutation, hormonal alteration, viral infectivity, genetic predisposition, or unknown factors.

In congenital adrenal hyperplasia, the enzymatic defects of steroid hydroxylation which have been mentioned cause a decreased synthesis of hydrocortisone. The severity of the enzymatic defect determines the degree of compensatory hyperplasia required to maintain adequate circulating hydrocortisone as well as the severity of the sex reversal. In the case presented, the enzymatic defect was relatively great, for it caused an almost complete sex reversal. The capacity to synthesize hydrocortisone, however, was sufficient to enable the patient to tolerate most physiological stressful situations.

As in carcinoma, the etiology of acquired bilateral adrenal hyperplasia is unknown. Abnormalities in either the central nervous system, hypothalamus, anterior pituitary, or adrenal cortex may be responsible for the

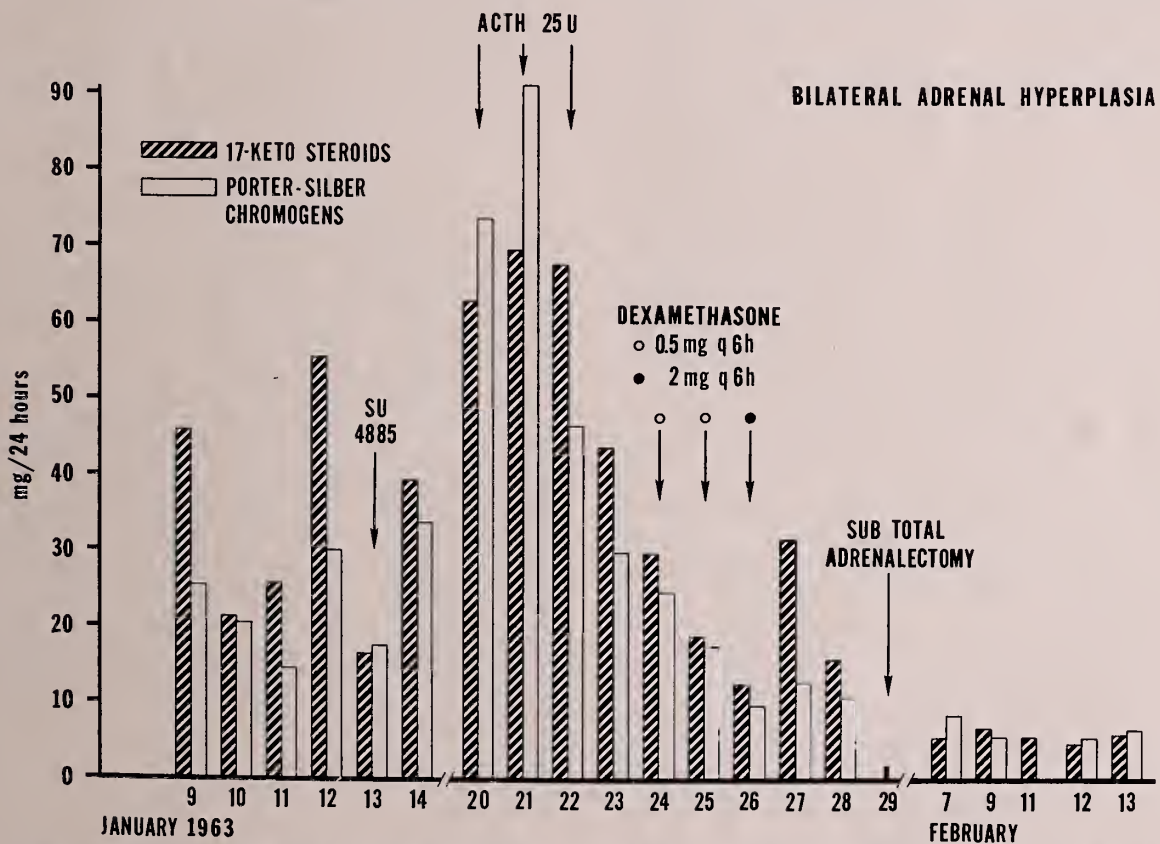
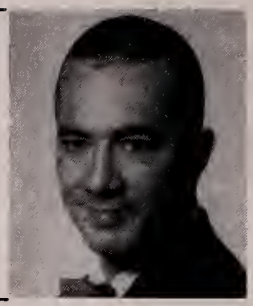


Fig. 6. Pituitary adrenal function studies in acquired bilateral adrenal hyperplasia. Note the daily fluctuations of steroid excretion in baseline period Jan. 9-12, 1963; excellent response to ACTH infusion, "suppression" of adrenal cortical secretion after 2 mg. of dexamethasone given every six hours; and normal levels of steroid excretion following subtotal adrenalectomy. On Jan. 27, by oversight, dexamethasone was not given and steroid secretion increased.

final overproduction of steroids which result in this condition. Unfortunately, there is a paucity of knowledge concerning the exact functional relationships between these organ systems.

Dr. Cole and his coauthors are associated with the Department of Medicine, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston.



Treatment and Prognosis

For each case of adrenal cortical hyperfunction cited in this paper, the treatment instituted was radically different. In Case 1, surgical extirpation of the adrenal carcinoma was impossible because of extensive involvement by slowly metastasizing adrenocortical tissue. The promising antitumor agent, o,p'DDD, was utilized, but without apparent effects for the patient steadily deteriorated. The usefulness of this agent for prolonging life in patients with adrenocortical carcinoma is limited. In the literature, investigators have reported a reduction in size of metastatic foci and diminution in steroid excretion following administration of o,p'DDD, as was shown in this case.[1] However, in our limited experience with this agent, postmortem examinations have demonstrated local necrotic destructive advancing tumor, apparently little affected by the drug therapy.

In contrast, the adrenal cortical status of the patient with congenital adrenal hyperplasia (Case 2) was left undisturbed. Since the patient had survived for 38 years without major disability, the decision was made to allow continued overproduction of adrenal 17-ketosteroids. Since continued masculinity was desirable, elevated levels of weakly androgenic 17-ketosteroids were essential in order to maintain a continued masculine psychic orientation and body build. The consequences of treating this patient with cortisol are obvious. Unless adequate male sex hormone therapy was given, loss of masculinity would ensue. Of interest is a report of a case of adrenal cortical carcinoma which developed at the age of 39 in an individual similar to the patient (Case 2) in this report.[11]

For treatment of the patient with acquired bilateral adrenocortical hyperplasia (Case 3), various forms of therapy have been suggested; these include pituitary irradiation,[14] total adrenalectomy, and subtotal adrenalectomy.[5] Recently, use of chemotherapeutic agents such as o,p'DDD and Amphenone have been reported with variable success.[1] Each type of therapy carries with it some adverse effect, however. Also, because steroid replacement therapy usually is necessary after treatment, long-term management may be tedious and may necessitate frequent clinic observations.

Despite attempts at surgical resection and the use of new antitumor agents, prognosis is poor for patients with carcinoma of the adrenal cortex. Prognosis is related to the presence or absence of extracapsular spread, including local and distant metastases, as well as to the biological aggressiveness of the tumor when diagnosis is first established. The case history presented in Case 1 was typical in that death occurred within one year after diagnosis.

In general, prognosis in congenital adrenal hyperplasia is good, providing patients are given adequate replacement therapy. If the diagnosis is established before the patient is two years of age, appropriate surgical corrective procedures and treatment with steroids will enable the individual to enjoy a relatively normal existence as a female. For patients who have not been diagnosed early and are being reared as males, prognosis remains excellent whether or not steroid replacement is necessitated.

In two recently reported large series, the overall mortality rate for Cushing's syndrome of varying etiologies approached 50 per cent.[5, 14] Many of the patients in those studies, however, were treated before steroid replacement therapy had become available. With the development of improved synthetic replacement steroids, further decreases in mortality from this disease may be expected. The patient presented in this report has done relatively well on maintenance hydrocortisone.

Summary

Three cases of adrenal cortical abnormality are reported; these abnormalities include carcinoma, congenital bilateral adrenal hyperplasia, and acquired bilateral adrenal hy-

perplasia.

The diagnostic value of total urinary 17-ketosteroids, Porter-Silber chromogens, and pituitary-adrenal function studies (ACTH, SU 4885, dexamethasone) are presented. Since the pathophysiology of the adrenal cortex in these conditions is diverse, for the successful management and prognosis of such disorders, the importance of early and correct diagnosis cannot be overemphasized.

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The authors' appreciation is extended to the members of the technical staff of the steroid laboratory, including Alonzo Guerra, Paul Ornelas, Raul Viada, Emmie Little, Anna Swander, and Jewel Reacard for their excellence in performing the steroid analysis herein described. In addition, we wish to thank Dr. T. C. Hsu of the Department of Biology for determining the presence of the normal female karyotype in Case 2. The pleasant cooperation of Mrs. Marjorie Christie in typing the manuscript is gratefully acknowledged.

Evidence Suggests Lincoln Had Marfan's Syndrome

Genetic and genealogic evidence "suggests strongly" that Abraham Lincoln's unusual body build was due to Marfan's syndrome, Dr. Harold Schwartz, Huntington Park, Calif., claims.

The syndrome is an inherited disorder of the connective tissue which affects skeletal development, vision, and the cardiovascular system.

Dr. Schwartz' interest in Lincoln was aroused by a seven-year-old boy with the classic features of Marfan's syndrome whom he saw in 1959 and later learned was connected with the Lincoln lineage.

The boy's genealogy was traced back nine generations to Mordecai Lincoln II, born in 1686, who was also the great-great-grandfather of President Lincoln.

The mother and 15-year-old sister of the patient also had atypical Marfan's syndrome, and a search of the archives revealed other scattered kin of Lincoln with Marfan characteristics as would be expected in an inherited disorder, Dr. Schwartz reported in the Feb. 15 *Journal of the American Medical Association.*

Study of Skin Temperatures

RESEARCH & INVESTIGATION

EVEN A cursory review of the literature on skin temperatures reveals extensive material on this subject.[5] Among other things, it has been established that because of the many controlling factors, both internal and external, conveniently grouped by Murlin,[3] there is no such thing as a "normal" skin temperature. However, there have been many studies of average determinations[2] using, for the most part, highly sophisticated calorimetric methods and resulting in findings of little significance to the average clinician. The interest in skin surface temperatures nevertheless remains, especially as these pertain to peripheral vascular disease. It was considered by this investigator that the determination under readily available conditions and with simple apparatus of some average readings might be of practical diagnostic use. Also considered of interest was any significant correlation between rectal and skin temperatures.

Material and Method

Two studies were made through the neuropsychiatric service of a general hospital that maintains approximately 200 chronically ill male patients.

For the first study, 95 were chosen for temperature determinations. All were in relatively good physical condition with no perceptible evidence of abnormal skin changes or peripheral vascular disease. Their ages ranged from 35 to 83 years, and more than one-half were between 60 and 70. It is possible that varying degrees of atheromatous involvement were present in many of them, however, this was not demonstrable, either objectively or subjectively. The skin areas tested were chosen arbitrarily over the courses of the larger vessels of the body and extremities. A reading was also taken on the forehead, which is considered to show the most nearly constant temperature.[5]

Skin areas tested are listed:

- Forehead, in the midline
- Preauricular areas (temporal artery)
- Submandibular region (carotid sinus)
- Supraclavicular region (innominate and subclavian arteries)
- Precordial area (heart)
- Cubital region
- Radial area, at wrist
- Ulnar area, at wrist
- Femoral area, just below Poupart's ligament
- Popliteal spaces
- Posterior tibial area, below medial malleolus
- Anterior tibial area, on dorsum of foot

The readings were taken in an ordinary, air-conditioned ward room where the temperature was maintained between 72 and 76 F. The patients were stripped of all clothing when the readings were taken.

The instrument used was a readily available, easily portable battery-actuated thermocouple (Tele-thermometer, Yellow Springs Instrument Company) that could be calibrated. Readings were taken bilaterally, where feasible.

The second study was made to determine any significant correlation between rectal and skin temperatures. For this purpose 100 temperature determinations were made using 32 patients in the infirmary ward of the service involved. Here there was no air conditioning. Some of the patients were febrile, others were not. The rectal and skin temperatures were taken simultaneously. The forehead was used for the latter reading.

Skin temperatures of various body areas were measured and evaluated. Forehead temperature readings were also compared with rectal readings.

HENRY V. MALIN, M.D.

Results and Evaluation

The results of the first study are given in Table 1 which shows the mean reading for each area, the extreme ranges, and the standard deviation for each.

TABLE 1.—*Skin Temperature Readings.*

Area	Mean (Degrees F.)	Range (Degrees F.)	S.D.
Forehead	92.6	96-90.5	1.00
Preauricular	93.7	97-90	1.31
Submental	94.6	97-92	1.19
Supraclavicular	94.7	98-92	1.26
Precordial	92.7	96.5-86.5	1.11
Cubital	93.7	97-90	1.10
Radial	92.3	96.5-87.5	1.75
Ulnar	92.0	96-87	1.83
Femoral	93.8	97-89.5	1.52
Popliteal	91.4	94.5-86	1.42
Posterior tibial	89.5	94.5-79	2.72
Anterior tibial	89.0	95-80	2.58

Statistical evaluation of the findings in the first study shows a high confidence level for the readings obtained in the individual areas, all of them being well within the .001 value. Comparison of readings taken of the two sides of the body or its extremities resulted in a p (probability) number well within the 95 per cent confidence limit. Reliability of these determinations, made under simple conditions available at almost any medical facility, was thus indicated.

Other workers[4] have cautioned that it is not safe to rely solely on the use of even elaborate instruments for making diagnoses of any kind, including circulatory disturbances. Much experience has shown that there are definite limitations to surface thermometry because of (a) extreme lability of surface temperatures in the upper extremities, (b) sluggish response to temperature variations in the lower extremities, and (c) the constant shifting of balance between vasodilation and vasoconstriction, which means that the surface temperature does not necessarily indicate the circulatory status of the deep vessels. These are control factors outlined by Murlin.[3] Other factors are hyperpyrexia, body activity, time of day, age of the individual, environmental temperatures, sleep, menstruation, and emotion.[1] Therefore, sole reliance should not be placed on skin temperature readings in making a decision regarding the circulatory status of an extremity or any other portion of the body. Higher readings should be anticipated where areas of pooled blood are in evidence, such as with venous congestion or areas of inflammation. Occlusive peripheral artery disease, functional or organic, will give lower readings. Temperature readings with a variation of more than two standard deviations from the norm should be questioned.

The mean, the range, and the standard deviation of findings in the second study are shown in Table 2.

TABLE 2.—*Comparison of Rectal and Skin Temperatures in 100 Determinations.*

Location	Mean (Degrees F.)	Range (Degrees F.)	S.D.
Rectal temperature	99.74	104-98	1.35
Skin temperature	94.34	101-90	1.86

The correlation coefficient is .0956 indicating that the correlation between rectal and skin temperatures is not significant.

The body temperature gradient under extreme conditions may range from an internal temperature of 100 F. to freezing conditions of the skin, according to Sheard.[6] In these studies, the internal gradient was shown by the mean difference of 5.4 F. for rectal and skin temperatures. The external gradient was shown by the variations between face, hands, and feet. These changes were minimal in the upper portion of the body and increased progressively from the hands to the feet.

Conclusions

Results of these studies show the following:

1. Complex or expensive equipment is unnecessary for the determination of surface skin temperatures.
2. There is no appreciable variation of skin temperatures under normal conditions on symmetrical sides of the body or its extremities.
3. Average skin temperatures (Table 1) have been established as norms for comparative use.
4. Deviations from these established norms may be anticipated in circulatory disturbances.
5. There is no statistically significant correlation between rectal and skin temperatures.
6. The presence of a temperature gradient was demonstrated, both internally and externally.

Dr. Malin is a staff physician in the neuropsychiatric service at Veterans Administration Center in Temple. This article reports the findings of a Veterans Administration research project.

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Drive-In Doctor's Office

An Abilene general practitioner serves his patients from a drive-in facility. It's an unusual structure, but patients like it, and Dr. Clyde N. Morgan says the design of his building has helped him shorten his workday by an hour and a half.

The building is shaped like a hexagon and has an entrance like a drive-in movie. Its seven examining rooms are laid out like motel units each with its own parking space.

An account of how Dr. Morgan practices medicine in his drive-in office is reported in the Jan. 13 issue of *Medical Economics*.

JOHN LOCKE, Physician-Philosopher

WILLIAM S. KEEZER, M.D.

HISTORICAL ARTICLES

QUACKERY has existed since time immemorial, and medicine has always attracted pretenders to its ranks. Quacks in medicine generally have enjoyed successful careers, since there have always been unfortunate sufferers in despair ready to become their dupes.

During the 17th century, numbers of quacks flocked to London and became prominent in the social life of the time. Many were accepted in medical circles. It was their golden age, and they appealed to all classes from king to peasant. They claimed control of mysterious and unknown powers beyond "human understanding." On the other hand, the legitimate practice of medicine was a pursuit of systems with little attention paid the patient at his bedside.

It was into such a potpourri that John Locke was thrust when he came to London as a young physician. We must be grateful for his association both with Dr. Thomas Sydenham and with the quacks for each must have influenced his masterful philosophic treatise, "Essay Concerning Human Understanding," with Sydenham the example of careful observation and the quacks examples of mystical falsehood.

Among the great men of the 17th century, Locke stands out as one of the greatest. He was a great philosopher; but in addition, he was a careful, observant practitioner of medicine, a fact that only recently has been recognized as an important basis for his philosophy.[3-5, 14-16]

He was born Aug. 29, 1632, near Bristol, England, and died at Oates on Oct. 28, 1704. During his lifetime, England went through one of the most troubled periods in her history. Locke witnessed the struggles between Charles I and Parliament, the rise to power of the Presbyterians and Independents, the execution of Charles I, the rule of Oliver Cromwell, the restoration and reign of Charles II, the reign of James II, and the Revolution of 1688 and the settlement under William and Mary. His writings deal with many of the public issues then at stake, and what is more, he had the gift of grasping the broad principles involved in the immediate issues and the perseverance of thinking necessary to formulate a general philosophical position.

While Locke raised many of the problems which have busied philosophers for more than 200 years, it was not until the appearance of H. R. Fox Bourne's "The Life of John Locke" in 1876 that it became known that he was a practitioner of medicine and that throughout his long life was ready to treat cases and give medical advice. Little was known regarding his medical essays, clinical reports, and papers prior to Sir William Osler's work, "John Locke as a Physician," which appeared in 1900.

It is the purpose of this article to show that Locke was an observer of the natural world and looked upon man as an object in nature not fundamentally different from other natural objects. He felt, this writer contends, that many cardinal errors arise

One of the greatest men of the 17th century was the English philosopher, John Locke. Recently, it has become recognized that an important basis for his philosophy was the fact that Locke was a careful, observant practitioner of medicine.

from mistaking words for things and that the findings of philosophers should not depart too widely from common sense beliefs. He supposed that he was following the scientific methods of Sir Isaac Newton, but his philosophy far more resembles that of a physician who seeks to cure diseases. A physician would trace diseases to their origins and examine their symptoms, and so would compile a manual in plain, lucid language on methods of healing.

It has been common knowledge for years that Locke was a close friend of Sydenham under whom he received much of his tutoring in medicine. For several years Locke followed Sydenham from bed to bed, observing his professional techniques and acting as his assistant and secretary. To what extent the teachings of Sydenham influenced Locke or Locke's philosophy influenced Sydenham is difficult to decide. Needless to say, Sydenham preached that the art of medicine was to be properly learned by the study of disease at the bedside, and that there was no substitute for the Hippocratic method of observation and evaluation made upon personal clinical experience.

OF LOCKE'S boyhood and early schooling, little is known. However, in 1646 he entered Westminster School under the famous Dr. Richard Busby. Among his fellow students were Richard Lower, Walter Needham, and John Mapletoft, all of whom became famous and well known physicians. In 1652 Locke entered Christ Church, Oxford, and received his B.A. degree in 1655 and M.A. degree in 1658. Locke appears to have been at a loss to know exactly what calling to follow until about 1660. He had broken with the old scholastic philosophy and had come under the influence of the philosophy of René Descartes. What may have turned his attention to medicine was the presence in Oxford of Robert Boyle who with his friends and associates had formed the "Invisible College," later to become the Royal Society of London. Newton also was in this group which founded the Royal Society. Locke felt his influence and spoke of him often as "the incomparable Mr. Newton." They remained friends for the rest of their lives and corresponded frequently.[6]

During the period of 1660 to 1665, Locke probably began studying the sciences cognate to medicine and working at problems suggested by Boyle. Although we have very little information as to the exact nature of his medical studies, we know that he did not comply with the easy requirements for a medical degree at that school.[6] All that was required was regular attendance during a period of three years at the lectures of the Arabic professor and at the lectures of the professors of anatomy and medicine. A certain number of debates in the medical school were required. The medical lectures were delivered twice a week and were limited to an exposition of the teachings of Galen and Hippocrates. For anatomy, the students had only to attend the dissection of one human body and to hear a total of four lectures on it, and then to hear three one-hour lectures on the skeleton.

> The teaching provided in the schools was nearly as useless in the education of competent doctors as were Latin verse-writing and Aristotelian disputation in the education of useful politicians and sensible clergymen.[6]

Locke did not take to medicine in order that he might follow the obsolete doctrines of Hippocrates and Galen; yet, in a way, it is strange that he did not comply with formalities. In November of 1666, he procured a letter from the Earl of Clarendon requesting that he be given a dispensation so that he could obtain the doctorship without previously becoming a bachelor of medicine. This request was not heeded. Cranston states that Locke had more than one motive for seeking this degree. One was that he wished to have academic recognition for its own sake and another was that he wanted the degree to secure a layman's place as a medical don at Christ Church. In November of 1666, he solicited a letter from the king himself and the royal command was, of course, obeyed. His academic position was now secure.[2] He remained in this position, as a senior student at Christ Church, and drew the salary attached to it until he was expelled in 1684 for political reasons. However, according to Cranston, Lower was the one who brought Locke to the study of medicine and introduced him to experimental philosophy. He also is the acknowledged source of a large proportion of the entries in a medical notebook which Locke kept from 1652 until about 1659. Most of the entries are recipes for medicines, many of which are in Latin.[2] Locke did not receive the bachelorship of medicine until 1674, and it was in

that year he was appointed to one of the two medical studentships of Christ Church. It was thought by his friends that this was a preliminary step to his taking his doctor's degree, but this he never did.

IN 1666 JOHN LOCKE, while helping and assisting Dr. David Thomas, a physician of Oxford, became acquainted with Anthony Ashley Cooper, the first Baron Ashley, later to become the first Earl of Shaftesbury. Two accounts of the meeting survive; however, these differ only in minor details. It appears that Lord Ashley had planned to spend some time with his son at Oxford and had decided to drink some of the Astrop medicinal waters.* Since Dr. Thomas had to leave Oxford, he requested Locke to take care of this matter, but there was some difficulty and delay, which Locke felt warranted a call on Lord Ashley to make his apologies. There immediately was a mutual attraction between these two men, and from this acquaintance began a friendship that lasted for many years. In fact, Lord Ashley was so impressed with Locke that, in 1667, we find that he was installed as the personal physician at Exeter House, the London residence of Lord Ashley. Locke's function was that of both personal physician and literary factotum.

Sydenham had obtained his bachelor of medicine degree from Oxford in 1648, and sometime prior to 1661, he had established a practice of medicine in London. In 1663 Sydenham was made a licentiate of the London College of Physicians, a group which never awarded him any higher rank. In 1666 Sydenham had published his first edition of "The Method of Curing Fevers," and apparently Locke had made a close study of this book. Whether Locke knew Sydenham prior to his coming to London in the summer of 1667 is of little moment, for he lost no time in forming an acquaintance with him. From this time until Sydenham's death in 1689 they were intimate friends.[6] It was Sydenham that induced Locke to pay more attention to the study of medicine and to put his medical knowledge to use. Locke collaborated with him on many cases, making calls on patients and discussing their treatments and cures, even writing introductions to Sydenham's papers. The fragmentary manuscript of Locke's entitled *"De Arte Medica"* which was written in 1669 shows how closely his thinking agreed with that of Sydenham:

> True knowledge grew first in the world by experience and rational observations; but proud man, not content with the knowledge he was capable of, and which was useful to him, would needs penetrate into the hidden causes of things, lay down principles, and establish maxims to himself about the operations of nature, and thus vainly expect that nature or in truth God, should proceed according to those laws which his maxims had prescribed him.[2, 6]

To quote Sydenham, who in essence follows this same basic type of philosophic reasoning:

> The function of a physician [is the] industrious investigation of the history of diseases, and of the effect of remedies, as shown by the only true teacher, experience. . . .
>
> True practice consists in the observations of nature: these are finer than any speculations. Hence the medicine of nature is more refined than the medicine of philosophy.[2]

Locke is indebted to Sydenham for two things that are closely related: First, he learned to appreciate the empirical method while accompanying Sydenham on his rounds of professional visits. Second, he learned that it is the responsibility of any physician to cure the sick, even though one cannot understand the essence or ultimate cause of any disease. That men must concentrate their efforts on what is useful in life became the pivot around which Locke's philosophy moved.[12]

The best evidence to date as to the amount of Locke's practice in 1667-1668 is found in a few sheets of paper which are in the British Museum. They apparently are only fragments of the whole collection of medical notes made by him. They not only contain information regarding Lord Ashley's memorable disease and the more natural ailment of Lady Dorothy Cooper, but also discuss other cases that have been well summarized by Fox Bourne:

> He was attending a kitchen-maid of the Ashley household, who was afflicted with dropsy, in September, 1667, and again in January, February, and March 1667-8, and May and June, 1668. In April and May he prescribed for a hard cough with which one of his cousins, a boy of fourteen, and the son of his uncle, Peter Locke, was troubled; and he cured one young child of inflammatory fever in June, and another of hysterics in November of the same year. He visited a "sturdy youth," laid up with rheumatism, every day between the 28th of August and the 19th of

*Richard Lower was credited with the discovery that the waters of Astrop had therapeutic mineral properties.[2]

> September, which he left him to continue using the medicine he had prescribed for him, and on the 1st of March, 1668-9, he was able to declare him convalescent. In the same March he treated a girl for fever, besides prescribing for two or three minor maladies; and in May, 1669, he cured a case of angina pectoris. We have no record of any other medical work done by him in 1669; but he had cases of erysipelas and gonorrhoea to deal with in January 1669-70; one of quartan ague in March; another malady, not specified with which William Sydenham, apparently the son of his great friend, was afflicted, in the same month; two of dropsy in April and May, 1670; one of inflammatory fever, which he treated nearly every day from the 26th of June till its fatal issue on the 11th of July; a case of disease of the kidneys, and another of stricture, in July; one of colic in August, and one of fever, which occupied him every day from the 19th of August till the 2nd of September, which time his patient died.[6]

The case referred to as angina pectoris may have been of this nature, though the disease was not recognized until a century or more after Locke. The Latin phrase that Locke uses, loosely translated, means "a choking sensation of the chest muscles and around the throat." The report on Sydenham's 11-year-old son is very complete. The terminology of the day describes it as morbilli, but according to Sir William Osler,[8] it was probably a characteristic case of the measles. The onset was with cough, a chill and fever, and a running nose. On the fifth and sixth days a rash appeared on the face first and spread over the entire body. Minute details are given. The state of the tongue, the state of the rash, the condition of the eyes, and so forth, are all given. By the tenth day, the rash had completely disappeared, but the notes continue until the 13th day.

Lord Shaftesbury's case, which Locke treated in June of 1668, is described in minute detail by Locke. Osler states, "I do not remember in seventeenth-century literature a more accurately reported case. It is one of the first instances of operation on a hydatid cyst. . . ."[8] This case shows that Locke went to a great deal of trouble to obtain opinions in the form of consultations from the leading medical minds of the time. In fact, it became almost a true symposium on the treatment of such a hydatid cyst. Above all, there is no single circumstance which better illustrates Locke's thinking as a physician than the elaborate details which he left on record of this case. It is this type of appeal to experience which characterizes much of Locke's philosophic thinking. A complete description of this case, translated into English from the Latin, is found in Osler's article in *Lancet*.[8] It is enough to summarize this by stating that it was in May of 1668 that Lord Ashley became seriously ill, enough so that even Samuel Pepys wrote about it in his "Diary." Locke consulted Sydenham and four other physicians and after obtaining opinions, had a barber-surgeon open Ashley's abdomen. Through this incision Locke drained the abscess through a silver tube of his own design, treating the wound three times daily until about the first week in September. Locke consulted his medical friends again. Should he completely remove the silver tube? Only one said yes; the other three, which included Sydenham, felt that it would be unwise, so the tube remained. Lord Ashley was restored to good health, and he wore the tube, which was later made of gold, for the rest of his life. The wits of the day found it a subject for much mirth.

As to the case of Lady Dorothy Cooper, Locke performed the functions of a male midwife on Feb. 26, 1670. To Lord Ashley's delight, the child was a boy, a grandson to inherit his title and estate. This boy grew to be Anthony Ashley Cooper, the third Earl of Shaftesbury, a moralist and author. Lady Dorothy's confinement may well have been on Locke's mind when in later years, he wrote some notes on midwifery, in which he says:

> For the mind being kept in constant apprehension of miscarriage every least occasion turns that apprehension into a real fright and what that produces in teeming women experience teaches us. Hence we see that amongst the poor and labouring country women not one of ten miscarry in comparison of the aborting to be found amongst people of quality.[2]

In August of 1679, Locke was called to consult in the case of a Mr. Beavis of Kent who was a member of Lady Northumberland's family. Exactly what Locke's relations were with these persons or whom they were is not known, but Locke gives a very detailed account of the symptoms and treatment of the patient, as well as many notes on the practice of Dr. Jacob, who was Mr. Beavis' attending physician. Mr. Beavis had a long illness which Osler feels was typhoid fever.[8] It is important to note that Locke was at this time paying much attention to his medical work. It was during this six weeks' stay in Kent that Locke himself fell ill, and

in his journal, a detailed account of his own illness is recorded. This points out his habit of carefully writing down every peculiar item that came his way, especially if it could be of any service to him in his current or future studies.

Of Locke's notable cases the final one to be discussed in this paper is that of Lady Northumberland. While in France in December of 1677, Locke was called to see the lady ambassadress who was in exquisite pain on the right side of her face. Her French doctors had felt that it was due to a toothache and had had two sound teeth pulled out with no relief. Locke gives a description of the pain as extremely sudden in onset.

> . . . as it were a flash of fire all of a suddaine shot into all these parts, and at every one of those twitches which made her shriek out her mouth was constantly drawn on the right side towards the right ear by repeated convulsive movements which were constantly accompanied by her cries.[8]

Locke made a diagnosis of trigeminal neuralgia and sent a report to his medical friends in London. His diagnosis is probably the first of its kind in medical history.[2] Locke then describes the treatment, a topical anodyne and purging. Bleeding was considered, but since it had not helped in the past, he felt it best not to try it again.† Locke felt that he had followed Sydenham's methods, and this was confirmed by a letter received in the next day or so from him.

These medical cases of Locke all show one thing in common: the careful, observant approach to the patient, based on experience and previous knowledge. This gives us a clue to his method of thinking—experience and what has been found to be valid in the light of past experience—in other words, what has been successful.

WE HAVE SEEN that John Locke was an observant, careful physician, who practiced according to no one "system," but rather treated patients in an empirical and pragmatic manner. This was in accord with the teachings of Hippocrates as modified by Sydenham. He had rejected the teachings of Galen and Avicenna, but had not accepted the then new knowledge based on the discoveries of Andreas Vesalius, William Harvey, William Gilbert, Aureolus Paracelsus, and Jean Baptiste van Helmont. This is evident when Locke states in *"Anatomia"* written in 1668:

> All that anatomy can do is only to show us the gross and sensible parts of the body, or the vapid and dead juices, all which, after the most diligent search, will be no more able to direct a physician how to cure a disease than how to make a man; . . .[6]

This paper is a forcible statement against the hope that anatomy can ever show the true nature of disease and reveal the cause of disease.

What then was the current thinking of the better physicians of Locke's day? There were two schools—the iatro-chemical school of Paracelsus, van Helmont, Thomas Willis, and others and the mechanical school of Giovanni Alfonso Borelli, Descartes, Galileo, and Sanctorius.

In addition to these, faith healing and quackery abounded. Charles II revived the practice of the king's touch for scrofula. He gave a total of over 100,000 treatments during his reign. On the day of the ceremony, those afflicted thronged to London, and as they kneeled before the king, he said, "I touch you and God heals you." Each was then handed a silver coin (touch piece). Those who remained sick were told they lacked faith.

During the plague (1665-1666), the shortage of doctors gave the London quacks a field day, for their humbug was made to order for the panic-stricken populace. It is no wonder that in 1669, Locke states in his *"De Arte Medica":*

> Length of life, with freedom from infirmity and pain, as much as the constitution of our frail composure is capable of, is of so great concernment to mankind that there can scarce be found any greater undertaking than the profession to cure diseases, *nor is there any art that so well deserves all the care and industry of its professors to improve it and bring it to perfection. . . .* [Italics added.]

In the same manuscript Locke again attacks quackery and its ilk:

> . . . nor would such a vanity be tolerable in weak, ignorant men to pretend to be the disposers of health and life, that are the free gifts of Almighty God. . . .[6]

These two quotations show Locke was even at this age, 37, questioning the truth and usefulness of things generally assumed and taken for granted.

He attacked the chemical and mechanical schools of medicine in this same manuscript

†This shows Locke's pragmatic approach to medicine.

even though the leading physicians of the day followed one or the other of the systems. Locke states:

> These speculative theorems do as little advantage the physic as food of men, and he that thinks he came to be skilled in diseases by studying the *doctrine of humors*, that the notions of *obstruction* and *putrefaction* assist him in the cure of fevers, or that by the acquaintance he has with sulphur and mercury he was led into this useful discovery that what medicines and regimen are as certain to kill the latter end of some fevers as they cure in others, . . .[6] [Italics added.]

Locke goes further in his criticism of systems of medicine when in his "Smallpox Fragment" (1670), he states:

> But tis ostentation & losse of time to lay downe hypothesis [hypotheses] wch are many times false always uncertain & make a show to enquire into the essences of things & pretend to shew the way & manner of their observacon things that we cannot know being beyond the information of our sences or the reach of our understanding & therefor with very little advantage pretend to them.[12]

The "doctrine of humors" naturally refers to Hippocrates, Galen, and Aristotle; and Locke's attitude toward these hypotheses was well shown by his attitude toward the medical teaching at Oxford. However, the notions of "obstruction" and "putrefaction" as hypotheses to the nature of the cause of disease stems from Paracelsus and van Helmont. It is worthwhile to digress into the concept of each as envisioned by these two physicians.

Paracelsus asserted that all disease can be traced to a "coagulation of undigested matter in the bowels"! Through a process of putrefaction and construction the three substances (salt, mercury, and sulphur) build up and break down the body of man. The two functions are inseparable; since just as putrefaction generates vermin—spontaneous generation of life (as was then thought), so anything which prevents putrefaction also will prevent health and cause disease. This, for Paracelsus, was the organic law of nature‡ which a good doctor must follow.[9, 11]

What then of the concept of obstruction? Van Helmont felt that fermentation in the stomach, under the direction of the soul and via the archeus was the basis of all health and disease. Food absorbed from the stomach was endowed by the liver with a natural spirit, the heart converts this to a vital spirit, and in the brain it is finally converted to animal spirit. All disease, according to him, was due to a perverted action of the archeus which sends the ferment of the stomach to improper places. The basic remote cause of this was a metaphysical causality based on the original "Fall of Man."[10] Thus he modified the Paracelsian concept, adding some of Descartes' ideas and came up with a poor hypothesis of disease when viewed empirically. Descartes' dualistic view of man as a machine endowed with a soul was the forerunner of the mechanical school which taught that all muscles and organs show a normal tonus when health prevails—the heart is a bellows, the stomach a store, the eyes are windows, and digestion of food due to muscle pressure. In such systems any obstruction or interference led to disease.

Since Locke could not "satisfactorily comprehend" the "insensible ferments," he was a "medical fallibilist" even in the beginning.[12] He shows the philosophic clarity in "*De Arte Medica*" of 1669, which was to apply in his treatment of metaphysical questions later in his finished "Essay Concerning Human Understanding." Locke's whole epistemology is expressed in a few words; true knowledge is gained from experience and observation and the lack of progress in medicine is due to the departure from experience and the reliance on vague hypotheses.

To examine further Locke's medicine as it applies to his philosophy, we should examine "Draft A" where he states:

> . . . to swallow downe opinions as silly people doe empiricks pills without knowing what they are made of or how they will work, and hath noe thing to doe but to believe that they will doe the cure, and these also are almost under the same necessity of ignorance and perhaps when we consider another part of their condition with the habitual constitutions of their mindes in a greater."[1]

What are the "empiricks pills" which he likens to ignorant opinions not based on knowledge and experience? The "*Parnassus Medicinalis*" of 1663 which corresponds to a modern text of therapeutics, tabulates some of these as standard remedies: powdered human bone in red wine cures rheumatism; prepared human skull is a cure for epilepsy; moss from the skull of a man hung in chains is a hemostatic; powdered Egyp-

‡For Paracelsus, putrefaction did not mean what it does today; rather it meant a breaking down and rearrangement of organic matter to form life—this he called the "law of organic matter."

tian mummy dissolves coagulated blood, relieves cough and pain in the spleen, hastens the menses, and is beneficial for excess gas; wearing a belt made of human skin hastens labor and eases the pain.[7] Perhaps the belt of human skin used in childbirth prompted Locke to write some notes on midwifery as previously mentioned. Certainly he did not use this belt in his conduct of Lady Dorothy Cooper's confinement and delivery in February, 1670. This example supports the contention that John Locke, the philosopher, is really John Locke, philosophical physician.

As late as 1692, in a letter to Thomas Molyneux, after the "Essay Concerning Human Understanding" had been published, we find Locke commenting on the medical practices of his day.

> I wonder . . . that, after the pattern Dr. Sydenham has set them of a better way, men should return again to the romance way of physic. But I see it is easier and more natural for men to build castles in the air of their own than to survey well those that are to be found standing. . . . What we know of the works of nature, especially in the constitution of health and the operations of our own bodies, is only by the sensible effects, but not by any certainty we can have of the tools she uses or the ways she works by; so that there is nothing left for a physician to do but to observe well, and so, by analogy, argue to like cases, and thence make to himself rules of practice. . . . If my zeal for the saving men's lives and preserving their health, which is infinitely to be preferred to any specilations never so fine in physic, has carried me too far, you will excuse it in one who wishes well to the practice of physic, though he meddles not with it."[6]

From this it can be seen that Locke still considered himself a physician and capable of explaining to a young doctor the right way for him to follow in his profession. In this letter he, as always, appeals to experience and careful observation. Romanell states that such an appeal to experience in matters medical becomes generalized in Locke's "Essay Concerning Human Understanding," and as a consequence, experience itself becomes the ultimate source for him of all human knowledge.[13]

The author originally prepared this paper for a seminar in philosophy under the direction of Patrick Romanell, Ph.D., at the University of Oklahoma where Dr. Keezer is working toward a degree in history and philosophy of science.

LOCKE'S most momentous gift to philosophy was his theory of knowledge. His approach to the problem of nature was that of the physician, and he criticized his original profession, medicine, freely for its failure to learn by direct experience and observation. In "*De Arte Medica*" he reacts to the traditional medicine of his day, since he believed that medicine was an art. In this manuscript, the three strains of Locke are seen: the empirical, the pragmatic, and the agnostic. He was sceptical about the future of medicine as can be seen by his letter to Thomas Molyneux of 1692, yet he felt that observation and experience could improve it. In comparing the two aforementioned manuscripts—"*De Arte Medica*" and the letter to Thomas Molyneux, the basic theme remains the same. One doubts that Locke really changed his views on epistemology in the years following 1669.

If such be the case, then "*De Arte Medica*" written shortly after his dramatic and successful treatment of Lord Ashley for the hydatid cyst of the liver must reflect his views on the theory of knowledge (medical) as learned by the above treatment, and have been born of this experience. Sydenham must have been influenced by Locke as well as Locke having been influenced by Sydenham. To paraphrase Romanell:[12] Locke's "historical, plain method" is Sydenham's method of natural history of disease. When used in the "Essay Concerning Human Understanding" it becomes the medical method generalized. That is to say it is a method that shuns all explanations of a speculative nature and restricts itself to the observation, description, and classification of the facts. Locke tried to be a natural historian of knowledge.

Locke followed the thought processes of a medical mind, and in "Essay Concerning Human Understanding" he advances the following ideas: First, all ideas come from experience, either by *sensation* or *reflection.* Second, the mind combines simple ideas to form complex ones. And third, the qualities of objects are either primary—a part of the object, or secondary—in the observer. These basic ideas all arose from Locke's training and experience as a physician, in spite of the fact that he never was too active in practice. The cases he did treat were unusual and his methods of treatment were based on observation and experience, using the knowledge at hand and not forming vague hypoth-

eses. John Locke, then, was a physician first, who later became a philosopher, and is now known for being the first philosopher in the British empirical school. His friend Sydenham, applying the jointly worked out epistemology, is the medically famous partner. One cannot study either man without some knowledge of the other.

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Lord Shaftesbury and English Literature

The silver tube which John Locke's patient Lord Shaftesbury wore after his operation provided sport for comedians and poets of the 17th century. Carryl, in his satire, "The Hypocrite," concludes:

The silver pipe is no sufficient drain
For the corruption of this little man.

John Dryden, who hated Shaftesbury, immortalized him in Act III of "Albion and Albanius" in which the King is represented by a huge drawing of a man and several fanatical heads sucking the poison from a tap in his side. Again, in a mock account of Lady Gray's apparition, he says: "Bid Lord Shaftesbury have a care of his spigot; if he is tapt all the plot will run out."

But the Poet Laureate did not confine himself to Shaftesbury's spigot. In "The Medal," he describes Shaftesbury as

A martial hero first with early care
Blown, like a pygmy, by the winds to war;
A beardless chief, a rebel ere a man,
So young his hatred to his prince began.

Of all the literature occasioned by Shaftesbury's political career, Dryden's "Albion and Albanius" contains the most scathing remarks:

A name to all succeeding ages curst;
For close designs and crooked counsels fit,
Sagacious, bold and turbulent of wit,
Restless, unfix'd in principles and place,
In power unpleased, impatient of disgrace;
A fiery soul which, working out its way,
Fretted the pigmy body to decay
And o'er informed the tenement of clay.

But Dryden's portrayal of Shaftesbury as "unfix'd in principles" because he deserted the Royalists, joined the Puritans, was later accused of conspiring against them to bring back Charles from France, and finally suspected of trying to overthrow Charles II becomes more interesting when one examines Dryden's own political thoughts. In 1658, he celebrated Oliver Cromwell in "Heroic Stanzas"; then in 1660, he too changed sides and applauded Charles II in "Astraea Redux." Similarly in religion, he upheld the Anglican viewpoint in "Religio Laici" in 1682, and in 1687 he switched to Roman Catholicism in "The Hind and the Panther." In each case Dryden was on the winning side.

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F. A. Bashour, M.D., A. C. DeGraff, M.D., Morris Ziff, M.D.,
R. L. Johnson, M.D., J. R. Crout, M.D., and C. T. Ashworth, M.D.

Clinicopathologic Conference

This is the first in the 1964-65 series of clinicopathologic conferences to be provided by the University of Texas Southwestern Medical School at Dallas. Dr. C. T. Ashworth, chairman of the Department of Pathology is guest editor and also presents the pathologic findings.

Clinical summary is presented by Dr. Wayne Smith of the Department of Internal Medicine; radiologic findings by Dr. K. T. Dockray, Department of Radiology; and clinical discussion by Drs. C. B. Chapman, F. A. Bashour, A. C. DeGraff, Morris Ziff, R. L. Johnson, and J. R. Crout of the Department of Internal Medicine.

Clinical Summary

DR. SMITH: This was the first admission to Parkland Hospital of a 39-year-old white female. Apparently she had been in good health until two days prior to admission. At this time she developed a cough which was productive of thick white mucus. The following afternoon she began to vomit every 15 to 20 minutes. She then noted a decrease in urine volume. A vague aching was also experienced in the epigastrium and in the middle back. There was no history of hematemesis, melena, or urinary changes.

On the day of admission she was seen by a private physician. He recorded a blood pressure of 70/50 and observed the presence of "congested chest and marked dehydration." She was referred directly to the emergency room at Parkland Hospital.

Physical examination on admission to the emergency room revealed a well oriented, but weak, white female. She was able to move from one stretcher to another without assistance. The skin was pale, and neck veins were not distended. Blood pressure was 50/0; pulse 140; temperature 93 F.; and respirations 20. The neck was supple. Examination of the lungs revealed moist inspiratory rales on the left side anteriorly, but otherwise the lung fields were clear. No clinical evidence of cardiac enlargement was detected and no murmurs could be heard. The abdomen had a doughy consistency and there was slight tenderness in the epigastrium. Neither rigidity nor rebound were present. High-pitched bowel sounds were audible over the epigastrium with each respiration. There was no peripheral edema, and the neurologic examination revealed no abnormalities. Rectal examination was negative except for guaiac-positive feces.

Laboratory findings were as follows: Hemoglobin 13.1 grams, hematocrit 39 per cent, WBC 14,100 with 65 per cent segmented neutrophils, 12 per cent bands, 21 per cent lymphocytes and 2 per cent monocytes. The blood urea nitrogen (BUN) was 34 mg./100 ml., blood sugar 200 mg./100 ml., and amylase less than 320 Somogyi units per 100 ml. Sodium was 130, potassium 4.8, carbon dioxide 20, and chlorides 98 mEq. per liter.

In the emergency room attempts were made to obtain blood by venipuncture and to administer fluids by this route. These efforts were futile and a cutdown was performed. The patient was immediately placed in Trendelenburg position because of hypotension. Two liters of lactated Ringer's solu-

A 39-year-old woman who had reportedly been in good health died following a suddenly occurring hypotension with electrical alternans.

tion were given rapidly. There was no response of the blood pressure to this therapy. Aramine was started and the systolic blood pressure rose slightly to 80 to 90. She became cyanotic, especially in the neck, face, and shoulders. The neck veins were distended. Trunk and extremities, however, were pale and clammy, and the veins collapsed.

A chest roentgenogram showed enlarged cardiac silhouette. Heart sounds were inaudible at this time. An electrocardiogram obtained upon entrance showed no ST-T change.

The patient became less responsive and respirations weakened. While preparations were made to monitor a pericardiocentesis, the patient became apneic, and no heart action could be detected. A needle was promptly placed into the chest through an intercostal space just left of the xiphoid process, and 20 cc. of dark red blood were obtained. Aspiration was discontinued and external cardiac massage was begun. It was impossible, however, to obtain an effective pulse, and an oscilloscopic tracing revealed a straight line. An external pacemaker was tried without success. The patient died five hours after being brought to the hospital.

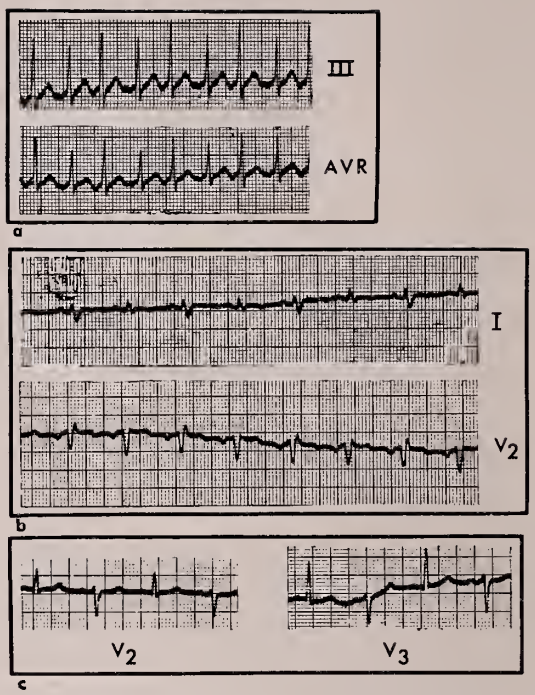

Fig. 1. a. Electrical alternans in a case with paroxysmal tachycardia.

b. Electrical alternans in a case with pulmonary heart disease showing conduction defect. Neither of the types of electrical alternans shown in a or b occur in pericardial effusion.

c. Electrical alternans of type seen in this patient. (EKG in the case presented was lost. This is an example of comparable electrical alternans in another patient with pericardial effusion.)

Clinical Discussion

DR. BASHOUR: At the time of her admission to the emergency service, an electrocardiogram was obtained which revealed the presence of electrical alternans. The significance of this finding will be discussed in the light of the present clinical picture. The pulse rate was 120 per minute; there was sinus arrhythmia; and the QRS voltage was slightly decreased.

Electrical alternans may be of three types —those associated with paroxysmal tachycardia, those exhibiting ventricular conduction defects, and those with normal QRS (in duration) showing only alternations in voltage from one beat to the next with moderately rapid pulse. This latter type of electrical alternation in QRS complex (ventricular) may be associated with alternation in P wave, giving rise to total electrical alternans (Figs. 1a, 1b, 1c).

In the present case only QRS (ventricular) electrical alternans was observed. The significance of this electrocardiographic finding was recently reported by us.[1] The total electrical alternans variety is practically pathognomonic for pericardial effusion and cardiac tamponade. In our six cases, this variety was present in only one case. On the other hand, the ventricular variety, usually associated with the same clinical entity of pericardial effusion with cardiac tamponade, was present in the remaining five cases.

The pericardial effusion was an exudate in all our cases. In one instance reported by Trout,[2] the fluid was a transudate. In four of our six cases, the pericardial effusion was hemorrhagic, secondary to a neoplastic infiltration of the pericardial sac, and in two the primary lesion was in the lung.

With these points in mind, the most likely diagnosis we can come to is that the patient had a neoplastic process involving the pericardium.

Question: Do you know of any other causes besides pericardial disease for this type of QRS change?

Dr. Bashour: We critically reviewed in our report all the reported causes of QRS alternation. This EKG finding was observed in the presence of coronary artery disease, cor pulmonale, following pneumothorax, and in one instance a normal woman.

DR. DEGRAFF.—This is a 39-year-old woman to whom something rather drastic happened two days prior to admission to this hospital. Whatever this was, it progressed very rapidly, resulting in death within a two-day period. The illness apparently began with a cough which was productive of thick white mucus not described as purulent. This progressed rapidly to vomiting and then to symptoms and findings of dehydration with reduced blood volume and so on. Later the process progressed to shock, and it was in this state that she was admitted to the hospital.

I would not like to indicate a particular diagnosis because I don't really know that I could, but I would like to suggest a direction for some thoughts in analyzing the case. A major problem which confronts us is the reason for her shock. Shock can be due to any one or a combination of several factors: diminished cardiac output, the effects of toxic products of bacteria or other toxins, or fluid volume loss.

We do have evidence that this woman lost a considerable amount of fluid volume. The serum sodium of 130 mEq. per liter in the presence of dehydration is consistent with loss of fluid by vomiting. Despite rather obvious dehydration, the hemoglobin was not increased. Therefore, the possibility of blood loss is raised although the mild anemia at the time of admission could just as well be explained by either preexisting anemia or intravascular hemolysis. If we take the anemia as secondary to blood loss, then the profound state of shock in which this woman was admitted is explained by diminished intravascular volume resulting from the combination of dehydration and hemorrhage.

Alternatively, the anemia may have been related to intravascular hemolysis. The presenting symptom was productive cough, followed shortly by vomiting. Salmonella infection is frequently associated with bronchitis and hemolytic anemia. Shock can result either from dehydration or from the bacterial endotoxins.

Dr. Bashour assures us that the patient had EKG evidence of pericardial effusion. The protocol states that on pericardiocentesis blood was found in the pericardial sac. As Dr. Bashour indicates, bloody pericardial effusion is most frequently associated with invasion of the pericardium by metastatic carcinoma, most often from the lung. X-rays fail to disclose any pulmonary mass; however, we have seen widespread metastases from small pulmonary lesions not detectable on plain chest x-rays. It is conceivable that the vomiting and cough could be the result of cardiac tamponade. It must be noted, however, that Salmonella infection has been known to cause pericarditis and that a complication of pericardiocentesis is hemorrhage into the pericardial space, resulting in a bloody effusion. From information available, I am unable to say whether this patient's shock and death were the result of fluid loss, bacterial toxins, or reduced cardiac output.

DR. ZIFF: I have seen several cases of acute arsenic poisoning in young women which progressed quite rapidly, and the course extended over two days. These patients died in shock with intestinal involvement, and a severe cyanosis occurred due to dilatation of the cutaneous capillaries. The capillary dilatation was widespread, leading to shock and to oozing into the gut. Many of these things this patient had, and I just wanted to mention it for consideration.

DR. JOHNSON: The possibility of a ruptured esophagus should be considered in an individual who has been vomiting frequently and develops shock. This will cause a severe mediastinitis which can produce shock as a consequence of sequestration of considerable

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Dr. Charles T. Ashworth, professor and chairman of the Department of Pathology at Southwestern Medical School, Dallas, reappears this month as guest editor of a new series of clinicopathologic conferences. Dr. Ashworth will serve TEXAS MEDICINE for 12 conferences through March, 1965. He formerly guided the CPC series for 1962-63.

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amounts of fluid within the mediastinum, causing hypovolemia and interfering with cardiodynamics, or by secondary mediastinal infection leading to septicemia and endotoxic shock. Of course, this should cause an enlarged mediastinum by x-ray; however, the heart alone appears enlarged. In a ruptured esophagus case I would expect to find a pleural effusion, usually on the left where tears are more apt to occur. Furthermore, the patient did not vomit blood which is common with esophageal tears, so I doubt if mediastinitis secondary to esophageal rupture is the correct diagnosis.

Another diagnosis which should be considered when an individual suddenly goes into shock and presents an enlarged cardiac shadow is a large saddle embolus to the pulmonary artery. This would cause acute cor pulmonale and a marked decrease in the cardiac output. Again, however, the EKG seems to rule this out.

So I would have to remain with the diagnosis Dr. Bashour gave us, that is, pericardial tamponade from unknown cause.

DR. J. R. CROUT: I would like to make a very short comment about her being placed in the Trendelenburg position. In a recent controlled study on dogs subjected to hemorrhagic shock, it was found that the head-down position, if anything, increased the incidence of death. It is one thing to elevate the legs to increase venous return to the heart but quite another to drop the head below the level of the heart. The latter probably serves only to stagnate blood in the head. I believe that the Trendelenburg position, at best, is without merit and is potentially harmful in shock.

DR. C. B. CHAPMAN: I have some questions about this case, and I would like to start by noting that this woman, according to the protocol, was perfectly well 48 hours before death. It follows that she must have had something catastrophic to kill her this quickly, and I don't know of any kind of pericarditis that would do this. Tuberculous pericarditis, or so-called nonspecific pericarditis, can move relatively fast, but I think this case has proceeded too rapidly for this diagnosis.

The next problem is that this patient was vomiting every 15 to 20 minutes. This developed within 24 hours after the onset of cough. This suggests the ingestion of a toxic agent. The other possible cause of this is obstruction, which is basically what Dr. Johnson had in mind.

I don't know that we really have enough here to make a diagnosis. On the basis of Dr. Bashour's interpretation of the electrocardiogram, I will accept the fact that this patient had pericardial effusion; however, I doubt that this is the cause of death. There was some other process, and it may have been ingestion of a toxic agent or a gram-negative bacteremia attendant upon some form of acute gastrointestinal-tract disease with obstruction which possibly perforated the chest. This could have yielded a blood stream infection. What the specific lesion might be, I don't think we can really say. It happens that up on the wards today we have a patient with a large hiatus hernia which developed rapidly, somewhat like this patient's course. One might assume a process is going on here that results in obstruction, perforation, and contamination of the mediastinum, but I think this is too far off. I would lean toward an acute GI-tract catastrophe leading to obstruction and shock probably on the basis of bacteremia. I believe the pericardial effusion was secondary and not the primary cause of death.

Dr. Bashour: Where did you get the history of vomiting? From the patient? Was the relationship of the vomiting to coughing determined? When some individuals cough they vomit, and then they can't stop either one.

Dr. Chapman: I think this is a good point. It may well be, of course, that she started off with cough and just vomited a few times, but that is not the way the record reads.

Radiologic Findings

DR. K. T. DOCKRAY: Posteroanterior and lateral chest films are of limited diagnostic value due to underpenetration, but they demonstrate an enlarged heart shadow with a cardio-thoracic ratio of 15.5 to 27 cm. Minimal fullness in the region of the pulmonary artery is thought to be present, although the pulmonary vascular volume does not seem to be increased. Angles formed by the cardiac margins and diaphragm do not appear to be consistent with a large pericardial effusion, and no chamber predominance is identified.

Lungs are free of confluent infiltrates. Minor stranding appears in both bases (Fig. 2).

Fig. 2. Anteroposterior roentgen-ray view of chest.

On a single abdominal film, visceral outlines are fairly well shown. No enlargement of liver or spleen is seen. The psoas margins are indistinct. The renal outlines appear to be within normal limits.

A relative paucity of fecal and gas shadows over the entire left portion of the abdomen may be a reflection of fluid-filled loops of the bowel. In the lower pelvis, also on the left, there is the suggestion of extrinsic pressure on a bowel loop, but no clear-cut mass lesion can be identified. Small, punctate calcifications are noted in the pelvis which probably represent phleboliths. The bones appear normal.

In summary, this film series shows an enlarged cardiac shadow and fails to demonstrate major pulmonary disease.

Pathologic Findings

DR. C. T. ASHWORTH: Examination of the opened chest revealed a tense and bulging pericardium. At the time of death, the heart was actually larger than it appears to be in the chest x-ray. Before opening into the sac, it appeared blue, clearly indicating hemopericardium. A massive accumulation of blood was not found inside the pericardium, but there were about 300 cc. of blood and blood clot and the pericardium was tense. Even with this comparatively small volume of blood, cardiac tamponade may occur if the blood accumulates rapidly, which apparently it had.

The heart was small and contracted as would be expected from this effusion. It weighed 240 grams, and the pericardial surfaces were granular due to deposits of fibrin. First observations at autopsy suggested the possibility of an infectious type of pericarditis. Accordingly, gram strains were done immediately and material for cultures was obtained. Gram stains and cultures, however, were entirely negative.

In proceeding with the examination, a distinctive lesion was found in the upper portion of the lower lobe of the left lung. It was located in such a position that it was obscured in the anteroposterior chest x-ray

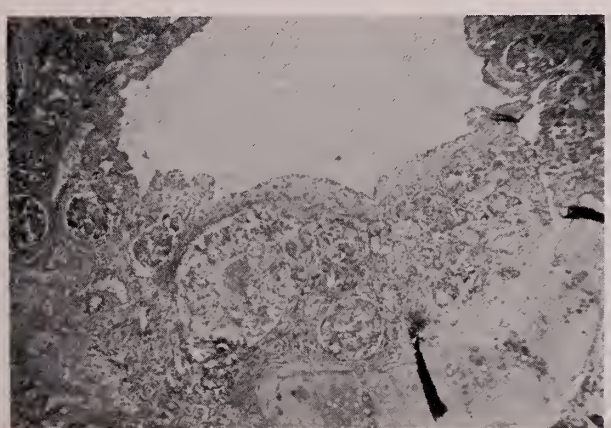

Fig. 3. Low power photomicrograph of area of primary bronchogenic adenocarcinoma. Tumor cell groups are seen throughout the bronchial wall. Some of the tumor cells are in lymphatics.

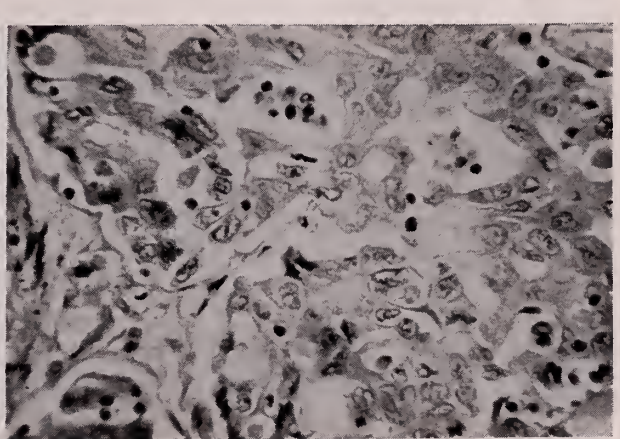

Fig. 4. High power photomicrograph showing the appearance of the neoplastic cells. A slight tendency toward acinus formation can be seen.

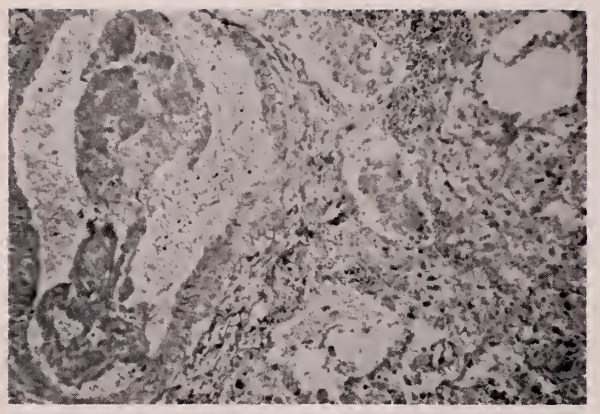

Fig. 5. Low power photomicrograph of a portion of left lower lobe showing thrombosis in a small artery and a few clusters of tumor cells in adjacent lymphatics.

of the heart, and I doubt that it will be possible to see it now even knowing there is such a lesion. It was a dense, hard mass, located near the hilus. It measured about 4 by 4 by 3 cm. In the central area this lesion was partially liquefied, making us think again that we were dealing with an infectious process. The smears obtained, however, were negative, and, in fact, did not even contain neutrophils. Only blood and degenerating cells were apparent, so we did a frozen section on the surrounding lesion. It proved to be a carcinoma of the lung, which surprised us considerably.

Histologic sections showed the tumor to have a pronounced tendency to invade lymphatic channels. I think this may be a clue to the eventual strange behavior of this tumor. The tumor cells revealed a slight tendency to form glands. The highly malignant tumor was classified as bronchogenic adenocarcinoma (Figs. 3, 4).

In the affected lower lobe we found an area of segmental atelectasis in which there was an acute and chronic inflammatory process. This was due, of course, to the partial bronchial obstruction by the tumor mass. In other parts of the lower lobe, foci of lymphangitic carcinomatoses were found which were frequently associated with small pulmonary artery thrombi (Fig. 5). This was found all throughout the lower lobe of the left lung, but the other lung was free from lymphangitic metastases. I think it was by means of this lymphatic dissemination that the tumor was able to penetrate into the lymphatics of the mediastinum and into the pericardium. There was only one hilar lymph node that showed metastatic carcinoma, and there were no distinct metastases elsewhere in the body. There was only a minimal degree of pulmonary edema.

In the pericardium itself the main alterations were a fine inflammatory reaction consisting of a few neutrophils; lymphocytes, macrophages, and deposits of fibrin; areas of extensive interstitial pericardial hemorrhage; and very sparse neoplastic cells (Figs. 6 and 7). The neoplastic cells, though few, were present in enough quantity that they were of primary importance in leading to the pericarditis.

The major pathologic diagnoses, then, in this case are

1. Bronchogenic adenocarcinoma of the left lower lobe bronchus.
2. Left lower lobar lymphangitic carcinomatosis.
3. Lymphatic extension of bronchogenic adenocarcinoma to the pericardium.

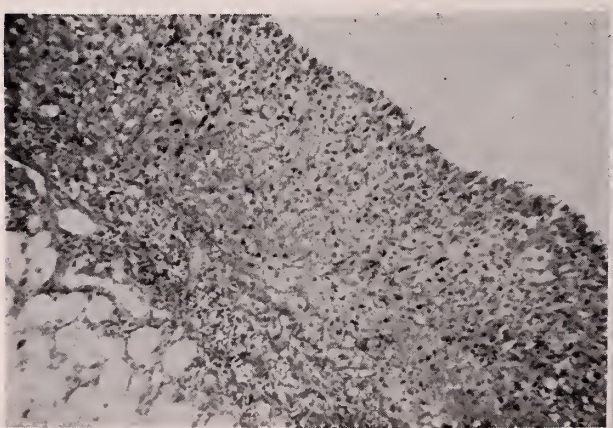

Fig. 6. Low power photomicrograph of visceral pericardium. Mild diffuse inflammation and recent interstitial hemorrhage can be seen.

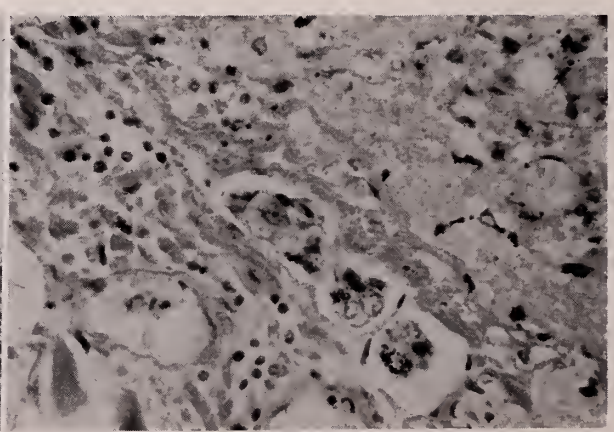

Fig. 7. High power photomicrograph of area of hemorrhagic pericarditis. Small groups of neoplastic cells are noted in lymphatic vessels of the epicardium.

4. Hemopericardium with cardiac tamponade.

DR. CHAPMAN: I would like to clear up one thing and say that 300 cc. of fluid in the pericardium, if it accumulated quickly, was quite enough to produce tamponade. I don't think that there is any question that this woman did die in tamponade, although it wasn't entirely clear from the description we had. The only thing I would like to say is that when carcinoma, usually bronchogenic, invades the pericardium, rapid accumulation of fluid occurs; frequently it is a bloody fluid, but usually it is not this rapid. This is the kind of thing that one would expect to produce tamponade over a period of some days rather than catastrophically. This must have happened in a few hours, and my only question now is whether there was some other factor to cause her to bleed so quickly. I don't think there is any question that the invasion of the pericardium was responsible for the bleeding and that the patient did die in cardiac tamponade.

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2. Traut, E. F.: Alternans: Report of Case Associated with Acute Pericarditis, Postgrad. Med. 8: 439 (Nov.) 1950.

Drs. Smith, Dockray, Chapman, Bashour, DeGraff, Ziff, Johnson, Crout, and Ashworth, University of Texas Southwestern Medical School, 5323 Harry Hines Blvd., Dallas.

Cure Believed Found For Trichinosis

The first specific cure for trichinosis is believed to have been found by three Texas physicians.

A 34-year-old woman who contracted trichinosis from eating uncooked pork made a rapid recovery after she was given a new broad spectrum drug to fight intestinal worms, thiabendazole, Drs. Orville J. Stone, Charles T. Stone, Jr., and J. Fred Mullins, Galveston, reported in the Feb. 15 *Journal of the American Medical Association.*

The drug, limited to investigational use in human beings, had been previously reported to be effective against trichinosis in animals, and the drug had also been used in patients for diseases caused by other intestinal parasites.

On admission to the hospital, the Galveston patient could not walk unaided and had generalized muscle pain and tenderness, according to the *Journal* report. Thiabendazole was administered on the sixth hospital day, and within 48 hours, she showed marked improvement. Seven days after this drug therapy was begun, the woman was discharged from the hospital with normal strength and no muscle pain or tenderness. The only side effect was mild, transient dizziness.

"Although this is but a single case report, the response was sufficiently dramatic to suggest that thiabendazole is highly effective in cases of human trichinosis since such rapid improvement is inconsistent with the normal course of this disease," the authors said.

NEWS

4,000 Expected for Annual Session

THE LARGEST annual medical meeting in the Southwest is only days away. The 97th Annual Session of Texas Medical Association will open in Houston on April 23 for four days filled with activity. Four thousand members and guests are expected to attend.

Coheadquarters will be at the Shamrock Hilton and The Towers. Members who have not yet made room reservations should contact Mr. E. N. Drake, executive assistant manager of the Shamrock Hilton, who will arrange for housing. Requests should indicate first, second, and third preferences. Insofar as possible, preferences will be honored and confirmation of reservations will be sent directly to the individual making the request. A list of hotels and motels, addresses, and rates appears on p. 395.

Those planning the Annual Session program have been fortunate to secure only a few weeks ago the commitment of Edward Teller, Ph.D., the father of the hydrogen bomb, to speak on Sunday, April 26, at a luncheon in the Continental Room of the Shamrock Hilton. Dr. Teller, director of Lawrence Radiation Laboratory, University of California, Berkeley, will speak on the subject "Operation Plowshare and Peacetime Uses of Atomic Energy." His lecture is being sponsored by the Committee on Nuclear Medicine.

Baylor University College of Medicine will hold an open house for members of the TMA and their families from 2 to 5 p.m. Thursday, April 23. Three new research buildings valued at $8,500,000 will be open for Texas physicians to visit. Refreshments will be served during the open house in the Rayzor Student-Faculty Center, a part of the new additions.

Dr. Teller

Dr. Berry

Since Houston is now a space-oriented city, with NASA Manned Spacecraft Center located nearby, it is fitting that one of the closed-circuit television presentations will concern space medicine. Dr. Charles A. Berry, chief of Center Medical Operations Office at NASA, will discuss the preparation of the astronaut for flight and his monitoring while in flight in a talk on "Medical Support of Man in Space," Saturday, April 25, at 11 a.m. before a general meeting session.

At the Friday general meeting session, physicians will see another television presentation, "Low Back Pain," by Dr. Mark B. Coventry, head of the Section of Orthopaedic Surgery, Mayo Clinic, Rochester, Minn.

Both television presentations may be seen in the Emerald Room of the Shamrock Hilton Hotel. By a two-way audio system, the audience will be given an opportunity to direct questions to the speakers. As in past years the television system is the large-screen color system, Eidophor, developed by Ciba Pharmaceutical Company, Summit, N. J.

Part of the Annual Session will be the meeting of 27 related and specialty groups.

Other attractions are 14 refresher courses, 11 scientific sessions, Curbstone Consultations, 200 scientific and technical exhibits, and the Texas Academy of General Practice Seminar.

The program, printed in its entirety in the March *Journal*, lists 32 guest speakers plus 50 special speakers.

Business of the Association will be dealt with in committee, council, and House of Delegates meetings. Some issues to be discussed are outlined in the following story on this page.

House To Consider Merits Of Resolutions, Annual Reports

Resolutions to be considered by the Texas Medical Association's House of Delegates this year include formation of special policy committees for liaison between the medical profession and the public; opposition to compulsory area-wide planning for health facilities; and the need for more family physicians. The resolutions will be presented during the Annual Session in Houston April 23-26.

Citing the need for cohesion and uniformity of opinion expressed by members of the medical profession to other groups, and the fact that physicians sometimes offer opposing views because they lack complete information about, or understanding of, the profession's policies and thereby create confusion with other groups, one resolution proposes a special policy committee at state and local levels. The committee would be charged with studying methods by which any deficiency in understanding and disseminating the policy of medicine can be corrected, with the goal of better implementation of liaison between medicine and the public in counsel and in service. Proposing this resolution is the Navarro County Medical Society.

From the Tarrant County Medical Society comes a resolution opposing area-wide planning for health facilities when such planning is compulsory and implemented by state agencies. The resolution notes that S.B. 885 passed the Senate without debate, and affords federal recognition and commendation for all such planning boards and commissions, and lays the groundwork for ultimate complete control by such boards.

Rooms Still Available At These Places:

NOTE: See story on page 394 about procedure for making room reservations for TMA Annual Session, Houston, April 23-26.

	Single	Double	Twin	Suite
Sheraton-Lincoln Hotel 777 Polk Avenue	$9.50-$15.50	$14.75-$16.50	$14.75-$19.50	$32.50 up
Hiway House 9300 South Main	$8-$12	$10.50-$16	$10.50-$16	
Tidelands Motor Inn 6500 South Main	$9-$14	$13-$20	$13-$20	$20-$29
Las Vegas Motor Hotel 9604 South Main	$7.75-$9.75	$9.75-$11.75	$10.75-$14.75	
The Warwick Main and Hermann	$8-$10	$11-$15	$13-$15	$25-$50
The Hotel Plaza 5020 Montrose	$6 up	$8.50 up	$8.50 up	$20-$35
Twenty-Nine Palms Motor Hotel 9430 South Main	$6.75-$7.75	$10.75		
Grant Hotel 8200 South Main	$7 up	$7.50-$8	$9-$12	
Sun Valley Motor Hotel 1310 Old Spanish Trail	$7.50	$8.50	$9.50	
Holiday Inn 10319 South Main	$7.25	$8.50-$9.50	$10.50-$12.50	$15.50 up
The Surrey House 8330 South Main	$9	$11.50-$16.50	$13-$18	

Another Tarrant County resolution concerns the need for educating more family physicians for Texas.

These, in addition to the major issues of creating a nominations board, giving the Board of Councilors explicit authority to divide a multi-county society into one or more societies, and an increase in TMA dues (see March issue of *Journal*, page 200) will be considered along with the annual reports of some 47 of the Association's boards, councils, committees, and officers. These reports are printed in the "Handbook for Delegates" which was mailed to all delegates late in March.

SITE OF TMA's 97th Annual Session is Houston, now a space-oriented city with NASA Manned Spacecraft Center located at nearby Clear Lake. An aerial view of the Center is shown below. One of the Annual Session closed-circuit television presentations will concern space medicine and will be given by the chief of Center Medical Operations Office at NASA, Dr. Charles A. Berry. His topic is "Medical Support of Man in Space" and concerns preparation of the astronaut for flight and the monitoring of the flight.

Pharmacists for Hospital Drug Rooms

ON OTHER OCCASIONS some references have been made to the advisability of hospitals employing pharmacists for their drug room, or having arrangements with pharmacists in the locale (if the hospital is in a smaller city) whereby drugs and medications are properly supervised. With the advent of newer, more effective, and correspondingly more dangerous drugs, such an admonishment can well be repeated and emphasized. The most recent pronouncement in this particular area comes from a case which has just received attention by one of the Courts of Civil Appeals in Texas. While the particular acts of alleged negligence will have to be determined at a later time, some of the comments from the Court, as well as some details from the factual situation involved, serve as excellent guide lines for the direction of other hospitals in our state.

Suit was brought against the particular hospital involved, a charity institution, by the parents of a little boy who was alleged to have died as the result of the negligent administration of a dose of ipecac by employees of the hospital. The child was taken to the hospital by his grandmother after having taken some tablets of an unknown origin. Upon orders from the physician in the emergency room, a dose of ipecac was given to the child by a hospital employee. The ipecac in question was in the emergency room and was marked "syrup of ipecac" although it may have been "fluid extract of ipecac." It was contended that as a result of his taking such ipecac, the child died. A second child was likewise administered ipecac during the same week at the same hospital and also died.

The hospital employee who administered the drug was not a registered pharmacist, had no training in pharmacy other than that taken during her formal nurse's training in the year 1923. There was no one on the staff of the hospital who was a registered pharmacist. Although the employee was in charge of the drug room, she did not know the difference between "fluid extract of ipecac" and "syrup of ipecac." Likewise, her deposition revealed that she did not know "whether or not the administration of straight extract of ipecac would be in any way harmful to the child to whom it was administered."

The facts likewise revealed that no control was exercised by the administrator of the hospital over the drug room. In fact, the "type bottle of ipecac" in question was "just sitting on the shelf in the drug room like any other bottle of medicine," and although the hospital employee and her assistant were supposed to break the big bottle of the drug to smaller ones, the employees had no idea who had received the "fluid extract" from the original pint bottle.

The child's parents alleged that the hospital was "guilty of administrative negligence in failing to provide adequate personnel for the operation of the hospital's pharmacy or drug room facilities and the distribution of drugs; and in failing to formulate and carry out a proper and adequate policy, with the necessary and proper rules and regulations, governing the operation of the hospital's pharmacy or drug room and the dispensing of drugs from the pharmacy or drug room. These failures, and each of them, were negligence, a proximate cause of the death of the child, and an exception to the rule of charitable immunity."

In answer to the plaintiffs' petition, the hospital alleged their immunity from liability by virtue of the charitable immunity doctrine which we have oftentimes discussed in the *Journal*. The trial court granted the hospital's motion for summary judgment (which in effect meant that there was not a sufficient fact question to be submitted to a jury or tried before the court). In reversing the action of the trial court and sending the case back to that trial court for a new trial consistent with the court's opinion, the Court of Civil Appeals made this observation:

> With these recognized principles of law applicable to the granting or refusing of summary judgments in mind, we have concluded, from the limited record before us, that appellee [the hospital] did not discharge the negative burden resting upon it of clearly showing that appellant [the deceased child's parents] did not have a cause of action against it. The charitable immunity doctrine does not protect appellee [the hospital] from liability for injuries caused by administrative negligence on the part of appellees as to non-delegable duties. . . .

Although, in this particular case, the determination of whether there was negligence

on the part of the hospital is yet to be established, the fact situation, the plaintiffs' contentions, and the Court's admonishment serve as excellent guide lines for other hospitals in Texas to protect themselves in this area. To allow the marvelous and effective drugs now available to us to be dispensed by persons unqualified to do so is to invite this type of action. If in a later case the court holds that the failure to employ a registered pharmacist was negligent and that such negligence was as a result of an administrative function, then the charitable immunity doctrine will no longer protect from this untoward result the hospitals now qualifying. When hospitals buy in large quantities for economic reasons, and it is then necessary for such drugs to be mixed, compounded, or assembled in dosage form, to allow the same to be done by those untrained in this area is to court the type of trouble exemplified in this case.

—PHILIP R. OVERTON, LL.B., Austin.

LEGAL-MEDICO INSTITUTE at Hillsboro Feb. 29 was attended by physicians and lawyers from Ellis, Johnson, Navarro, and Hill counties. Pictured above are Dr. Robert Mayo Tenery of Waxahachie, President of Texas Medical Association; Dr. Dick Cason of Hillsboro, president of the Hill County Medical Society; Buster Cole of Bonham, president of the Texas Bar Association; and Judge Sam Johnson, Hillsboro, president of the Central Texas Bar Association.

Personals

A physician who was aboard ship in Manila Harbor when the Japanese attacked the Philippines Dec. 8, 1941, has retired from the Navy and will live in Mt. Pleasant, Tex. He is *Capt. Edward T. Byrne.*

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Dr. Ralph Hanna, Austin pediatrician, was reelected vice president of United Cerebral Palsy Association's southwest region, at the national organization's 14th annual conference in Dallas. The southwest region includes Arkansas, Louisiana, Oklahoma, New Mexico, and Texas.

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New officers of the Texas Tuberculosis Association include *Drs. John A. Wiggins,* Fort Worth, president; *Travis Smith,* Abilene, first vice president; and *Robert B. Morrison,* Austin, treasurer.

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Members of the Tarrant County Medical Society heard *Mr. Robert P. Dupree,* chairman of the board and chief executive director of the Continental National Bank of Fort Worth, speak on "An Amateur Considers the Professional Spirit" at a recent meeting in Fort Worth.

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One of 25 Markle Scholars in Academic Medicine whose appointment begins in 1964 and whose field of interest is medicine will become an assistant professor effective July 1 at Baylor University College of Medicine, Houston, the medical school that will receive a $30,000 grant to support his work for the five years, 1964-1969. He is *Dr. Robert R. Carpenter, Jr.*

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Dr. J. Tom Payte of San Antonio made his debut as an amateur matador in a Ciudad Acuna, Mexico, bullring on Jan. 26. In his first fight he escaped without a bruise or scratch, but really does not expect to be so lucky every time. However, he says, the mortality rate of matadors in Mexico City is less than one-tenth of 1 per cent.

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Dr. J. Sid Jones, Waco, presented a scientific program on urological problems at the March meeting of the Hill County Medical Society in Whitney.

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Dr. George R. Herrmann was the recipient of the first honorary Jeweled Key Award of the American College of Cardiology at the 13th annual convocation in New Orleans, Feb. 15.

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The Harrison County Medical Society with the Harrison County health committee has obtained funds from city and county governments for the establishment in 1964 of a City-County Health Unit and a Mental Health

Guidance Center. The health committee includes three physicians appointed by the Society: *Drs. L. M. Redding, R. Orin Littlejohn,* and *Richard L. Berry. George McKay,* executive secretary of the Society, serves as secretary-treasurer of the Harrison County health committee. *Dr. Lake Littlejohn, Jr.,* a society member, although not a committee member, was active in the efforts to get public support of the movement to establish the health unit. *Dr. H. P. Bray* was president of the Society in 1963. *Drs. Berry* and *Orin Littlejohn* were recently elected president and secretary of the Society for 1964.

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Dr. James H. Sammons of Highlands has sent a letter to each of his patients explaining the shortcomings of the King-Anderson bill and urging each to write his Congressman to vote against it.

Nurses Encouraged To Learn External Cardiac Resuscitation

The Committee on Cadiovascular Diseases is encouraging nurses and other paramedical personnel to learn the fundamentals of external cardiac resuscitation.

The Committee believes that there are physicians in most communities who can instruct nurses and others in this technique and is suggesting to nurses' groups that they invite these physicians to demonstrate this method at their meetings.

Aerospace Medical Group Sets Miami Meet May 11-14

The 35th annual scientific meeting of the Aerospace Medical Association will be held May 11-14, 1964, at the Americana Hotel, Miami Beach, Fla. More than 2,000 of the world's specialists in aerospace medicine and allied sciences from the United States and many foreign countries are expected to attend.

The scientific program will consist of reports on research and developments on testing and operational applications of clinical aerospace medicine, life sciences, allied scientific specialties, bioaeronautics, and astronautics. Approximately 160 papers on the latest developments in clinical aerospace medicine and the allied sciences will be presented during the four-day scientific meetings, workshops, and round table discussions. The scientific sessions will be augmented by 150 scientific and technical exhibits.

Further information may be obtained by writing to Dr. William J. Kennard, executive vice president, Aerospace Medical Association, Washington National Airport, Washington 1, D. C.

American Proctologic Society To Meet in Philadelphia

The joint meeting of the American Proctologic Society and the Section of Proctology of the Royal Society of Medicine will be held at the Bellevue Stratford Hotel in Philadelphia May 9-14.

Texas physicians participating in the program include Drs. Robert J. Rowe and Curtice Rosser, Dallas; J. Arnold Bargen, Temple; James M. Hampton and Herbert T. Hayes, Houston; and Hugh Beaton, Fort Worth.

Chicago Meet to Concern Environmental Health

The American Medical Association will sponsor a national Congress on Environmental Health Problems May 1-2 in Chicago.

The Congress will deal with the physician's role in evaluating and preventing environmental health problems, with emphasis on air pollution, pesticide hazards, radiological hazards, and water supply and pollution.

Moderator for one of the panels will be Dr. John S. Chapman of Dallas.

Hospital Groups Plan Austin Gathering in May

The 35th annual Texas Hospital Association convention and exhibit show has been scheduled for May 16-20 at the Municipal Auditorium in Austin.

Allied hospital groups which will meet in Austin concurrently include the following: Texas Conference of Catholic Hospitals, May 16-17; Hospital Purchasing Agents Association of Texas, May 18-20; Texas Association of Hospital Auxiliaries, May 17-20; Texas Association of Hospital Governing Boards, May 20; Texas Association of Medical Record Librarians, May 18-20; Texas Associa-

tion of Nurse Anesthetists, May 18-20; Texas Association of Operating Room Nurses, May 18-20; Association of Catholic Chaplains in Texas Hospitals, May 18-20; Hospital Chaplains in Texas, May 18-20; Texas Association, Staff Directors of Hospital Volunteer Services, May 17.

Obstetricians, Gynecologists To Meet in Bal Harbour, Fla.

The American College of Obstetricians and Gynecologists will meet May 16-22 at the Americana Hotel in Bal Harbour, Fla.

Texas physicians listed on the advance program include the following: William F. Guerriero, Ralph Cox, Weldon Tillery, George Race, William P. Devereux, and Oran V. Prejean, Dallas; Robert R. Franklin, J. Alan Alexander, Gilbert H. Fletcher, Felix N. Rutledge, Herman L. Gardner, John A. Wall, Warren M. Jacobs, Robert A. Johnston, and Erwin O. Strassmann, Houston; William J. McGanity, Galveston; S. Foster Moore, Jr., San Antonio; and Jack L. Turner, Fort Worth.

Public Health Association Maintains Antimedicare Stand

The Texas Public Health Association has reaffirmed its position opposing medical care for the aged under Social Security and will support further implementation of the Kerr-Mills Act in Texas by adding Medical Aid for the Aged.

The action was taken at the recent meeting of the association in Austin.

Current officers of the association are president, Dr. C. A. Pigford, Houston; president-elect, Dr. W. V. Bradshaw, Jr., Fort Worth; first vice president, Dr. Albert G. Randall, Amarillo; and second vice president, Kenneth T. Crane, San Antonio.

Principles Endorsed Regarding Status of General Practitioners

The Joint Commission on Accreditation of Hospitals has recently announced its beliefs regarding the status of general practitioners.

General practitioners, the Joint Commission states, should have the opportunity to practice medicine as active staff members in hospitals and should have granted to them such hospital privileges as their training and demonstrated skills indicate.

Texans Have High Level Of Health Insurance

The people of Texas have reached a high level in health insurance protection. As of Dec. 31, 1962, 65 per cent of the state's population was covered by some form of health insurance for the costs of hospital and medical care. This compares with the national figure of 76 per cent of the civilian population—some 141,000,000 persons—having health insurance. State data follow:

Extent of Health Insurance Coverage and Other Figures		
Number of persons with hospital expense insurance		6,517,000*
Number of persons with surgical expense insurance		6,072,000*
Number of persons with regular medical expense insurance		3,991,000*
Number of persons with major medical insurance		1,717,000*
Total health insurance benefits paid (1961)		$229,626,000
By insurance companies for hospital, surgical, and medical care, including loss of income	$170,447,000	
By Blue Cross-Blue Shield and other plans for hospital, medical, and surgical care	$ 59,179,000	
Number of health insuring organizations licensed in the state (415 insurance companies, two Blue Cross-Blue Shield and similar groups, and 26 other health plans)		443
Hospital Statistics		
Number of hospitals† in state (1962)		481
Number of hospital beds available (1962)		32,823
Number of persons admitted during the year (1962)		1,349,013
Number of persons under hospital confinement on an average day		22,908
Average length of time patient remained in hospital		6 days
Average cost per hospital stay		$210.61
Professional Services		
Number of practicing physicians (1962)		7,770
Number of practicing dentists (1962)		3,640
Number of registered pharmacists (1962)		6,038

*Net total of people protected—eliminates duplication among persons protected by more than one kind of insuring organization or more than one insurance company providing the same type of coverage.

†Nonfederal, short-term general and other hospitals.

Rehabilitation Symposium Announced for May 7-8

A physical rehabilitation symposium is being sponsored May 7-8 by the Texas Employers' Insurance Association on the occasion of its 50th anniversary. This symposium will be held at the Statler Hilton Hotel in Dallas.

A full two-day program with speakers and panelists who are acknowledged leaders in their respective fields has been planned. The program will emphasize recent clinical advances in rehabilitation as well as related administrative and financial areas.

Persons desiring additional information may write to Smith Pettigrew, Manager of Medical Services, Texas Employers' Insurance Association, P. O. Box 2759, Dallas 21.

Over 18,000 Claims Paid By Dependents' Medical Care

During the calendar year 1963, 18,719 claims were paid to Texas physicians for a total dollar value of $1,606,077.87, according to B. H. Patterson, administrator of Dependents' Medical Care Program, Mutual of Omaha.

In a progress report to C. Lincoln Williston, Executive Secretary of Texas Medical Association, Patterson stated that this brings total payments to physicians in Texas since May 1, 1958, the date his company assumed responsibility for physicians' payments, to 130,150 claims and $11,243,714.62.

"Every effort is made to provide the physicians with fast, accurate claim service," he concluded.

Informative Pamphlet Goes To Association Members

Well over 200,000 copies of "Texas Leads the Way in Providing Medical Care for the Aged" have been sent to members of the Texas Medical Association from the central office.

The pamphlet is available in quantity, without cost, for placing in waiting rooms or for other distribution. It cites the positive measures which have been initiated in Texas to provide an effective program of medical care for the needy aged and to assist in financing health services for Texas' 745,000 residents who are 65 years of age or older.

New Books in TMA Library

Ackerman, L. V.: Surgical Pathology, 3rd ed., 1964, St. Louis, C. V. Mosby Co., 1964.

Adriani, J., Ed.: Appraisal of Current Concepts in Anesthesiology, St. Louis, C. V. Mosby Co., 1964.

Alesen, L. A.: Mental Robots, Caldwell, Idaho, The Caxton Printers, Ltd., 1957.

Wilson, C. O., and Jones, T. E.: American Drug Index 1963, Philadelphia, J. B. Lippincott Co., 1963.

Ball, T. L.: Gynecologic Surgery and Urology, 2nd ed., St. Louis, C. V. Mosby Co., 1963.

Blechschmidt, E.: Stages of Human Development Before Birth, Philadelphia, W. B. Saunders Co., 1961.

Fabricant, N. D.: Why We Became Doctors, New York, Grune & Stratton, 1954.

Geschickter, C. F., and Cannon, A.: Color Atlas of Pathology; Central Nervous System, Philadelphia, J. B. Lippincott Co., 1963.

Gilmer, W. S., Jr., et al.: Atlas of Bone Tumors, St. Louis, C. V. Mosby Co., 1963.

Goth, A.: Medical Pharmacology, 2nd ed., St. Louis, C. V. Mosby Co., 1964.

Hanlon, J. J.: Principles of Public Health Administration, 4th ed., St. Louis, C. V. Mosby Co., 1964.

Humphrey, J. H., and White, R. C.: Immunology for Students of Medicine, Philadelphia, F. A. Davis Co., 1963.

LeMesurier, A. B.: Hare-Lips and Their Treatment, Baltimore, Williams & Wilkins Co., 1962.

Merck & Co., Inc.: By Their Fruits, West Point, Pa., Merck Sharp & Dohme Research Laboratories, 1962.

Morley, M. E.: Cleft Palate and Speech, Baltimore, Williams & Wilkins Co., 1962.

Moseley, J. E.: Bone Changes in Hematologic Disorders, New York, Grune & Stratton, 1963.

Overzier, C., Ed.: Intersexuality, New York, Academic Press, 1963.

Rank, B. K., and Wakefield, A. R.: Surgery of Repair as Applied to Hand Injuries, 2nd ed., London, E. & S. Livingstone, Ltd., 1960.

Shirkey, H. C., Ed.: Pediatric Therapy, St. Louis, C. V. Mosby Co., 1964.

Starrett, R. S., Find a Career in Medicine, New York, G. P. Putman's Sons, 1960.

Sturgis, S. H., et al.: Gynecologic Patient, New York, Grune & Stratton, 1962.

Beckman, H., Ed.: Year Book of Drug Therapy, 1963-1964, Chicago, Yearbook Medical Publishers, 1964.

Book Notes

Books reviewed in this column are available from TMA Memorial Library.

Of Many, One. John Mastin Travis, M.D. 114 pp. Jacksonville, Tex., Kiely Printing Company, 1963. $2.50.

AS A RULE I do not care for autobiographies because they usually leave out the most interesting portion of the writer's life. When the story is written by a man of the stature of Dr. John Mastin Travis of Jacksonville

it is a different story. This is contemporary history of a great period in Texas medical history and leaves a record for posterity that is a benefit to all of us.

Living now in this country so bound by governmental restrictions, the rugged individualist like Dr. Travis is a thing of the past. These men thrive on frontiers without restrictions which no longer exist. Dr. Travis's life story written by him is entitled "Of Many, One." It makes an evening of good reading. I would recommend this book to the many friends of Dr. Travis and anyone else interested in Texas medical history from 1900 to 1960.

There are a few mistakes which are trivial and do not detract from the book. In a note at the beginning, Dr. Travis states, "In presenting this story, I wish to say that all the information contained in it is from memory with a few exceptions." Being from Fort Worth I shuddered to read on page 69, "Much of my indoctrination into the workings of the Texas Medical Association came through my association with the late Holman Taylor, who was secretary and 'prime minister' for many years. His term of office came to a tragic end in Dallas a number of years ago when he died suddenly on the platform during a recognition dinner in his honor." Holman Taylor died on the speakers platform in the Crystal Ballroom of Hotel Texas in Fort Worth in 1947. We folks in Fort Worth are beginning to get used to this sort of error by now.

This is a readable book about a good doctor and a man of character during the pioneer period of Texas medical history.

—William M. Crawford, M.D., Fort Worth.

Results of Surgery for Peptic Ulcer. R. W. Postlethwait, M.D., and James C. Thoroughman, M.D., Eds. 308 pp. Philadelphia, W. B. Saunders Co., 1963. $8.

IN A DETAILED RETROSPECTIVE STUDY, the authors surveyed nearly 3,000 patients operated on for peptic ulceration in a selected group of VA hospitals during the period 1947-1957. They have correlated every available pertinent factor concerning the patient, the disease, the operation, and the result. This is presented in more than 225 tables and figures. While this is not an easily readable book, its value as a teaching reference and a statistical survey is unquestioned.

In many instances, one's clinical impressions are confirmed by the authors' statistics. In others, the reader will be surprised. Examples are: 38.4 per cent of the patients operated on for perforated peptic ulcer later required definitive ulcer surgery; in patients over 50 years of age requiring emergency surgery for bleeding gastric ulcer, that mortality rate was 52.2 per cent, and finally, the mortality rate for gastric resection with vagotomy was less than that for gastric resection alone!

While the authors raise many questions concerning the management of peptic ulceration, they make no attempt to present solutions. A prospective study is planned for the future in any effort to resolve many of the problems defined. Until that report is available, "Results of Surgery for Peptic Ulcer" belongs on the bookshelf of everyone who deals with the problem.

—Walter C. Barnes, Jr., M.D., Texarkana.

Practical Anesthesiology. Joseph F. Artusio, Jr., M.D., and Valentino D. B. Mazzia, M.D. 318 pp. St. Louis, C. V. Mosby Co., 1962. $7.75.

THIS BOOK, as the foreword states, is a fine reference for the occasional anesthetist or the medical student. It contains generally accepted methods and ideas concerning anesthesia which should be common knowledge to all who give anesthesia. All of the more frequently used agents are described and the indications and contraindications are listed. The newer halogenated agents are mentioned but not discussed as these are not drugs to be used by those untrained in their use. The book is divided into five parts: basic considerations, preanesthetic considerations, anesthetic considerations, techniques of administration, and special considerations. Each part is short and well written. Those who choose to use this book will enjoy it.

—Ann T. Bouldin, M.D., Galveston.

Experimental Pharmacodynamics. T. Koppanyi and A. G. Karczmar. 3rd ed. 246 pp. Minneapolis, Minn., Burgess Publishing Company, 1963. $6.50.

PURPOSE OF THE BOOK is to develop a scientific foundation for the use of chemical agents in the prevention, diagnosis, and treatment of disease. The authors' further purpose was to prepare a text that would serve not only as a conventional laboratory manual, but as an introduction to pharmacology, supplementing the textbooks on the subject.

Recent Deaths

DR. P. HUBBARD, JR.

Dr. Prevost Hubbard, Jr., died Dec. 9 in Valhalla, N. Y. The Odessa physician had left last June to take a residency in New York.

Born in 1914 to Prevost and Birdie (Beuler) Hubbard, he attended high school in White Plains, N. Y., Antioch College in Yellow Springs, Ohio, and Springfield College in Springfield, Mass. He was graduated from the University of Maryland School of Medicine in Baltimore in 1942 and completed his internship at French Hospital in New York City. He served surgery residencies at South Baltimore General Hospital in Baltimore and St. Paul's Hospital in Dallas.

Dr. Hubbard was a captain in the Army Air Force Medical Corps in World War II.

After beginning his practice in Schulenburg, he moved to Hamilton in 1948, serving as secretary of the Hamilton County Medical Society (now Bosque-Hamilton Counties Medical Society) in 1949. He had been practicing in Odessa since 1950.

He married Elizabeth Jane Lawson in 1959. She died two years later.

Dr. Hubbard was a member of the Andrews-Ector-Midland Counties Medical Society, Texas Medical Association, American Medical Association, American Legion, and Veterans of Foreign Wars.

His hobbies were fishing, hunting, and skin diving.

Survivors include his father; a brother, Andre Hubbard of Long Island, N. Y.; two sisters, Barbara Hubbard of White Plains, N.Y., and Mrs. Nancy M. Law, South Salem, N.Y.; a son, Prevost Hubbard, III, of Houston; and two daughters, Rebecca Jane and Clair Renee Hubbard, both of Odessa.

DR. E. A. JONES

Dr. Elmer Arthur Jones, 44, died of a heart attack Dec. 30 in his quarters at the Veterans Administration Center in Temple. Dr. Jones had been appointed Surgeon, chief grade, at the Center last year.

He was born in Iron City, Tenn., in 1919, the son of Arthur and Maggie May Clark Jones. After attending Humboldt High School in Humboldt, Tenn., and Memphis State College, he went to the University of

DR. E. A. JONES

Tennessee from which he received his M.D. degree in 1946. He interned at Baptist Memorial Hospital in Memphis and served his residency at Good Samaritan Hospital in Portland, Ore., and Methodist Hospital in Dallas.

He married Idell Lasiter of Longview in 1944 at Memphis, Tenn. They had two children, Ann and Arthur.

Before going to the VA Center in 1959, he was in private practice in Albany, Ore., for five and one-half years as a general surgeon. He was president of the Albany Hospital staff and an elected city councilman during 1958.

He was a member of the Albany Chamber of Commerce, the Jaycees, and the Elks Lodge. He was president of the Albany Lions Club and served as a member of the board of directors of the Community Concert Association and the YMCA.

His chief hobby was flying and while in Albany, he was president of the Oregon Pilots' Association.

He served as a captain in the Army from 1947 to 1949 in Wakayama, Japan. In Temple Dr. Jones was a member and deacon of the First Presbyterian Church, a member of the Lions Club, and a major in the Civil Air Patrol.

He was a member of the Texas Medical Association and the American Medical Association.

In addition to his wife and children, Dr. Jones is survived by his mother and a sister, Mrs. H. T. Williams, both of Nashville, Tenn., and a brother, C. F. Jones of Hendersonville, Tenn.; and several nieces and nephews.

DR. A. COLLETTE

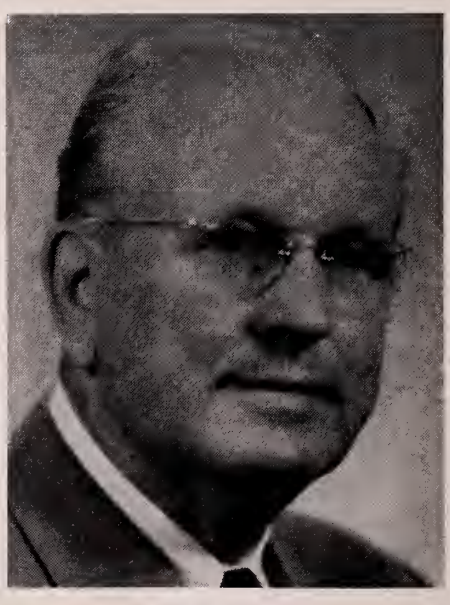
Dr. Allan Collette, professor emeritus at Baylor University College of Medicine, died Jan. 4 at Methodist Hospital in Houston after a brief illness.

The 73-year-old physician had retired from private practice in Houston last July, remaining active on the Board of Civil Service Examiners and as the physician in charge of the medical unit, U. S. Postal Service, Houston.

Born in De Pere, Wis., in 1889, he received a Ph.G. from the University of Mississippi in 1916 and his M.D. degree from the University of Tennessee in 1918.

During both world wars, Dr. Collette served in the U. S. Navy Medical Corps. He was discharged from active service in World War II with the rank of captain. He served as chief of medical office procurement and as Naval examiner for Naval R.O.T.C., Rice University and the University of Texas. He saw service in the South Pacific area from 1942 to 1946 on hospital ships. His last assignment was in the headquarters of Admiral Chester Nimitz on Guam.

Moving to Houston in 1924 from Laurel, Miss., he helped found the School of Pharmacy and the student health service at the University of Houston. He taught in the pharmacy school from 1946 to 1960, also serving as physician for the University football teams. He was professor emeritus in pharmacy at the University when he died.



DR. A. COLLETTE

At Baylor University College of Medicine, he taught obstetrics and gynecology. He was on the board of directors of the Andy Anderson Memorial Fund.

Dr. Collette was a member of the First Baptist Church and the Downtown Rotary Club, and his medical affiliations included the Southern Medical Association, American Society of Abdominal Surgeons, Harris County Medical Society, the Texas Medical Association, the American Medical Association, American College of Surgeons, American Geriatrics Society, Texas Society of Athletic Team Physicians, American Association of Railway Surgeons, Texas Railway and Traumatic Surgical Association and American College of Sports Medicine.

Survivors include his wife, Mrs. Cherry A. Collette; daughter, Mrs. Carl Mengden of Comfort; son, Allan Collette, Jr., of Houston; sister, Mrs. George Engeldinger of Portland, Ore.; and three grandchildren.

DR. W. L. HOLLISTER

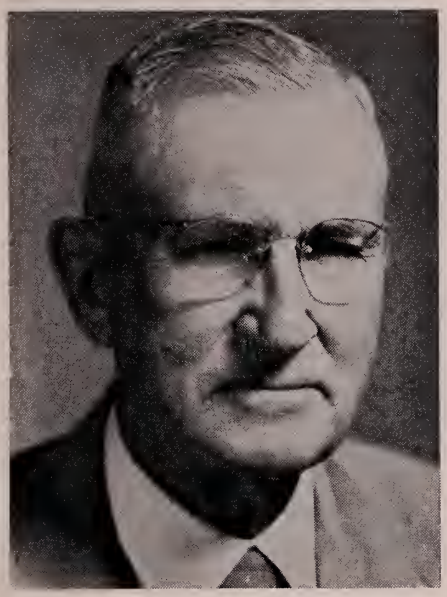
A San Angelo physician, Dr. Wilber Leeper Hollister, died Oct. 11 from a cerebral hemorrhage.

Dr. Hollister was born in DeWitt, Mo., on March 7, 1875, to William Spencer and Mary Frances McKinney Hollister. He received his premedical education from the University of Missouri in Columbia and his M.D. degree from Keokuk Medical College, College of Physicians and Surgeons in Keokuk, Iowa, in 1903.

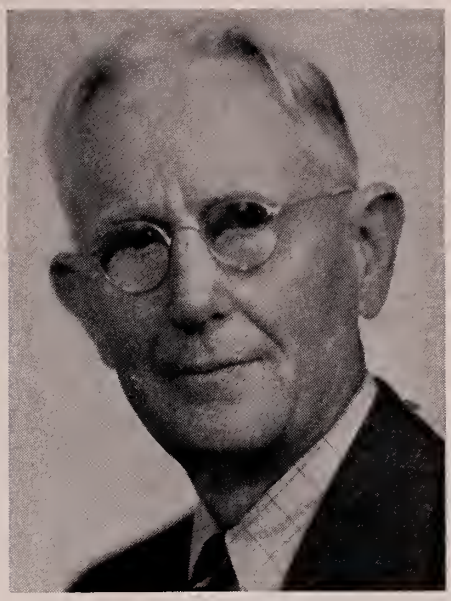
After practicing in Kansas City, Mo., 29 years and serving as a major in the U. S. Army Medical Corps, he came to Texas. From 1939 to the time of his death, Dr. Hollister had practiced in Corpus Christi, Uvalde, Nixon, Palmer, Miles, and San Angelo.

He was a member of the medical staff of St. John's Hospital in San Angelo from 1952 to 1960. He continued his private practice until a few months ago.

In 1905, he married Mrs. Minnie Dickson Brookover who preceded him in death. He



DR. W. L. HOLLISTER



DR. J. CAMP

married Drucie Tyler of Miles in 1955 at San Angelo.

Elected to inactive membership in the Texas Medical Association in 1953, Dr. Hollister was a member of the Tom Green-Eight Counties Medical Society and the American Medical Association.

He was a Mason and a member of the First Evangelical Methodist Church of San Angelo.

Survivors include his wife; a brother, George Hollister of DeWitt, Mo.; and three sisters, Mrs. Lutie Lee of Oklahoma City, Mrs. Elizabeth Brandom of Excelsior Springs, Mo., and Mrs. Ada Morris of Visalia, Calif.

DR. J. CAMP

A horse and buggy physician and General Practitioner of the Year in 1950 died on Jan. 22 in Pecos of lymphosarcoma.

Dr. Jim Camp, 86, one of two doctors between Midland and El Paso at one time, came to Pecos in 1900 only a few days after graduating third in his class at the University of Tennessee College of Medicine. He was born in Doyle Station, Tenn., in 1877 to Miles Newton and Elizabeth (Gillentine) Camp.

One of his first callers in Pecos was a young cowboy suffering from appendicitis. Dr. Camp performed on a kitchen table what is believed to be the first appendectomy in Pecos.

The 23-year-old physician made calls on a bicycle while longer trips were made on horseback, in buggies, and even by freight trains. These modes of transportation were replaced by a car in 1915.

His medical equipment consisted of a stethoscope, thermometer, hypodermic syringe, pocket instrument case, and black bag, according to a report in the *Pecos Daily News*.

In 1901, Virgie Maud Stroud of Simpson Mills, Tenn., came to the home of her sister in Lancaster where she and Dr. Camp were married on Oct. 1.

During World War I, Dr. Camp served as a first lieutenant in the Medical Department. He was on his way to Maryland for overseas assignment when the Armistice was signed.

When the flu epidemic hit Pecos and Reeves Counties, Dr. Camp returned on an emergency ten-day furlough and was discharged shortly after returning to the Army.

He built the first hospital in Pecos in his home and later erected the Camp Hospital. When the Reeves County Memorial Hospital was built in 1954, he became a member of its staff and closed his hospital.

During his career, he was surgeon for both the Texas and Pacific and the Santa Fe railroads, district surgeon for the Texas Highway Department, and surgeon for the U. S. marshal at Pecos.

In 1950, the Pecos community honored him by proclaiming a day in his honor. In 1961, the Southern Medical Association tendered him an award for his 45 years of service, and the University of Tennessee present-

ed him with a gold key award for being an outstanding alumnus.

In addition to the Texas and Southern Medical Associations, Dr. Camp was a member of the Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Medical Society, Texas Railway Surgeons' Association, Academy of General Practitioners, and the American Medical Association.

An elder in the First Christian Church for many years, he was also a charter member of the Rotary Club, a member of the Pecos Chamber of Commerce, and active in both Democratic Party activities and local school affairs. He held every office in the Pecos Valley Masonic Lodge, serving as Worshipful Master in 1910.

In addition to his wife, he is survived by a son, Keith Camp, Pecos; a daughter, Mrs. Bob Dean, Pecos; a brother, Dr. Will Camp, Rock Island, Tenn.; and one sister, Mrs. A. G. Bowles, Tulsa, Okla.; seven grandchildren; and several nieces and nephews. A son, Dr. Hilliard Camp, died in 1941.

DR. P. H. SCARDINO

Former chief of staff of old Jefferson Davis Hospital and St. Joseph's Hospital, Dr. P. H. Scardino, 80, died Jan. 27 in Houston.

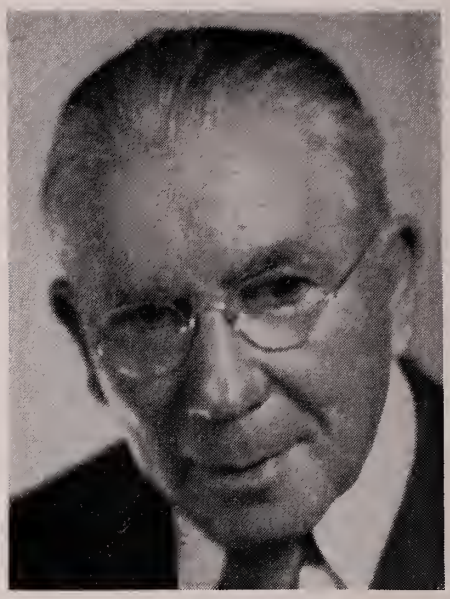
After a career that won him honors from two governments, Dr. Scardino retired in 1948, having practiced medicine in Houston 41 years.

Born in 1883 in Poggioreale, Sicily, to Vito Scardino and Nunzia Culmone Scardino, Dr. Scardino came with his parents to America when he was seven years old.

He attended the old Houston High School and St. Thomas Academy, now the University of St. Thomas. Graduating from Tulane University School of Medicine in 1907, he was first assistant surgeon at St. Joseph's Hospital from 1912 to 1916 and chief surgeon of a municipal hospital from 1916 to 1918. He was city health officer from 1916 to 1918.

He won the Italian War Cross serving as a captain in the Medical Corps on the Italian front. On returning to Houston, he was appointed Italian vice consul, and for his services to both America and Italy, he was knighted by King Umberto of Italy.

He married Maud Muller of Houston in 1910.



DR. P. H. SCARDINO

Dr. Scardino was elected to honorary membership in the TMA in 1951. He was a member of the Harris County Medical Society, the South Texas Medical Association, the American Medical Association, fellow of the American College of Surgeons, the International College of Surgeons, Southern Medical Association, and South Texas Postgraduate Medical Assembly.

He was an early member of the River Oaks Country Club where he became an avid golfer. He was also a member of the American Legion and the Catholic Church.

Dr. Scardino is survived by his wife; three sisters, Mrs. Ralph West, Mrs. Canessa Dominek, and Mrs. J. P. Callahan; and one brother, James Scardino, all of Houston.

DR. H. POWELL

Dr. Homer Powell died of myocardial infarction on Sept. 9 in San Antonio.

Born in Brownwood Oct. 4, 1889, his parents were Allen A. and Betty Powell. After receiving his preliminary education in Kentucky, he attended Texas Christian University in Fort Worth and Southern Methodist University in Dallas, where he was graduated in pharmacy in 1913. He graduated from Vanderbilt University School of Medicine in 1920 and served his internship at Nashville General Hospital and the Army hospital at Fort Sam Houston. He practiced general medicine and functioned as chief of

DR. H. POWELL

neuropsychiatric section in the Veterans Administration Hospital and regional office in Dallas and Waco from 1920 to 1944. He moved to San Antonio from California in 1948.

Dr. Powell was a lieutenant colonel in the Army, having served in the cavalry in the Philippine Insurrection, in the European theater during World War I, and the Fourth Army in World War II.

He had done postgraduate work in neuropsychiatry at New York Clinic and the University of California.

He married Selma Kelm in 1946 at Richmond, Calif.

His medical affiliations include the Dallas County Medical Society, the Southern Medical Association, the American Medical Association, the National Association of Retired Civil Employees, Disabled Officers Association, American Association of Physicians and Surgeons, Association of Military Surgeons, and the Retired Officers Association. He was elected to honorary membership in the Texas Medical Association in 1948.

At Vanderbilt, he was a member of Delta Omicron Alpha.

He was a charter member of the American Legion No. 404 and a member of St. Matthew Lutheran Church, San Antonio.

He is survived by his wife, Mrs. Selma Powell, and a sister, Mrs. Judy Webb, Burkesville, Ky.

DR. Z. T. SCOTT

Dr. Zachary Thomson Scott, 83, died Jan. 19 in Austin after an illness of several weeks.

A student at the University of Texas Medical Branch in Galveston during the storm of 1900, Dr. Scott is said to have rescued 200 persons by carrying them in waist and neck-deep water to more substantial houses. The account is written in the "The Great Galveston Disaster."

Born in Fort Worth in 1880 to R. L. and Abby Boyle Scott, Dr. Scott attended public schools in Bosque, Tex., and college at Belair School in Lewiston, Va. He was graduated from medical school in 1903, and between 1905 and 1929, he attended postgraduate clinics in New York, Chicago, and Vienna.

He married Sallie Lee Masterson of Fort Worth in 1909 at Truscott, Tex. They had three children, film star Zachary Thomson Scott, Jr., Mrs. Fanning Hearon of Tryon, N. C., and Mrs. Richard Kleberg, Jr., of the King Ranch.

Dr. Scott practiced in Clifton after graduating from medical school, but moved to Austin in 1909 where he purchased one-half interest in the old Austin Sanitarium. His partner was Dr. Thomas J. Bennett.

During his years in Austin, Dr. Scott founded the Scott-Gregg Clinic and was a director of the Texas Tuberculosis Association. He was instrumental in starting the first Christmas Seal sale, a project that now raises a million dollars annually for research and operation of hospitals.

In World War I, Dr. Scott was in charge of a hospital unit which he organized in Travis County and moved to the Naval Hospital at Gulfport, Miss. He had the rank of lieutenant commander.

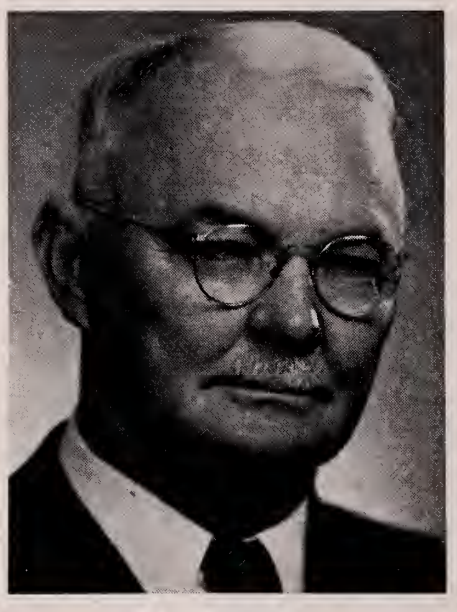
He retired from active practice in 1947 and was elected an honorary member of TMA in 1953. He was a member of the Travis County Medical Association and the American Medical Association.

Interested both in banking and ranching, Dr. Scott was a director of the Capital National Bank in Austin. After retiring from medical practice, he began raising and developing a breed of cattle known as "San Gerfords" on a farm near Buda, Tex.

He was a member of the Austin Selective Service Board in World War II.

He was an officer and member of the University Presbyterian Church.

The Scotts were prominent in the pur-



DR. Z. T. SCOTT

chase and removal of the O. Henry honeymoon cottage to Pease Park in Austin in the interest of the Heritage Society, of which Dr. and Mrs. Scott were charter members. In 1929, they moved and restored "Sweetbrush," the old John Swisher house. Swisher was the only treasurer of the Republic of Texas.

In addition to his wife and children, Dr. Scott is survived by a sister, Mrs. William Kimbrough of Itta Bena, Miss., eight grandchildren, and four great-grandchildren.

DR. A. M. LONG

Dr. Austin M. Long, a Valley Mills resident, died of a heart attack Dec. 29 while visiting in Dallas.

Interested in both medicine and education, Dr. Long had served as Vice President of the Texas Medical Association in 1939-1940 and president of the State Board of Education in 1946. He was a member of that board from 1943 to 1950.

Born in Lampasas in 1904, the son of Austin M. and Mattie Berry Long, Dr. Long attended Lampasas High School and Baylor University. He graduated from the University of Kansas School of Medicine in June, 1932, and served his internship at St. Margaret Hospital in Kansas City.

On the same day he graduated from medical school, he married Gail Hamilton of Waco.

Moving to Valley Mills in 1933, he was appointed city health officer. In 1947 he established the Long-Koerner Clinic and Laboratory which became the Long Clinic after a few years. He held the position of county health officer from 1936 to 1938.

Dr. Long was a member of the Texas Medical Association, the American Medical Association, and the Bosque-Hamilton Counties Medical Society. He was president of the former Bosque County Medical Society in 1935 and again in 1944.

He served as chairman of the board of directors of the First National Bank of Valley Mills, and he was president of the Valley Mills School Board at the time of his death. He was a member of the Baptist church, Valley Mills Chamber of Commerce, and Karem Shrine Temple in Waco.

Survivors include his wife; one daughter, Mrs. James R. Butler of New York; two sons, John H. Long of Largo, Fla., and Monty Long of Waco; and five grandchildren.

DR. J. A. HALAMICEK

Dr. John A. Halamicek of El Campo died Feb. 10 in a Houston hospital following a long illness.

Born on March 10, 1886, at Roznov, he was the son of John M. and Anna (Baron) Halamicek. He was a 1903 graduate of Sam Houston Normal and a 1908 graduate of the University of Tennessee College of Medicine. He had practiced medicine in El Campo from 1921 until his retirement in 1954. Prior to

DR. J. A. HALAMICEK

moving to El Campo, he had practiced in Nada and Garwood.

He had done postgraduate work at Tulane University School of Medicine and Cook County Hospital, Chicago.

He was a member of the Wharton-Jackson Counties Medical Society, Texas Medical Association, and American Medical Association.

He married Frances Frnka of Nada in 1908. She died in 1933. In 1935 he married Vlasta Dressler of El Campo.

Besides his wife, he is survived by two sons, Dr. John F. Halamicek and Frank A. Halamicek of El Campo; two daughters, Miss Arlene S. Halamicek of Houston and Mrs. Evelyn Sublett of El Campo; five sisters, Mrs. Charlie Havlik of Sweet Home, Mrs. Annie Kopecky of El Campo; Mrs. Charlie Hilsher of Park, Mrs. Henry Teplicek of Rowena, and Mrs. Claude Lancaster of Hamlin; two brothers, Dr. Henry Halamicek of El Campo and Ben Halamicek of Roznov; ten grandchildren; and two great-grandchildren.

DR. P. B. STOKES

Dr. Paul Bacon Stokes died Feb. 19 at his home in Crockett after a long illness. Death resulted from liver failure and congestive heart failure.

He was born May 11, 1899, in Crockett, the son of Edgar Bacon and Cora (Davis) Stokes. He attended the Crockett schools, Southwestern University, Georgetown, and Southern Methodist University where he was a member of the varsity football and baseball teams. He was graduated from the University of Texas School of Medicine at Galveston in 1924.

He interned at the Alameda County Hospital in Oakland, Calif. Following this, he entered the practice of urology in Wichita Falls. He moved from there to Crockett in 1932 where he remained in the practice of general medicine and surgery until his death.

Dr. Stokes was elected to fellowship in the American College of Surgeons in 1947. He was a member of the Houston County Medical Society, the Texas Medical Association, and American Medical Association.

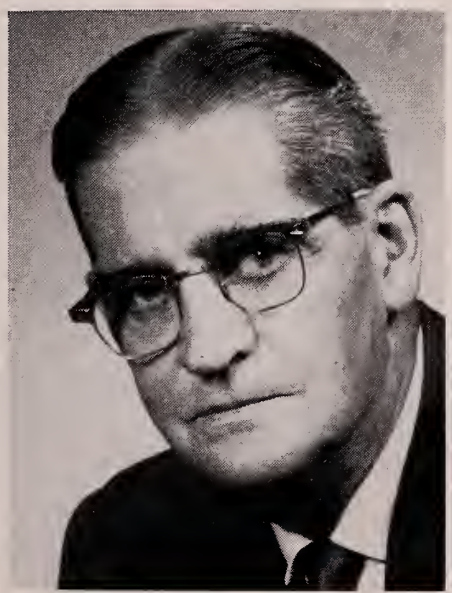
A Methodist, he was a charter member and first president of the Crockett Rotary Club, a 32nd Degree Mason, a Shriner, and a member of the Pi Kappa Alpha fraternity. He was interested in electronics.

On Feb. 5, 1932, he married Clarite Elliott of Crockett in Houston. She survives as does his daughter, Mrs. Jamie Dawson of Longview. Others are his mother, Mrs. Cora Stokes, and three sisters, Mrs. R. E. Cornelius and Mrs. Smith B. Wootters of Crockett, and Mrs. E. A. Ellison of Tyler.

DR. G. G. HENNING

Dr. Garold Graham Henning, a resident of San Antonio for 18 years, died Feb. 18 in Brooke General Hospital, Fort Sam Houston. Death was attributed to cirrhosis of the liver

DR. P. B. STOKES



DR. G. G. HENNING

with complications and upper gastrointestinal hemorrhage.

He was born in Platteville, Wis., in 1903. after receiving his B.S. degree from the State University of Iowa in Iowa City in 1926, he attended the State University of Iowa Medical School in Iowa City, receiving his M.D. degree in 1929. He served his internship at the State University of Iowa Hospitals in Iowa City and a residency at Children's Hospital in Los Angeles and the State University of Iowa Children's Hospital. He took postgraduate courses at Barnes Hospital in St. Louis. After practicing in Norfolk, Neb., and Milford, Iowa, he entered the Army reserves medical corps in January, 1940, at Fort Snelling, Minn. Before his year of reserve service expired, war was declared and he remained in active service until Feb. 24, 1946, serving in the Third and 96th Divisions, and was separated with the rank of lieutenant colonel. His medical battalion was the first to land on Leyte Island under Japanese fire. He came to San Antonio in 1946. He specialized in diseases and surgery of children.

His medical affiliations included the Bexar County Medical Society, Texas Medical Association, and American Medical Association.

He was also a member of Grace Presbyterian Church, Alamo Heights Lions Club, and Gloaming Lodge No. 482, AF&AM, in Milford, Iowa.

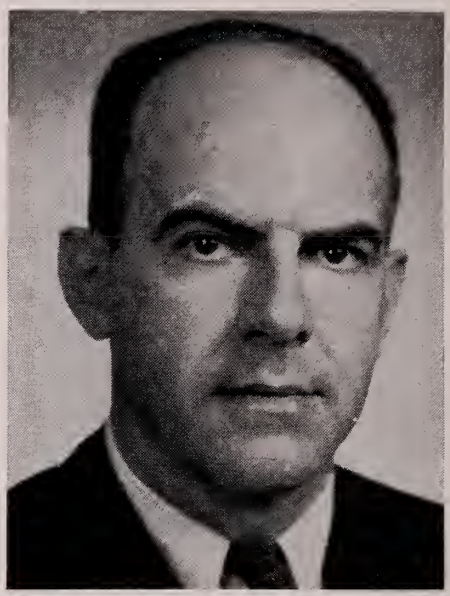
Survivors include his wife, Mrs. Ada Henning of San Antonio; a son, Garold N. Henning, Richardson; a daughter, Mrs. Robert Hundley, Jr., Bigfoot, Tex.; three brothers; and five grandchildren.

DR. A. A. EISENBERG

A clinical instructor of dermatology at the University of Texas Southwestern Medical School, Dr. Alex A. Eisenberg, died Jan. 19 in Dallas. He had practiced in Dallas since 1947.

He was born in Washington, D. C., in 1915 to Dr. Arthur A. and Julia Berlin Eisenberg.

Receiving his B.S. degree from New York University in 1937, he attended New York



DR. A. A. EISENBERG

University College of Medicine, graduating in 1942. He served his internship and residency at City Hospital, New York, and completed a preceptorship with Dr. Arthur Schoch in Dallas in 1948. In 1948-1949 he completed his specialty training at Los Angeles County General Hospital, Los Angeles.

From 1943 to 1946, he held the rank of captain in the Army. He was discharged as a major in the National Guard, 49th Armored Division. At the time of his death he was consultant in dermatology at the VA Hospital. He was a former consultant at Sheppard Air Force Base in Wichita Falls.

Dr. Eisenberg was a member of the Big Brothers Club, a lay reader and member of St. Paul's Episcopal Church, a former member of Bishop Mason's executive committee for the Episcopal Diocese of Dallas, and past president of the Wynnewood Lions Club. His hobbies were tennis, the arts, and gardening.

His medical affiliations included the Dallas County Medical Society, Texas Medical Association, Southern Medical Association, American Medical Association, Dallas Dermatological Society, and Texas Dermatological Society.

Survivors include his wife; two daughters, Miss Julie Eisenberg and Miss Caryle Eisenberg; and a son, Marc Edmond Eisenberg, all of Dallas.

Coming Meetings

American Medical Association, Annual Meeting, San Francisco, Calif., June 21-25, 1964. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, Houston, Annual Session, April 23-26, 1964; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

APRIL

American Academy of General Practice, Atlantic City, April 11-17, 1964. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 12, Exec. Sec.

American Academy of Pediatrics, Philadelphia, April 20-22, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association for Thoracic Surgery, Montreal, Canada, April 27-29, 1964. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American College of Physicians, Atlantic City, April 6-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American Gastroenterological Association, Dallas, April 24-25, 1964. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N.C.

American Surgical Association, Hot Springs, Va., April 1-3, 1964. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., Chicago, April 3-4, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Dallas, April 23, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Southwest Allergy Forum, Hot Springs, Ark., April 5-7, 1964. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwestern Surgical Congress, San Antonio, April 26-29, 1964. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

Texas Air-Medics Association, Houston, April 25-26, 1964. Dr. C. F. Miller, Box 1338, Waco, Sec.

Texas Association of Public Health Physicians, Houston, April 23, 1964. Dr. W. S. Brumage, 2600 Westover Rd., Austin, Sec.

Texas Chapter, American Academy of Pediatrics, Houston, April 24, 1964. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, Houston, April 26, 1964. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Houston, April 25, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, Houston, April 26, 1964. Dr. John W. Chriss, 2436 Morgan, Corpus Christi, Sec.

Texas Industrial Medical Association, Houston, April 23, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197. Houston, Sec

Texas Neuropsychiatric Association, Houston, April 26, 1964. Dr. E. I. Bruce, Jr., 1014 Strand, Galveston, Sec.

Texas Ophthalmological Association, Houston, April 25-26, 1964. Dr. Harold Hunt, 150 Eighth St., S.E., Paris, Sec.

Texas Orthopaedic Association, Houston April 25-26, 1964. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, Houston, April 24-25, 1964. Dr. J. P. Moran, 3154 Reid Dr., Corpus Christi, Sec.

Texas Physical Medicine and Rehabilitation Society, Houston, April 23-26, 1964. Dr. D. Hoehn, Doctor's Bldg., Fort Worth, Sec.

Texas Proctologic Society, Houston, April 23, 1964. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Society of Anesthesiologists, Houston, April 24-26, 1964. Dr. L. Duflot, 173 San Marino, Galveston, Sec.

Texas Society of Athletic Team Physicians, Houston, April 23, 1964. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section. Houston, April 26, 1964. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Pathologists, Inc., Houston, April 26, 1964. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, Houston, April 23, 1964. Dr. W. C. Sellman, Jr., 610 Medical Arts Bldg., Dallas, Sec.

Texas Surgical Society, Waco, April 5-7, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, Houston, April 23, 1964. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

MAY

American Association of Plastic Surgeons, Chicago, May 13-16, 1964. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N.Y., Sec.

American College of Obstetricians and Gynecologists, Miami Beach, Fla., May 17-22, 1964. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American Gynecological Society, Hot Springs, Va., May 25-27, 1964. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Medical Women's Association, Roanoke, Va., May 31-June 2, 1964. Dr. Jessie Brodie, 1790 Broadway, New York 19, Exec. Sec.

American Proctologic Society, Philadelphia, May 10-14, 1964. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

International College of Surgeons, Vienna, Austria. May 11-16, 1964. Dr. W. F. James, 1615 Lake Shore Dr., Chicago, Sec.

South Texas District, American College of Surgeons, Corpus Christi, May 15-16, 1964. Dr. Sanford Glanz, 2425 Medical Center, Corpus Christi, Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, San Francisco, Calif., June 18-22, 1964. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Oct. 2-3, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Hawaii, June 27-July 2, 1964. Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Neurological Association, Atlantic City, June 15-17, 1964. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

Medical Society of United States & Mexico. Dr. C. Kalil, 200 East Monterey Way, Phoenix, Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Regional Cancer Conference. Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

State

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists. Dr. H. W. Savage, 815 Fifth Ave., Fort Worth, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas State Board of Examiners in Basic Sciences, April, 1964. Mrs. Maria P. Hathaway, 1012 State Office Bldg., Austin, Exec Sec.

Texas State Board of Medical Examiners. Fort Worth, June 22-24, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Tuberculosis Association, Houston, March 18-20 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. B. C. Bates, 1108 Nueces, Austin, Sec.

United States-Mexico Border Public Health Association, Monterrey, Mexico. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea. Scott and White Clinic, Temple, Sec.

Thirteenth District Society. Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals. Mr. R. L. Heath, Malone-Hogan Clinic, Big Spring, Sec.

MAX E. JOHNSON, M.D.
SAN ANTONIO, TEXAS
Ninety-ninth President, Texas Medical Association

TEXAS
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Devoted to the interests of the medical profession and public health in Texas

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Max E. Johnson

HIS MOTHER wanted him to be a minister, but Max Johnson felt he was better qualified to serve others by becoming a physician. After practicing medicine for 40 years, he is still saying he wouldn't change his chosen work for any other in the world.

Thirty-eight of those years he has spent in one office on the 12th floor of the Medical Arts Building in San Antonio. From there he can look out over the city and down below him, see the Alamo, relic of a long-ago struggle of people wanting a better government. Better government is another long-held and deeply felt concern of Dr. Johnson's. He has been heard to say, "It is not enough for a doctor to be a good physician; he must also be a good citizen, taking an active part in religious, civic, social, and political life of his community."

Such is the concept of service he brings to his office as 99th President of Texas Medical Association.

A man of boundless enthusiasm and energy, Dr. Johnson has long been active in medical organizations. He has belonged to the AMA, TMA, and Bexar County Medical Society since 1925, and last year when he was President-Elect of TMA, he was also president of his county society.

His decision to enter general practice rather than a specialty was based on his desire to be a personal physician to all members of a family, and he has continued to believe that general practice is "a way of life, second to none." Over the years he has been active in the general practice organizations. He has been a member of the American Academy of General Practice since it was founded; he was vice president of Texas Academy of General Practice in 1962, and he was president of the Alamo Chapter in 1956.

His interest has extended to other medical groups including the International Medical Assembly of Southwest Texas of which he was president in 1961,

Texas Industrial Medical Association (president, 1960), American Association of Physicians and Surgeons, Industrial Medical Association, and Southwest Surgical Congress.

He has been a member of Laurel Heights Methodist Church for more than 35 years and has been on its official board for much of this time.

Many civic groups and activities have claimed Dr. Johnson's attention. He has been active in Boy Scout work for a number of years and in 1955 was presented the Silver Beaver award. He is a member of the San Antonio Chamber of Commerce, San Antonio Medical Foundation, San Antonio Livestock Association, Beautify San Antonio Association, and formerly was chairman of both Bexar County and the San Antonio boards of health. He has been a member of San Antonio Planning and Research Council and of the board of directors of Community Nursing Service.

He belongs to a number of social organizations including the Conopus Club, Torch Club, Texas Cavaliers, San Antonio German Club, Quarterback Club, and the San Antonio Country Club. He is a member of Patterson Masonic Lodge, a 32nd degree Scottish Rite Mason, Knights Templar, and member of Alzafar Temple Shrine.

Dr. Johnson became interested in politics as a pre-med student at the University of Texas and this interest has never waned. He was one of the original members of the Good Government League in San Antonio which was able to replace the commissioner form of government with the council-manager form in 1952. He served as a member of the San Antonio city council for three years (1958-1961) and devoted many hours to city government.

A staunch believer in the two-party system as a necessity for preservation of a good conservative government, he has actively supported the Republican Party. He is a charter member of TEXPAC, serving as its chairman in 1962 and 1963, and is an active and sustaining member of AMPAC.

Dr. Johnson was born in Wilson County on July 26, 1900, the son of Elliott Vernon and Jessie Willie (Spear) Johnson. His early schooling took place in Floresville. He attended the University of Texas and was graduated from the Medical Branch in Galveston in 1924. He interned at Robert B. Green Memorial Hospital, San Antonio, and was chief of orthopedic service there for many years.

During World War II he was an Army Air Force major.

When asked about his hobby, Dr. Johnson has always said, "My family is my hobby." His wife attests that he has never been too busy nor too tired to attend birthday parties and special occasions for his children and his grandchildren. She has seen him decline numerous personal invitations so that he could be present at some special function in which his children were interested or involved, such as ball games, track meets, Scout outings, and their dances, parties, or trips during their school days. His family includes Elizabeth, whom he married in 1929; three children, Mrs. James C. Anderson, San Antonio; Dr. Douglas E. Johnson, a resident in urology, University of Iowa; and George Milton Johnson, an independent geologist in San Antonio; four grandchildren; his brothers, Dr. Ted Johnson, also in general practice in San Antonio, and Morris Johnson of Floresville; and his sister, Mrs. Jessie Lee Gant of San Antonio.

When Dr. Johnson turns to recreation, he pursues it with the same zesty spirit with which he attacks his work. At the bridge table, he wants to make the most out of each hand and enjoys every minute of the game. He especially likes to drive to his country home, 60 miles away in the Bandera hills, where he turns his attention to doctoring trees and animals or assists in farm, yard, and household chores. He excels as a cook and is becoming an avid bird-watcher. Equally as much as these occasions for relaxation near home, he likes to travel to distant places, spending each day of the journey in sightseeing.

A challenge Dr. Max Johnson has sounded before and is expected to make again as he begins his year as President of TMA is a call for active participation on the part of every member of the Association in working together toward a common goal. Actual participation, not merely the paying of dues, is the only reason to retain membership in any organization, he has long believed.

"Texas medicine is fighting your battles," he has stated. "Many problems are facing medicine today. They are your problems. You are in politics. Why not be effective?"

Tumor Seminar: Use of Total Knowledge

PUBLICATION of the 1962 San Antonio Society of Pathologists' Tumor Seminar in this issue of the *Journal* focuses attention on an annual educational event of the greatest interest to pathologists and other physicians in the Southwest.

For the past 20 years the pathologists of San Antonio and those at the Brooke Army Medical Center have jointly sponsored this annual seminar at which nationally and internationally known surgical pathologists discuss at length, and before an enthusiastic audience, the differential histologic diagnosis in instructive problem cases. Plaudits are due the pathologists of San Antonio for their earnest endeavor and hard work in bringing these seminars to the medical public.

Texas physicians can be justifiably proud of the fact that this popular and effective teaching method of the slide seminar originated in the annual meetings of the Texas Society of Pathologists, Inc., in the early 1930's. A formal seminar was instigated in Texas by Dr. B. F. Stout and other pathologists of San Antonio in 1944, and is now being presented as an annual event by the San Antonio group and the Brooke Army Medical Center. Teaching by slide seminars has spread to many other states, and is now used effectively by national societies.

Dr. Raffaele Lattes is one of the country's outstanding pathologists, and it is fitting that his contribution in this seminar should be singled out for special notice. This arises from the fact that Dr. Lattes is professor of surgical pathology at Columbia University College of Physicians and Surgeons, New York City. He now holds the post formerly occupied by Dr. A. P. Stout, who was the first conductor of the Annual Tumor Seminar at San Antonio and returned for repeat performances on several occasions.

The proceedings published herein exemplify exceptionally well the manner in which the surgical pathologist's knowledge of the biology of disease is skillfully brought into focus in establishing a final diagnosis. In Dr. Lattes' remarks it is apparent that the achievement of a diagnosis from the examination of tissue sections involves more than the relatively simple process of looking through the microscope and making an unpremeditated interpretation of the histologic appearances. More significant, there are involved the intricate processes of deductive reasonings, recall of past experiences, and utilization of knowledge from follow-up studies, which every physician in any branch of medicine must employ. While advances in histologic techniques and microscopy, histochemical methods, tissue culture studies, and more recently electron microscopy may add to the specificity with which diagnoses can be made, it is well to bear in mind that a surgical pathologic diagnosis constitutes a physician's findings in a case study, utilizing his knowledge of the nature of disease as well as his skill in microscopic interpretation.

—C. T. ASHWORTH, M.D., Chairman,
Department of Pathology,
University of Texas Southwestern
Medical School, Dallas.

Small Businessmen Are Powerful Allies

DOCTORS WHO WORK at the hometown level to strengthen opposition to the King-Anderson bill may look to the small businessmen for support in this fight.

Long advocates of free enterprise and individual responsibility, men who are in business for themselves are in no mood to accept government intervention in medicine, according to Mr. C. Wilson Harder, president of the National Federation of Independent Business, Inc.

He believes that all citizens should be given the ground rules for the "numbers game" so that none may be deluded by it. Proponents of medicare tout the fact that it would require only a .25 per cent increase in the tax deducted from the individual's paycheck. They seldom mention that the employer would pay a like amount, or that both would be taxed on the first $5,200 of annual salary instead of the first $4,800 (as is currently done), thus increasing by 15.8 per cent the actual amount of social security tax paid. In effect, this proposal alone would wipe out at least one quarter of the gain to the economy achieved by the income tax reduction.

Another angle of finance to be scrutinized is the fact that H.R. 3920 proposes the somewhat unsound method of drawing on the general funds of the United States Treasury to give lifetime medical care to persons who have paid nothing into the Social Security System or the Railroad Retirement plan, but who will reach age 65 on or before Dec. 31, 1966. On paper, such costs would be borne by a social security trust fund, but with the provision that the trust fund would be reimbursed by appropriations from the general funds of the Treasury. Obviously, the 1966 deadline would be considered discriminatory and would be extended indefinitely. Such proposals can have little appeal for men who back their spending programs with a clear idea of how much money they need, where it is coming from, and how it is to be repaid.

The initial proposal is only the beginning. The tax almost certainly would plumpen, perhaps unobtrusively, into an obstructively fat tax of several times the proposed $3.5 billion per year. Businessmen have seen this happen before — several times — and they have no illusions that it could not happen again. They also are acutely aware that every time the employer half of social security taxes is raised, labor cost increases, thus adding to inflation and eroding through higher prices the benefits that employees expect to receive.

In businessmen, physicians have strong allies: they are well organized, energetic, and devoted to basic American freedoms and responsibilities. In a recent poll, about three-fourths of the independent proprietors who responded voted against social security medicine. They realize that it is, among other things, bad business, and it is on that basis that they will join with physicians in the battle now under way.

AMA: For or Against You?

CERTAINLY YOU HAVE heard this allegation: "The AMA is always 'against' everything. It's never 'for' anything."

This is not so. The AMA has compiled an enduring history of advancing the science and art of medicine. In the 86th Congress, 700 bills were introduced which affected medicine; there were 1600 the next year.

Was the AMA against all these bills? Certainly not. Of course, only a few bills get before committees, and in recent sessions, the AMA record stands *for* 51 bills and *against* 14. A negative record? Hardly.

Another frequent charge is that the AMA opposed Blue Cross when it was first proposed.

However, the AMA has *never* opposed voluntary health insurance. In the 1930's, the AMA House of Delegates expressed displeasure at projects then being developed in which physicians were hired by a third party and their services sold at a profit. But sound voluntary prepayment insurance plans for medical and hospital benefits always have been supported by the AMA.

Oh, but—our critics say—you opposed the Social Security Act in 1935 when it was adopted.

Nonsense. The AMA testified before Congress on only one section of the legislation—the section calling for extended public health services. The AMA *supported* that section.

Yes, but the AMA opposes the legislation financing health care for the aged via the Social Security System.

Definitely we do, because we are solidly opposed to using the Social Security Act to impose a compulsory national health scheme on the American people.

But we are *for* old people; we are *for* helping those needy and near-needy old people who need help; and we are *for* the Kerr-Mills Law, Medical Aid for the Aged, which provides all the help that these persons require.

These are the facts; they are in the record.

And how the proponents of social security medicine love to damn the AMA by charging that the Kerr-Mills Law, which we support and promote, has failed, is failing, and will fail!

But what is the record?

All states have made use of either the medical assistance to the aged facet of the law, or of its provisions for state old age assistance programs. Kerr-Mills is failing? Hardly.

There is another tired charge that the AMA controls the supply of doctors in this country, and that it does not want more physicians.

Yet, the AMA long has worked to increase

the number of qualified physicians. Since 1847 the AMA has sought to improve the nation's medical schools, and its activities led to the house-cleaning of medical "diploma mills." The AMA and the Association of American Medical Colleges have periodically inspected all schools to insure high standards.

As early as 1922, organized medicine encouraged the expansion of existing medical school facilities and the construction of new ones.

A record number of 7,265 new physicians were graduated from U. S. medical schools last year; 31,491 students are now enrolled in our medical schools, and some 40,000 graduate medical trainees—a grand total of about 55,000 medical students, interns, and residents in this country.

Still, the medical profession recognizes the need for more physicians and is doing something about it.

Each year, for example, physicians contribute approximately $5,000,000 to the American Medical Association Education and Research Foundation, and to their own medical schools. They also help to underwrite AMA's multimillion dollar loan program.

The medical profession also has a nationwide medical careers information program to attract superior students, and answers more than 300 letters a week.

A few years ago we decided to do something about those medical students, interns, and residents who needed financial help to complete their training.

The result was a plan of guaranteeing bank loans to them. The AMA in effect agreed to become a cosigner of a long-term note at reasonable interest rates, and bankers agreed to make $12,500,000 available in loans for every $1,000,000 we put into the guarantee fund. The first loans were made in March, 1962. Physicians and others were asked to help make the plan work by investing in the loan guarantee fund. And they did.

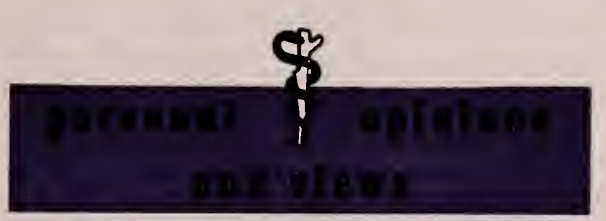
Through 1963, 11,875 loans totaling $13,700,000 had been made to 8,430 individuals. They represent students in 85 medical schools and interns and residents in 583 hospitals.

We are proud of this program, for it represents a major advance in the life of American medicine. And we believe that medicine once again has demonstrated the truth that in a free society, men can find ways to fulfill their capabilities and desires.

In the fight for freedom, one must be opposed to government intervention and federal compulsion.

Medicine, doctors, and the AMA always have been *for* freedoms of patient, physician, and American. We always have been *for* the voluntary way and the free enterprise system. We do not intend to change or to back away from those convictions and these principles now in the fight against government —no matter how big it is.

—GEORGE M. FISTER, M.D., Ogden, Utah, Past President, American Medical Association.



Medical Societies Must Expand Their Goals

SERVING as president-elect and president of a large county medical society is an education in itself. No longer is one concerned merely with intraprofessional problems; one becomes involved with both politics and policing at local, state, and national levels, and interprofessional and civic responsibilities must be met.

It is becoming mandatory for the medical society to establish and keep up a close liaison with the community council and other welfare agencies in large cities. The ideas of salaried professional planners who serve these groups are not always in accord with medical thinking, and unless physicians are in a position to point out inherent problems in some proposals, the medical society may find itself embroiled in projects contrary to sound medical principles.

There must be, and soon, an awakening of the general membership of medical societies

to their overall responsibilities to the public, their profession, and their own consciences.

Another situation which could be improved by more careful attention from the medical societies concerns the newcomers to our ranks. Young doctors apparently often leave medical schools today in a rather confused frame of mind as to their status as doctors. The medical society should constantly present a guiding light to these young people to keep them in the main channel, and away from the rocky shoals. There can be no better beacon than the helping hands and ethical conduct of the older members.

The philosophies given impetus during the depression have expanded to such a point that the "something-for-nothing" attitude has become a way of life in our country. This concept has been fostered by political leaders in their grab for votes and power, and has been grasped by lower political leaders and those in our populace who can turn it to their own advantage. The voices crying out for sanity, self-reliance, and integrity—the principles upon which this country rose to greatness—echo in the wilderness and are smothered by castigation and abuse. The great American public shudders at the thought of socialization, but fails to recognize the insidious forms in which it is being offered; instead, Americans accept these plans because high officials say that they are not socialistic.

From my experience, I would place physicians in three large categories:

First are those who attempt to preserve the independence of the medical profession. This group includes some zealots who, although dedicated and sincere, often create difficult situations. This entire group, generally speaking, will give of their time, money, and talents to promote the interests of the profession and to keep informed on the issues confronting it.

Second are those who are concerned with things which affect them immediately and personally, but who are apathetic and not too well versed on long-range problems facing the profession. They fail to see the immediate and acute problems as phases of the long-range ones. Some in this group are convinced that governmental control of medicine is inevitable and that fighting it is a waste of energy. The fringes of this group include physicians who are basically in agreement with those in the first group, but because they have ties of various strengths with the third, their allegiance is split many ways.

Third are the physicians who would change the whole concept of the private practice of medicine as we know it today, financing each step with a free flow of the taxpayers' money, with no strings attached. This philosophy favors such practices as:

1. Development of large "health centers" affiliated with, and centered around, medical schools.
2. Expansion of physical facilities and staffs to stimulate more funds and thereby alter the traditional primary function of the medical schools from teaching to research.
3. Entrance into community service fields, when funds accompany such projects, regardless of existing facilities or competition with private practitioners.
4. Replacement of the general practitioner with the internal medicine specialist as the "family doctor" of the future.

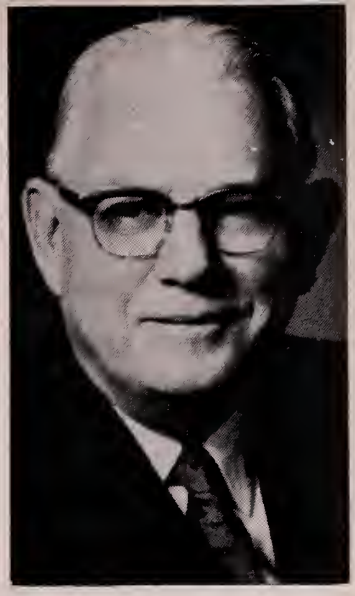
Recently, the president of the Association of American Medical Colleges said, "the American Medical Association cannot speak for the medical colleges" and "fundamental differences in goals" now exist between it and the A.A.M.C.

In this group of doctors are some who do not wholly agree with its overall philosophy but are enmeshed by their positions and must, on the surface at least, appear to follow it. The ones who break away from the system frequently become its most outspoken critics.

I am convinced that the greatest danger to the free private practice of medicine lies within our own ranks, and unless we have a mass awakening to our moral obligations, future pathways will be rugged.

As a final thought, it is amazing how many "impossible" things become possible if a concerted effort by those involved is really made.

—C. W. YATES, M.D., Houston.



A YEAR FOR DECISION AND ACTION

Members of the medical profession MUST present a united front. There may be no tomorrow. We do have today. We must decide what we want most in life, and then act immediately if we are to obtain it. The President of the United States, the Department of Health, Education and Welfare, and those members of Congress who favor medical care under social security have announced their all-out efforts to pass the King-Anderson bill this year. Is this what you want? If it is, do nothing and you shall surely have it; if not, act at once to defeat it.

As a doctor you hold a unique place in the life of your community. You have unlimited resources of help seldom realized or acknowledged—your many loyal patients. Have you enlisted their help in this struggle to protect the private practice of medicine? Have you discussed the issues with your friends? Has your wife, informed as she surely is through the Woman's Auxiliary, done her share to retain your way of life?

I earnestly believe that our 9,000 Texas doctors, joining hands with the 150,000 other physicians over this great land of ours, plus their wives, friends, and patients, can rise up and defeat this scheme to socialize our profession. If medicine falls victim, there is little reason to believe that all other avenues of free enterprise will not be closed.

A few doctors openly advocate government medicine. Our greatest danger is not from this paltry number; it is from the large numbers who possess a defeatist attitude by resigning themselves to what they believe inevitable and by accepting this type of legislation without making any effort to oppose it. This is the group that we must arouse and enlist.

It is with full realization of the tremendous task ahead that I embark on my year as your President. I appreciate the honor of serving in this capacity, but am filled with humility and a deep sense of responsibility. I assure you that I will do everything within my power to justify your faith in me. Together let us meet the challenge. I am confident that we can defeat the King-Anderson bill. 1964 is our year for decision and action.

Mat E. Johnson

President
Texas Medical Association

Tumor Seminar

Case 1

Diagnosis.—Thymoma, mixed epithelial and lymphoid type, with foci of necrosis in myasthenia gravis.

Contributors.—Drs. N. H. Jacob and J. K. Lowry, Santa Rosa Medical Center, San Antonio.

History.—A 50-year-old diabetic man with myasthenia gravis complained of difficult respiration and swallowing for two weeks. He had noticed a swelling of the neck with minimal tenderness. Radiographs of the chest revealed a superior mediastinal mass. A median sternotomy was performed and immediately beneath the superior sternum was a white, gristly, multilobular tumor mass invading the base of the heart along with the aorta and great vessels.

Submitted Diagnoses.—Thymoma, 42; malignant thymoma, 13; thymoma, epithelial, 12; thymoma, other, 4; metastatic carcinoma, 1.

DR. LATTES: The history of myasthenia gravis and of roentgenographic demonstration of a superior mediastinal mass of course has a great bearing in the correct appraisal of this case. We are not told the exact size of the tumor, but there is an interesting statement—namely that the tumor "invaded" the base of the heart along with the aorta and great vessels. We shall return later to this point.

This is a variant of thymoma. The tumor consists of nests and lobules of high cellularity separated by abundant dense collagenized fibrous tissue, and it also shows multiple foci of necrosis. This latter finding is unusual and I wonder whether preoperative radiation therapy was used. The areas of high cellularity show a typical mixture of cells of lymphoid type with more or less conspicuous epithelial cells. The latter are sometimes arranged in small nests of obvious epithelial pattern; elsewhere they are intimately admixed with lymphoid cells. In some places the cytoplasm of these cells of epithelial type is fairly abundant and clear, and this is a well-known finding in thymomas associated with myasthenia gravis. In one of the sections submitted to me there was a small focus, not larger than one microscopic field, in which the epithelial cells were spindle-shaped. This is extremely unusual in myasthenia patients, and I have seen it personally only in cases that have been previously treated with radiation therapy. Reticulum stains show absence of reticulum fibers within the nests and cords of lymphoid and epithelial cells. In the sections submitted to me there was no recognizable thymus gland. In two or three places what was probably the outer border of the tumor was demonstrated, and this showed an incomplete demarcation between tumor and surrounding adipose tissue.

The diagnosis of thymoma is obvious. As mentioned before, there are some features of interest, one being the gross description of a tumor "invading" the base of the heart. This is in agreement with some microscopic fields suggesting lack of true encapsulation. In our experience the prognosis of those thymomas which lacked encapsulation has been definitely worse than that of the well-encapsulated ones. This, however, was a feature more obvious in the non-myasthenia cases, among which of 30 patients with well-encapsulated thymomas, 21 were free of disease following surgery, 6 died of unrelated disease, 1 died immediately postoperatively, and only 2 showed persistent or recurrent tumor. Of 5 patients having non-encapsulated, non-myasthenia thymomas, 2 were alive two and three years postoperatively with pleural implants, 2 died because of recurrent or persist-

Proceedings of the 19th Annual Tumor Seminar presented by the San Antonio Society of Pathologists at Brooke Army Medical Center, Fort Sam Houston, on Dec. 8, 1962. The conductor was Dr. Raffaele Lattes, professor of surgical pathology at Columbia University College of Physicians and Surgeons, New York City.

ent tumor, and only 1, who received irradiation following biopsy, was still alive after six years. If, however, we look at the significance of encapsulation in 17 cases with myasthenia gravis, we find a less clear correlation. Generally, if these patients had further trouble this was the result of persistence or worsening of the myasthenia rather than of recurrence of the tumor. I mentioned earlier that there were other features worthy of interest; they are the extreme fibrosis, the occasional focus of spindle cells, and the large foci of necrosis. These three features combined make me wonder whether this patient was irradiated preoperatively. As hinted, in our experience the prognosis of the myasthenia gravis disease overshadows generally that of the tumor of the thymus.

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Case 2

Diagnosis.—Benign lymphoid mass of probable hamartomatous nature of mediastinum.

Contributors.—Drs. D. L. Galindo and J. E. Espejo, Robert B. Green Memorial Hospital, San Antonio.

History.—A 42-year-old woman was found by roentgenographic examination to have a

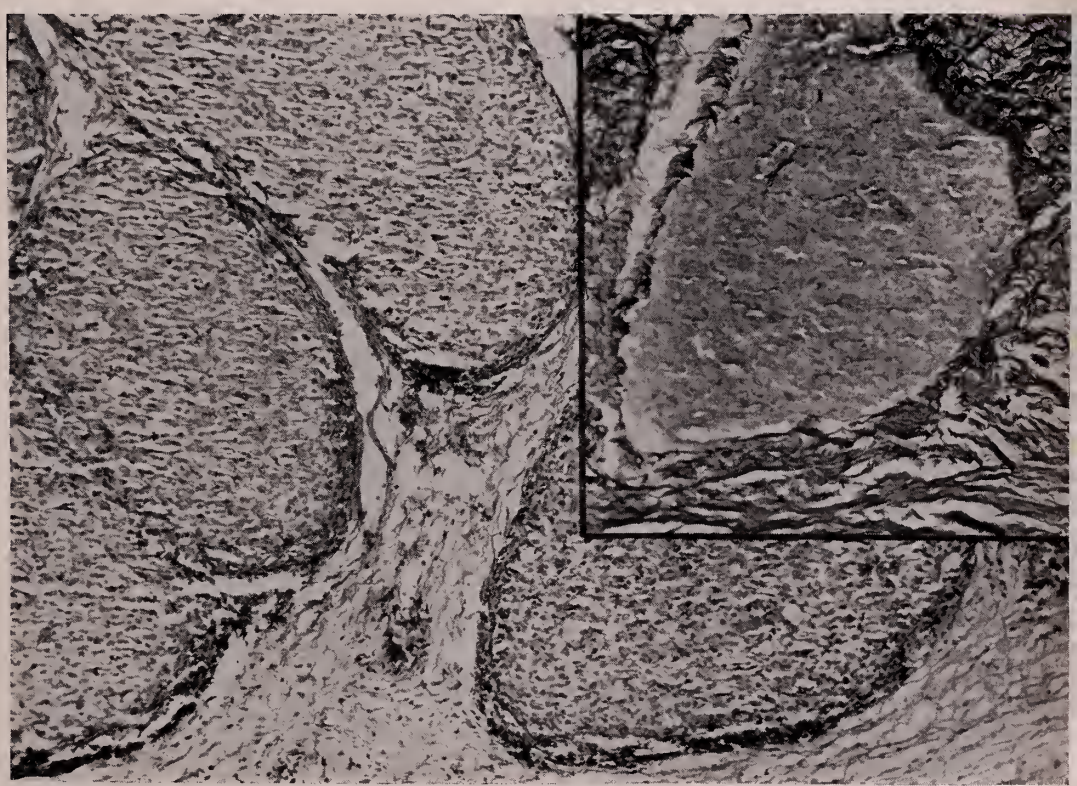

Fig. 1. Case 1. Thymoma in myasthenia. Nests of predominantly epithelial cells separated by thick, fibrous trabeculae. Reticulin stain (insert) shows epithelial pattern. Hematoxylin and eosin and Laidlaw stains, X 120.

calcified mass below the tracheal bifurcation. She had had a simple mastectomy followed by radiation for papillary carcinoma. The tumor was lobulated, discoid, resilient, partially calcified, and well encapsulated; it measured 8 by 7 by 2 cm.; and on section was finely granular and tannish pink, with focal calcific nodules.

Submitted Diagnoses.—Lymph node hyperplasia, 24; thymoma, 23; benign lymphomatous mass (? hamartoma), 18; thymus, radiation change, 3; lymph node, radiation change, 2; thymic lymphosarcoma, 1; lymph node, Hodgkin's disease, 1.

DR. LATTES: In this interesting mediastinal tumor found below the level of the bifurcation of the trachea in a 42-year-old woman with a history of radiation therapy to the thoracic region, the resemblance to a thymoma is only superficial. This mass consists of lymphoid tissue with a tendency to form follicles with small germinal centers. Many of these germinal centers are partly hyalinized and some of them are calcified. In practically all of them it is possible to recognize thick-walled arterioles. In some of these germinal centers there are occasional cells of probable reticuloendothelial type which have large, somewhat bizarre, nuclei. The tissue is quite vascular and the germinal centers and related follicles are scattered diffusely throughout it, and not only in the cortical area as one would expect in an enlarged lymph node. In addition no cortical or medullary sinuses are recognizable. Reticulin stains show abundant reticulum in other microscopic fields. In addition to the bizarre cells in the germinal centers, other bizarre nuclei are seen here and there, and they resemble strongly those fibroblasts which are sometimes observed in heavily irradiated areas.

These lesions frequently have been mistaken for thymomas because the heavily hyalinized germinal centers can be mistaken for Hassall's bodies. However, they certainly are not tumors of thymic tissue. Several good papers already have been published on this subject. Castleman interpreted them as benign hyperplastic lymph nodes; others have suggested lymphnodal hamartoma or giant hemolymph nodes. In our laboratory we have recently analyzed 12 cases which occurred not only in the mediastinal region but also elsewhere in the soft tissues of the body. All had in common an absolutely benign course and the histological features already mentioned; namely, a diffuse follicular architecture, a lack of sinusoidal pattern, a marked vascularity both in the follicles and between follicles, and changes in the germi-

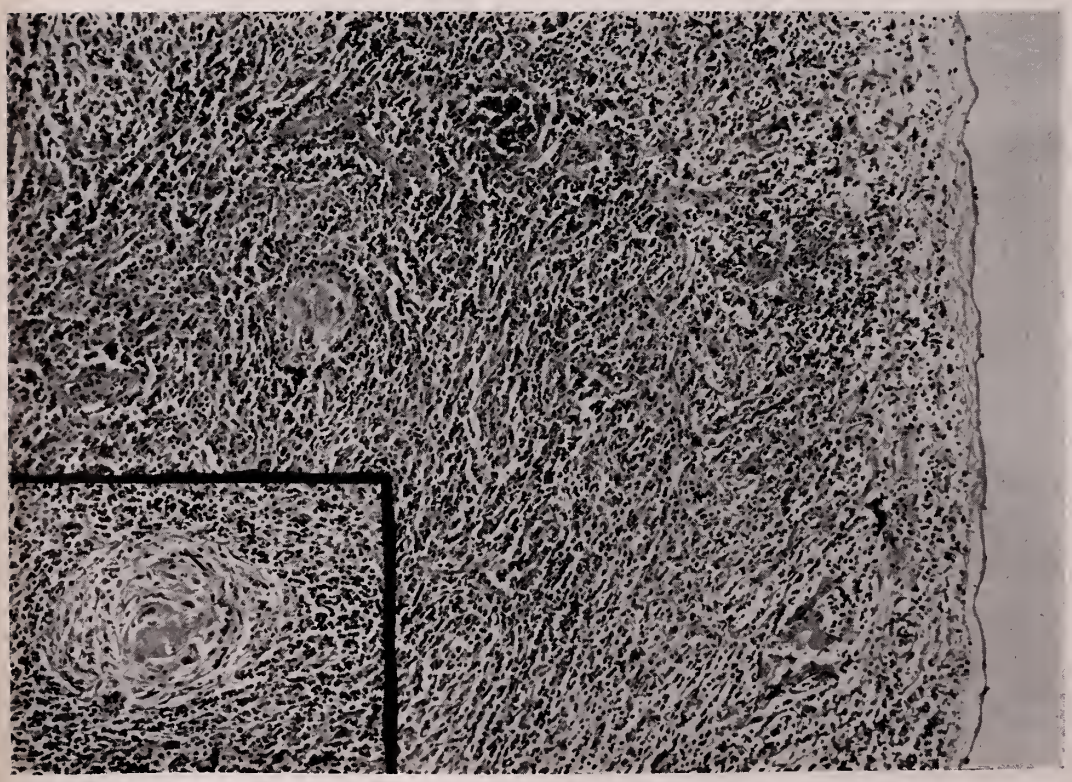

Fig. 2. Case 2. Benign lymphoid mass. Lymphoid tissue shows numerous blood vessels and no peripheral or medullary sinuses. Insert shows a hyalinized follicle center, bearing a vague resemblance to a Hassall's corpuscle. Hematoxylin and eosin stain, X 120.

nal centers which can be mistaken for Hassall's corpuscles. However, in none of our cases did we see bizarre cells, and I believe in the case being discussed here the bizarre cells might be related to previous radiation. At any rate I believe that this is not a thymoma and not a lymphosarcoma of any variant, but a benign lymphomatous mass.

REFERENCE

1. Lattes, R., and Pachter, M. R.: Benign Lymphoid Masses of Probable Hamartomatous Nature; Analysis of 12 Cases, Cancer 15:197 (Jan.-Feb.) 1962.

Case 3

Diagnosis.—Granulomatous thymoma (so-called Hodgkin's disease of the thymus).

Contributor.—Dr. J. G. Webster, Lackland Air Force Base Hospital, San Antonio.

History.—A 21-year-old white man had a total thyroidectomy and right radical neck dissection. Papillary carcinoma involved the right lobe, isthmus, and cervical lymph nodes. Follow-up chest films showed growth of a hilar mass noted on roentgenogram at the time of surgery. Thoracotomy was performed four months after the neck dissection. An anterior mediastinal tumor involved the area of the thymus. A discrete specimen measuring 14 by 11 by 6 cm. and weighing 225 gm. was lobulated and tannish pink, with cystic areas surrounded by fibrous stroma.

Submitted Diagnoses.— Granulomatous thymoma (specified as Hodgkin's, 48), 62; thymoma, 5; thymic cyst (with squamous cell carcinoma, 1; teratomatous, 1), 3; malignant thymoma, 1; thymus, reactive hyperplasia, 1.

DR. LATTES: Here we are dealing with a disease about which there probably will be considerable controversy regarding its correct interpretation. The patient, a 21-year-old man who has had papillary carcinoma of the thyroid, developed an anterior mediastinal mass which was interpreted preoperatively as representing either a metastasis from the thyroid or Hodgkin's disease of the mediastinal lymph nodes.

All the sections submitted to me show similar lesions, and they show that unquestionably the tissue involved is the thymus gland and not lymph nodes. It is also unquestionable that this thymus gland is severely involved by a process of extensive fibrosis, infiltration of eosinophils and histiocytic cells which vary in size, and multiple foci containing bizarre cells and cells with double nuclei and conspicuous nucleoli which have many of the morphological characteristics of Reed (Sternberg-Reed) cells of Hodgkin's disease.

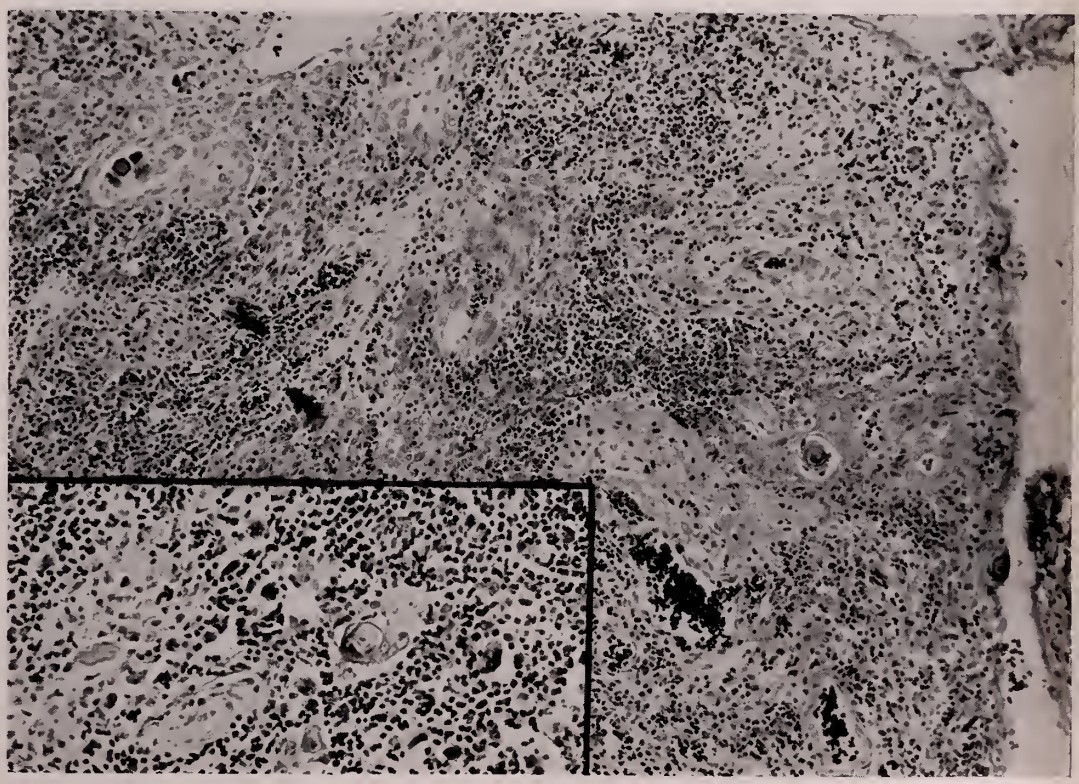

Fig. 3. Case 3. Granulomatous thymoma. Note thymic tissue with prominent epithelial sheets and Hassall's corpuscles, disrupted by fibrosis. Shown in the insert are bizarre cells associated with Hassall's corpuscles. Hematoxylin and eosin, X 120 (insert: X 180).

I have now seen several cases of this type. More precisely, in four analogous cases the disease seems to have been completely controlled two, five, seven, and eight years after surgery plus irradiation, or biopsy and irradiation. In one other case the disease has spread to the cervical region, apparently to the cervical lymph nodes. In another case the patient died elsewhere with clinical symptoms of a retroperitoneal mass, but no autopsy was performed. One more recent case has had a pathological fracture of the spine with the cause undetermined even after biopsy. In all our cases, and also in this present case, there were features suggesting that this lesion is different from the common garden variety of Hodgkin's disease. Cells of the Reed type often seem to arise from the thymic epithelium. They often contain periodic acid-Schiff (PAS) positive granules, which are characteristic of thymic epithelium and which should not be found in typical Reed cells of Hodgkin's disease. The tendency in the cases that we have studied has been for the lesion to be limited to the anterior mediastinal area, or if it spreads, to do so by way of direct extension rather than by becoming systemic as in typical Hodgkin's disease. On the other hand I agree that many of the morphological features are indistinguishable from those of Hodgkin's disease. It is possible that this is a peculiar type of Hodgkin's disease, made peculiar by the fact that it involves the thymus, which is a lympho-epithelial organ instead of a lymphoreticular organ. I know that some authorities in this country do not accept the concept of granulomatous thymoma or Hodgkin's-like thymoma, and believe that this is just a manifestation of Hodgkin's disease. However, other authors, starting with Dr. James Ewing and continuing until recently, have accepted the concept that in certain malignant tumors of the thymus there can occur secondary granulomatous changes which resemble those of Hodgkin's disease. We still have much to learn on this subject, but I believe that for the time being it is worth keeping these cases apart from the general group of Hodgkin's disease.

REFERENCE

1. Lattes, R.: Thymoma and Other Tumors of Thymus, Analysis of 107 Cases, Cancer 15:1224 (Nov.-Dec.) 1962.

Case 4

Diagnosis.—? Inflammatory pseudotumor of the mediastinum. ? Leiomyoma of mediastinum.

Contributor.—Dr. H. A. Van Auken from Fitzsimons General Hospital, Denver, Colo., now at Baptist Memorial Hospital, San Antonio.

History.—A three-year-old white boy had repeated respiratory infections, chest pain, fever, and cough for two months. Roentgenograms (negative one year before) showed a large mass in the right anterior chest. A large lobular tumor in the superior and anterior mediastinum, extending into the right upper lobe, was removed with the right upper lobe and thymus.

Submitted Diagnoses.—Inflammatory pseudotumor, 27; thymoma (spindle cell type, 9; other types, 16), 25; leiomyosarcoma, 5; mesothelioma, 4; plasmacytoma, 4; miscellaneous sarcomas, 4; hemangiopericytoma, 1.

DR. LATTES: This is a large tumor of the superior and anterior mediastinum occurring in a three-year-old baby and documented as not having been present one year before. Apparently it was intimately connected with the lung since the right upper lobe had to be removed with it.

This is one instance in which, with the histological material available, the proper classification of this tumor can be made only with approximation. We are dealing here with a highly cellular tumor which contains areas of necrosis and degeneration with inflammatory infiltration. In the portions where the tumor tissue is best preserved, it apparently consists of interlacing bundles of spindle-shaped cells which have strongly eosinophilic cytoplasm. There appears to be little intercellular substance stainable with the usual hematoxylin-eosin stains. The trichrome stains made have not been too successful. One possibly important clue is given by the reticulin silver stain, which shows that within the individual bundles of tumor cells there are moderate numbers of reticulum fibers, and that they are definitely arranged parallel to the long axis of these bundles, resembling in this the pattern that we are used to seeing in Schwann's cell tumors and in smooth muscle tumors. Not all the tumor cells are spindle-shaped; some of them appear to have rounded up, but it is hard to know whether this is related to degenerative changes. Occasional tumor cells are larger

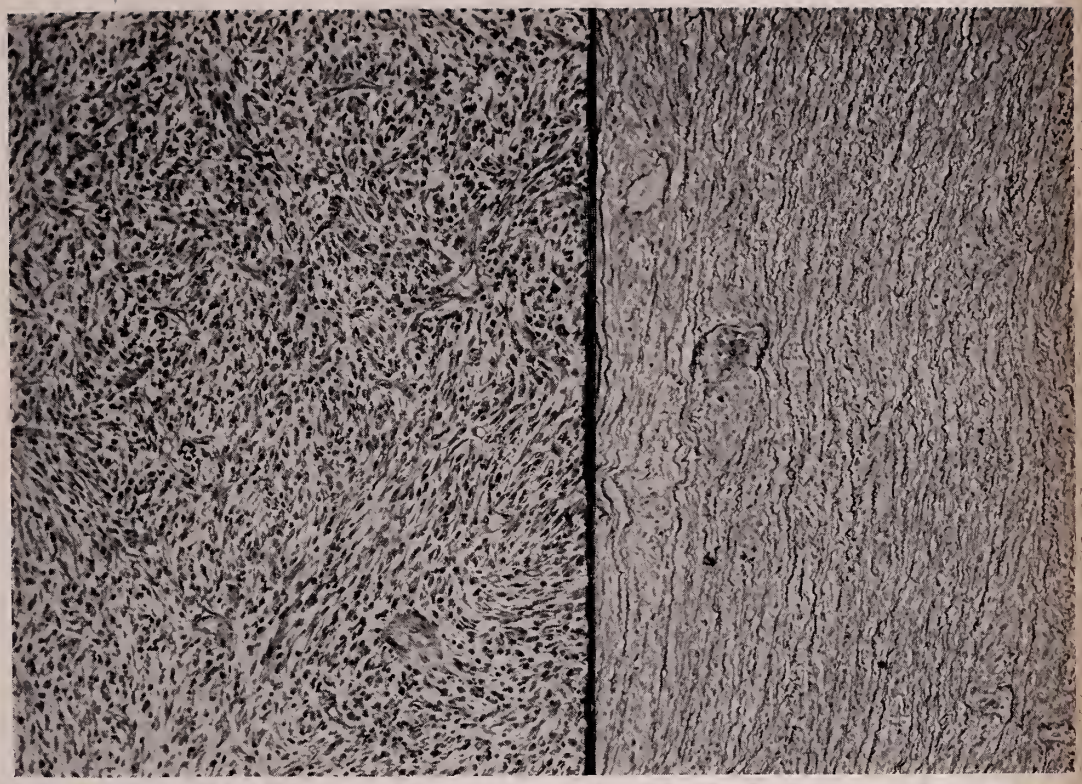

Fig. 4. Case 4. Leiomyoma of mediastinum or inflammatory pseudotumor. Left, interlacing bundles of spindle cells resemble smooth muscle cells; right, a reticulin stain shows striking parallel arrangement of the fibrils. Hematoxylin and eosin and Laidlaw stains, X 180.

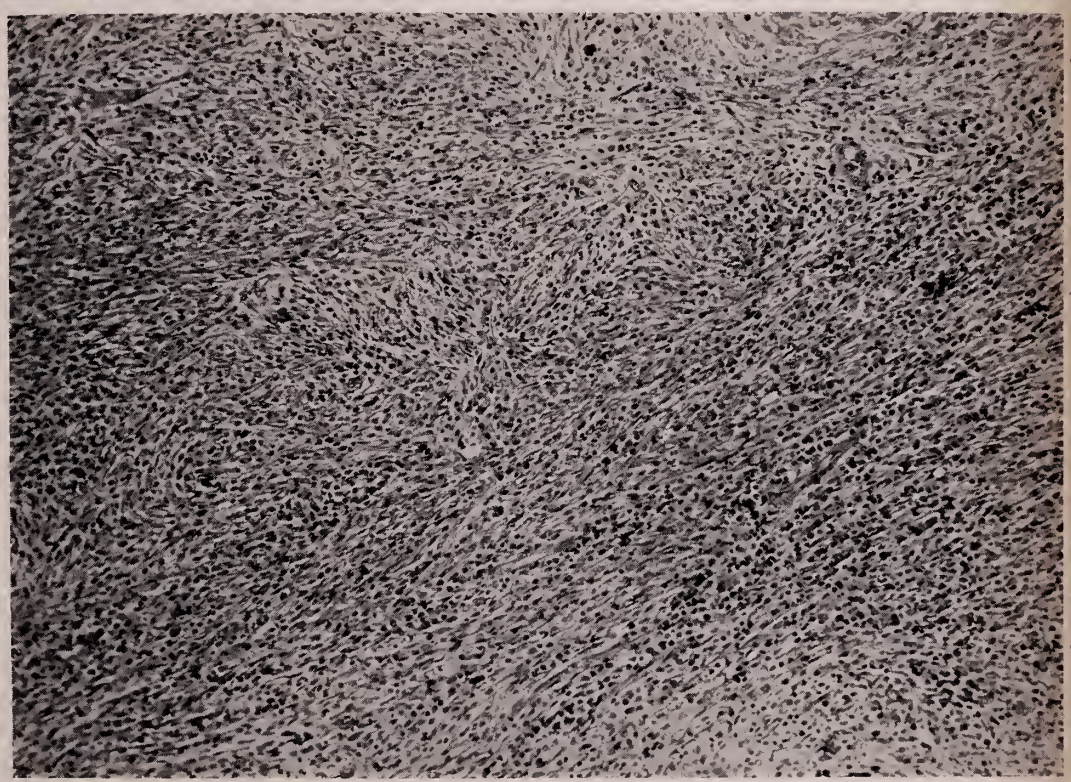

Fig. 5. Case 4. Another field of the same tumor shows a more "reparative" type of tissue, associated with diffuse inflammatory infiltration. Hematoxylin and eosin stain, X 180.

and show a finely granular eosinophilic cytoplasm. I have not seen any cross-striations. Mitoses are not a conspicuous feature—in fact it takes careful scanning of many microscopic fields before finding even one mitosis.

The features described would suggest a tumor of smooth muscle origin. However, the frequent occurrence of foci of increased vascularity, associated with collections of histiocytes and inflammatory cells, makes it probable that this is in fact one of those poorly understood lesions generally classified

as inflammatory pseudotumors. They probably belong to the group of fibroxanthomas. I have in similar occasions battled between the diagnosis of intrathoracic leiomyoma and xanthomatous or inflammatory lesion, and I am still baffled by the reticulin framework that these tissues exhibit. It is possible that a smooth muscle proliferation occurs in association with inflammatory masses in these regions. However, these pseudoneoplastic xanthomatous lesions have been described in the lung, and not in the mediastinum.

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Case 5

Diagnosis.—Nonchromaffin paraganglioma (chemodectoma) of aortic body.

Contributors.—Drs. C. L. Green and M. S. Hart, Hotel Dieu, El Paso.

History.—A 39-year-old pregnant woman's chest film revealed a tumor in the midmediastinum. A 7.5 cm. tumor found compressing the superior vena cava and partially surrounded by lung tissue was removed with extracapsular cleavage plane. The dense capsule was up to 1 mm. thick. The cut surface was soft and pink.

Submitted Diagnoses.—Nonchromaffin paraganglioma (chemodectoma), 36; hemangiopericytoma, 26; bronchial adenoma, 4; thymoma, 3; neuroblastoma, 1; metastatic melanoma, 1; hemangio-endothelioma, 1.

DR. LATTES: This is a tumor which could be defined as having "organoid" or endocrine arrangement. Essentially the microscopic pattern is that of nests of relatively small cells with small vesicular nuclei and scanty eosinophilic cytoplasm. The cells are uniform in size and show no tendency to form glandular lumens or rosettes or other obvious recognizable structures. Between these nests are inconspicuous connective tissue trabeculae which always contain thin-walled blood vessels. In some places the shape of the cells changes from round or polygonal to spindle-shaped. There is some mitotic activity. Reticulin stains emphasize the organoid pattern in some places in that they show no reticulum fibers within the nests of tumor cells and bring out beautifully the vascularity of the lesion. However, elsewhere these reticulin stains are not as easy to interpret in that they show irregular distribution of reticulum also between the individual tumor cells. It is possible that these are areas of secondary fibrosis. Trichrome stains do not add much to the description except that they confirm that the collagenous stroma is quite scanty. The spindle-shaped cells do not contain recognizable myofibrils. Mucicarmine stains are noncontributory. Unfortunately no material was available for PAS stains or for argentaffin granules stains.

The problem of course is how to classify this tumor. I believe that in the differential diagnosis one should consider the following possibilities: The so-called extrapulmonary mediastinal bronchial adenoma of the carcinoid type which, although it is a rare condition, has been found acceptable by some authors and of which we have a few examples; a parathyroid adenoma, arising in mediastinal parathyroid glands which again is a well-known entity; hemangiopericytoma; and paraganglioma originating from aortic bodies. I believe that extrapulmonary bronchial adenoma is unlikely because of the absence of ribbons and rosettes. The cytoplasm does not appear to be granular. Parathyroid adenoma is a possibility to be considered seriously, and in one microscopic field in one of the several sections which I could study I was able to find a structure which resembles a colloid-filled follicle not unlike what one can see in thyroid and parathyroid glands. However, again the absence of ribbon formation, the absence of any variation in cell type (no oxyphilic nor chief cells), and the tendency to spindle shape in some microscopic fields seems to me to make this hypothesis unlikely also. I think that there is no doubt that some areas could be acceptable as hemangiopericytoma. I refer to those areas in which the predominant cell type is spindle-shaped. In fact, when I first saw some sections of this tumor, sent to me by Dr. Green, I interpreted it as hemangiopericytoma. However, since the dominant feature is that of definite cell nests and cords of endocrine type, I believe that the most likely explanation for this tumor is that it

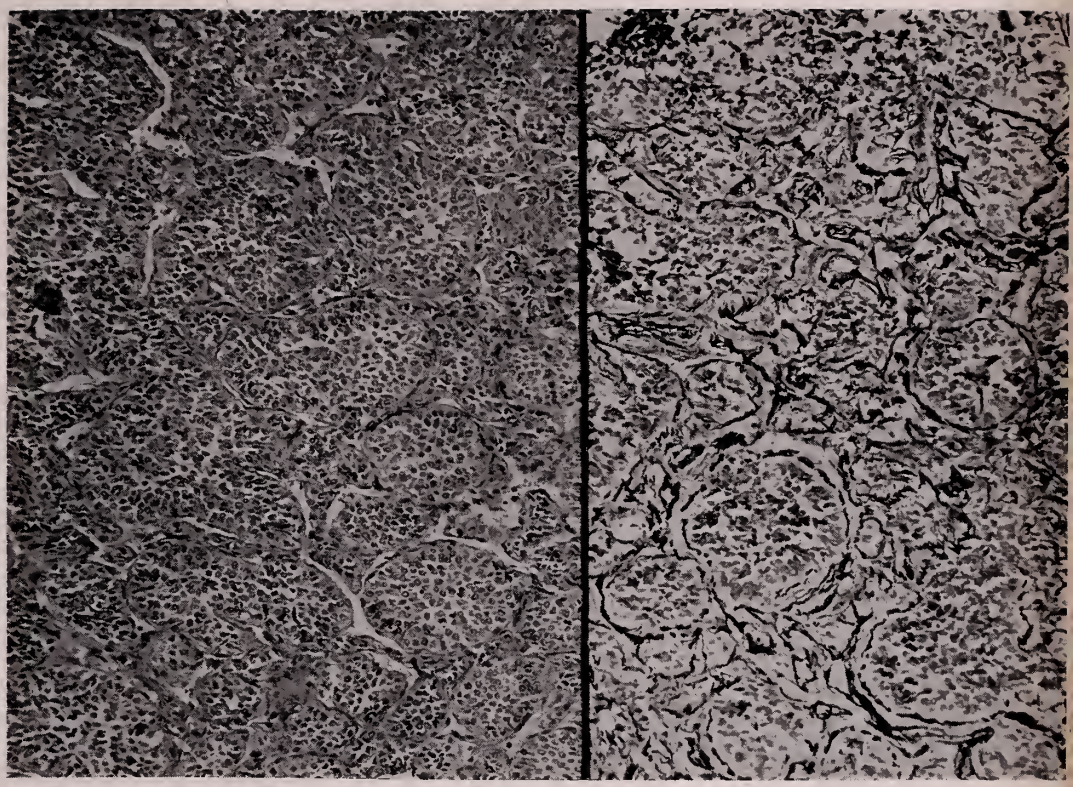

Fig. 6. Case 5. Nonchromaffin paraganglioma of aortic body. Note at left the "balls" of epithelioid tumor cells separated by thin vascular septa. The endocrine pattern is emphasized by the reticulin stain at right. Hematoxylin and eosin and Laidlaw stains, X 140.

belongs to the so-called nonchromaffin paraganglioma (chemodectoma) or aortic body tumor group. Tumors of this type are known to occur even though they are unusual. Some cases had been reported a few years ago from this department.* In addition, more recently, during a review of all our mediastinal tumors, we singled out eight additional cases which we felt were acceptable as nonchromaffin paragangliomas of the aortic bodies, and these have been published.[3] Of these eight cases, five presented as tumors of the anterior-superior mediastinum and three were in the anterior-inferior mediastinum. None was situated posteriorly. None of them behaved as a malignant tumor; in fact four of these were routine findings in patients who were asymptomatic.

Whether the tumor discussed here actually invaded the lung, or just impinged on it, I was unable to determine.

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*Columbia University College of Physicians and Surgeons, Department of Pathology.

Case 6

Diagnosis.—Granulomatous arteritis of gastrocnemius muscle with myositis fibrosa.

Contributor.—Dr. Lattes.

History.—A 61-year-old white man had painful swelling of the left calf for four weeks. The clinical diagnosis was phlebitis. Following biopsy, a poorly outlined fibrous-appearing mass replacing most of the gastrocnemius muscle was excised. The specimen measured 9.5 by 5 by 3.5 cm. No discrete mass could be identified, but the entire muscle showed increased consistency and diffuse gray to yellow-tan streaking parallel with the fibers. The process extended from the muscle into the deep fat.

Submitted Diagnoses.—Myositis (specifying granulomatous arteritis, 7), 29; collagen disease group (specifying vasculitis, 20), 27; necrotizing vasculitis (local), 6; fasciitis group (specifying vasculitis, 3), 5; myositis ossificans, 2; other, 3.

DR. LATTES: This case was added by me to this seminar because both the clinical and the pathological findings were most confus-

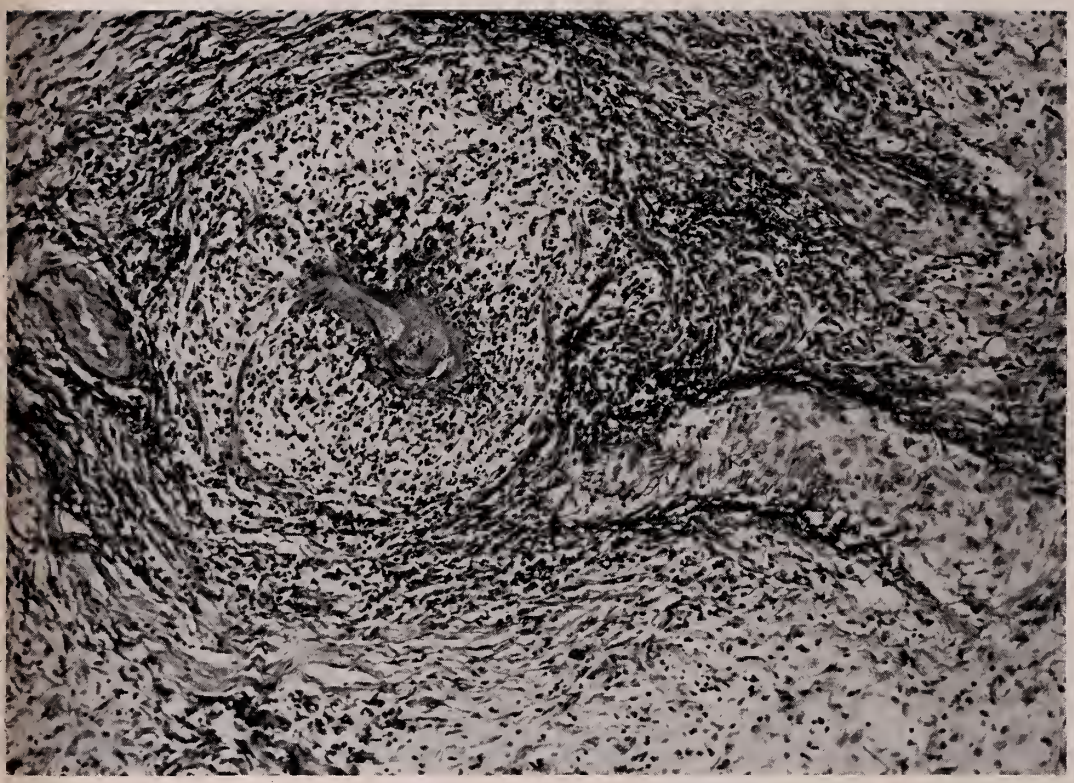

Fig. 7. Case 6. Granulomatous arteritis of gastrocnemius muscle. Note the smudging of the small artery wall and the broad granulomatous cuff surrounding it. Verhoeff stain, X 120.

ing. As far as I am concerned the interpretation of this lesion is still quite uncertain. As you have read in the brief clinical outline, the patient presented a moderately tender, tumor-like mass in the gastrocnemius muscle, and following an incisional biopsy it was decided to remove that mass. This resulted in the excision of a good portion of the gastrocnemius muscle measuring overall 9.5 by 5 by 3.5 cm. There was no discrete mass but the muscle was replaced in part by gray to yellowish tan streaks which blended with the remaining muscle fibers.

First, I want to show a section that was not made available to the seminar, and which represents the small bit of tissue removed for biopsy prior to the definitive operation. This section shows that the skeletal muscle is to a great extent replaced by what appears to be mature, dense, heavily collagenized fibrous tissue. There were only rare collections of mononuclear inflammatory cells and no other obvious significant findings. On the basis of these findings and of the clinical history of a poorly outlined intramuscular "mass" our laboratory made a diagnosis of well-differentiated fibromatosis of the skeletal muscle, and since it was obviously a poorly outlined growth, it was decided that the way to handle it was to excise most of the involved muscle. The subsequent histological findings were a surprise. As you certainly have seen, the intense fibrosis which replaces the skeletal muscle bundles is only one facet of the lesion. What is much more impressive and obviously more important is the diffuse vascular involvement by an inflammatory granulomatous, and occasionally necrotizing, process involving arteries and veins. Essentially, small arteries, arterioles, and small veins are often surrounded by large zones of loose inflammatory tissue containing polymorphonuclear leukocytes, eosinophils, lymphocytes and plasma cells, and large mononuclear cells, and also showing proliferation of fibroblasts and small blood vessels. The lumen of the vessel is often occupied by a fibrinous or fibrinoid mass, and the wall is not infrequently undergoing degeneration. There is also definite fibrinoid necrosis of the walls of some of them. This can be shown beautifully with trichrome stains. My impression is that the majority of vessels involved are arterial in type.

Upon these findings we investigated very carefully the possibility of a systemic disease in this patient. At the time of operation he had a high sedimentation rate and there was a history of occasional mild albuminuria for many years. However, it has been impossible to pin on this patient a diagnosis of systemic arteritis or periarteritis.

He certainly has no Wegener's disease. There is no history of allergy, drug intake, or of any other possible exogenous factor leading to a hypersensitivity status. At present, the patient is symptom-free. Still, the evidence is pretty strong that this is not just a nonspecific inflammatory post-traumatic or postphlebitic condition but a definite granulomatous vasculitis. Further follow-up of this patient probably will reveal the final answer.

One of the lessons to be learned from this case is that not every mass in which a small biopsy shows intense fibrosis is necessarily a fibrous tissue tumor.

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The author has been unable to find any article or book chapter dealing with lesions exactly analogous to this case, namely a vasculitis of granulomatous "hypersensitivity" type, but limited to one area. There are of course many excellent papers on generalized vasculitides, special types of vasculitis such as "giant cell arteritis," temporal arteritis, and Wegener's granulomatosis, but none seemed to apply to this particular case.

Case 7

Diagnosis.—Lipoblastomatosis, infantile, of neck.

Contributors.—Drs. A. O. Severance and H. A. Van Auken, Baptist Memorial Hospital, San Antonio.

History.—A 12-year-old girl developed a small, painless lump in the left supraclavicular region when she was hit with a ball. It was soft and not well limited. The soft, rubbery, pinkish yellow, lobulated, 7.5 by 5 by 4.5 cm. specimen differed from the usual lipoma on section, appearing glistening tan and opaque. Scharlach R stains were positive.

Submitted Diagnoses.—Liposarcoma, 51; infantile lipoblastomatosis, 7; myxoma, 6; lipoma (various types), 5; fasciitis, 2; vascular lipoleiomyoma, 1.

DR. LATTES: The clinical and gross information available suggests of course some variant of an adipose tissue growth. Histologically that preliminary impression is confirmed. We have here a mass of tissue which, on low power, shows a lobulated pattern analogous to what we see in normal, fully developed adipose tissue. Each one of these lobules, however, is composed of vacuolated cells which vary greatly in size and which often have the features of immature fetal or embryonal lipoblasts. These cells are smaller and have a "signet ring" appearance with the center vacuole pushing the nucleus at the periphery without, however, flattening it entirely as we see in the normal adult fat. Each one of these lobules is heavily vascularized. Occasionally some lobules lack recognizable adult fat cells, but consist exclu-

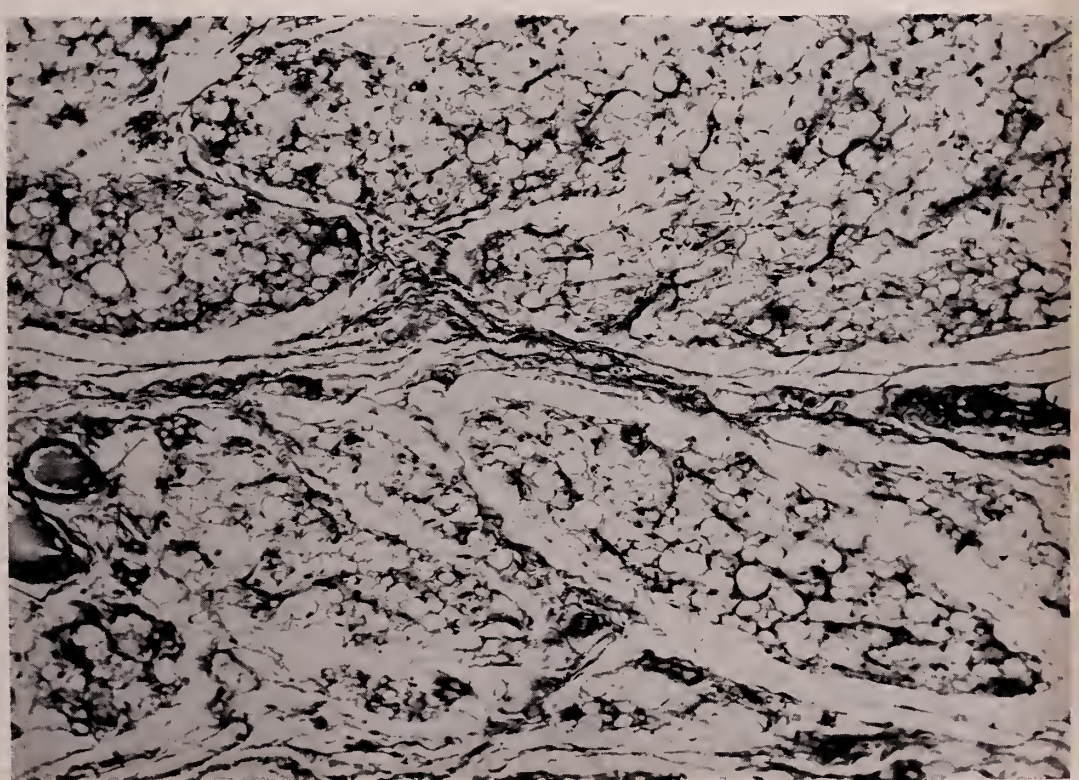

Fig. 8. Case 7. Lipoblastomatosis of neck. Note the lobulated pattern of the fetal fat. Hematoxylin and eosin, X 120.

sively of a capillary vascular meshwork associated with spindle-shaped or stellate cells in a mucoid stroma. There are no bizarre nuclei, no recognizable mitotic activity, and no definitely disorganized neoplastic areas featuring an invasive type of growth. Everywhere the tissue is lobulated. This lobulated pattern is demonstrated very nicely with reticulin stains, which also confirm the vascularity of the individual lobules and also the even distribution of the reticulum fibers within these lobules.

All the features listed are, I believe, important in the correct evaluation of this lesion. Obviously this is not just another case of lipoma, and on the other hand I think it would be incorrect to classify it as a liposarcoma because of its well-organized pattern, which resembles that of normal adipose tissue. Vellios and associates have recently described a case similar to this in an infant, and suggested the term lipoblastomatosis, implying that these are benign tumors of incompletely differentiated adipose tissue similar to the adipose tissue of the fetus, but different from hibernoma. Other terms, such as fetal lipoma or embryonic lipoma, also have been suggested. It is worthwhile to take tumors of this group out of the liposarcoma category, because apparently an entirely benign course is to be expected.

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Case 8

Diagnosis.—Malignant fibroxanthoma of thigh with metastases to inguinal lymph nodes and to viscera (malignant dermatofibrosarcoma protuberans).

Contributors.—Dr. C. L. Green, Hotel Dieu, and Lt. Col. P. L. Child, MC, William Beaumont General Hospital, El Paso.

History.—A 47-year-old man had a slightly painful mass for three months on the anterior aspect of the right thigh. The specimen was a discrete, firm, 2 cm. mass within the corium extending into the subcutaneous fatty tissue, and appearing to have been widely excised. Cut surface showed bright yellow and pale gray, firm, nongranular tissue. Six months later a recurrence was widely excised. The specimen contained a 25 mm. lymph node infiltrated by the same type of tumor tissue. Two months later there was roentgenographic evidence of lung metastases. The patient died within a year.

Submitted Diagnoses.—Fibrosarcoma, 28; malignant fibroxanthoma (malignant dermatofibrosarcoma), 18; leiomyosarcoma, 9; rhabdomyosarcoma, 6; liposarcoma, 3; fibrous xanthoma, 2; neurofibrosarcoma, 1; malignant mesenchymoma, 1.

DR. LATTES: This case is quite unusual because of a series of unexpected developments which are mentioned briefly in the short clinical abstract supplied with this seminar. The section distributed, I believe, is from the tumor removed in June, 1960, from the thigh of this 47-year-old man. It was stated to be about 3 by 2 cm. in dimension, attached to the corium of the skin, and to show a bright yellow and pale gray firm tissue on cut surface.

Histologically this tumor was, at that time, classified as a dermatofibroma, which is a term used for mature fibrous tissue tumors of the corium of the skin which can occasionally extend into the underlying subcutaneous tissue, and which are generally benign except for a tendency toward slow local infiltration. On examining the section from this excision, the predominant features of the growth are the presence of interlacing bundles and whorls of generally elongated cells associated with moderate amounts of collagenous fibers. Toward the surface, the tumor merges imperceptibly with the upper dermis, and in depth, with the subcutaneous fat which is definitely infiltrated. There are foci of inflammatory infiltration. The individual tumor cells vary in size and shape. Most of them are elongated and spindle-shaped but others are irregular in shape. The sizes, especially that of the nuclei, vary considerably and large nuclei with conspicuous acidophilic nucleoli are not too infrequent. Some mitotic figures are noted. Occasionally multinucleated giant cells resembling those seen in fibrous xanthomas, except that the nuclei are atypical, can be observed.

About three months later the tumor recurred at the same site. It was again removed. The sections from that recurrence showed that while the general low power pattern of growth was similar to that of the original tumor, there were considerable differences on medium and high power examination. The tumor was decidedly more pleomorphic. The spindle cells varied greatly in

size; their nuclei were often hyperchromatic; multinucleated bizarre shaped cells were frequent; and mitoses were still rare. As in the original tumor, occasional foam cells could be seen scattered between the bundles of fibroblast-like cells. At the periphery there were some large venous and sinusoidal type blood vessels, some of which appeared to have part of their wall replaced by tumor cells. In the sections available to me from this recurrence, there was no way of studying the pattern of growth of this tumor towards the depths. One interesting feature which was present already in the original tumor is that trichrome stains showed large areas in which collagen was almost absent. However, reticulum fibers were abundant practically everywhere. They followed the interlacing and whorled pattern of the bundles of tumor cells.

There were no further local recurrences. In November, 1960, an enlarged inguinal mass appeared. This was excised and was described as a 25 mm. lymph node containing partly necrotic grayish tumor tissue. Following this the patient was treated with external irradiation, but in the following several months he lost ground steadily, losing weight, becoming progressively weaker, and showing on chest roentgenograms that both lung fields contained multiple lesions interpreted as metastatic tumor. He was admitted to the hospital and, in spite of several blood transfusions, did not improve. A gastrointestinal series revealed a possible lesion of the duodenal region. He later vomited blood several times and in June, 1961, he finally died. An autopsy revealed ulcerated metastatic tumors in the stomach and in the jejunum. In addition there were scattered neoplastic deposits in the soft tissues of the abdominal wall, thigh, and back, and metastases in the pancreas, thyroid, and both lungs. There was no residual or recurrent tumor at the site of origin.

The tissue obtained at autopsy and made available to me showed a much more anaplastic-appearing mesenchymal tumor than the original or first recurrence of the tumor of the thigh. There were areas which easily could be accepted as poorly differentiated fibrosarcoma, since they showed elongated and spindle cells with hyperchromatic nuclei which varied greatly in size and sometimes had coarse chromatin arrangement. These cells also varied in size, and there was little intercellular substance. Scattered in this tissue, which incidentally was quite vascular and still had the original whorled appearance, there still were large numbers of multinucleated giant cells which, however, did not resemble any more benign xanthoma cells. They had hyperchromatic nuclei and large nucleoli, and mitotic activity could be seen in them as well as among the fibroblast-like cells. Some of these bizarre giant cells had a foamy cytoplasm. There were multiple necrotic foci similar to those one sees often in rapidly growing malignant tumors. Frozen section stains for fat showed lipid in groups of tumor cells both of the spindle cell and the giant cell variety. The only additional comment on the morphology of this unusual lesion is that reticulin stains showed a more unevenly distributed production of reticulum than was seen in the original tumor of the thigh.

Summing up, this is the history of a skin nodule which was originally thought to be a harmless dermatofibroma and which later showed a completely unexpected potentiality not only to recur locally but also to metastasize to the inguinal region, probably to the lymph nodes, and eventually through the blood stream, finally killing the patient as a definitely malignant tumor. Parallel with this history of recurrences and then metastases, the histology of this tumor had changed considerably from an exceedingly mature to a definitely immature and poorly differentiated growth. However, the essential morphological features were still present in the recurrences and metastases. These were a tendency to form closely interlacing bundles and whorls and curlicues, the high vascularity, and the presence of giant cells of the xanthoma type which contained lipid.

First, are we justified in accepting the sequence of events as representing always the same tumor which increased gradually in pathological and clinical malignancy? I believe so. Second, how should this tumor be classified and what can we learn from this case? The original diagnosis was dermatofibroma. Obviously it did not behave as would be expected of the common garden variety of dermatofibromas. What does the term dermatofibroma mean? As we know, there are multiple synonyms; namely, sclerosing hemangioma, histiocytoma, fibroxanthoma, and so forth. I believe that the fact that in all the tissue available we also could find giant cells containing lipid associated with the fibroblast-like elements suggests

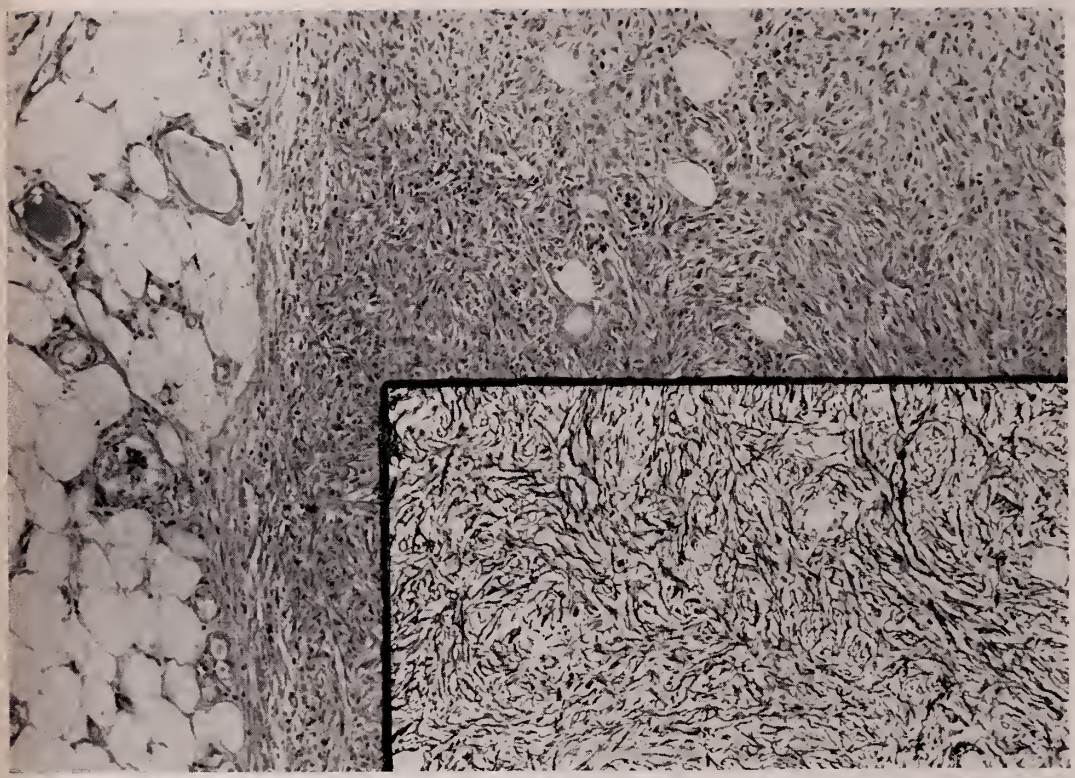

Fig. 9. Case 8. Malignant metastasizing fibroxanthoma. Photomicrograph of original excision. Note the pinwheel pattern, emphasized by the reticulin stain, and the mature appearance of the tissue. Hematoxylin and eosin and Laidlaw stains, X 120.

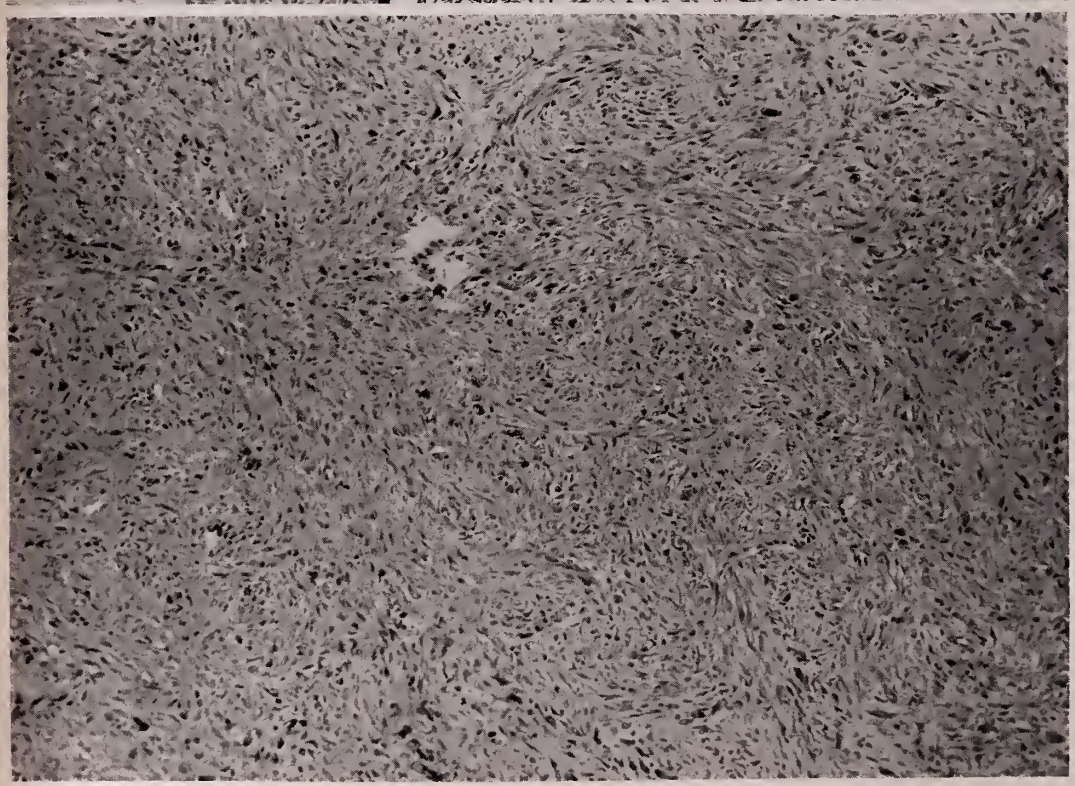

Fig. 10. Case 8. Local recurrence. Note the bizarre nuclei scattered in the tissue. Otherwise, there is a strong resemblance to the original tumor. Hematoxylin and eosin, X 120.

that in this case, at least, a diagnosis of fibrous xanthoma or histiocytoma would have been justified. This then would lead to changing our concept of some invading and potentially metastasizing tumors from a fibroblastic histogenesis to a histiocytic one. There is now good evidence, especially by tissue culture, that histiocytic cells can behave as facultative fibroblasts. This means that they can imitate morphologically the fibroblasts and also produce some amount of intercellular collagenous substance. It was

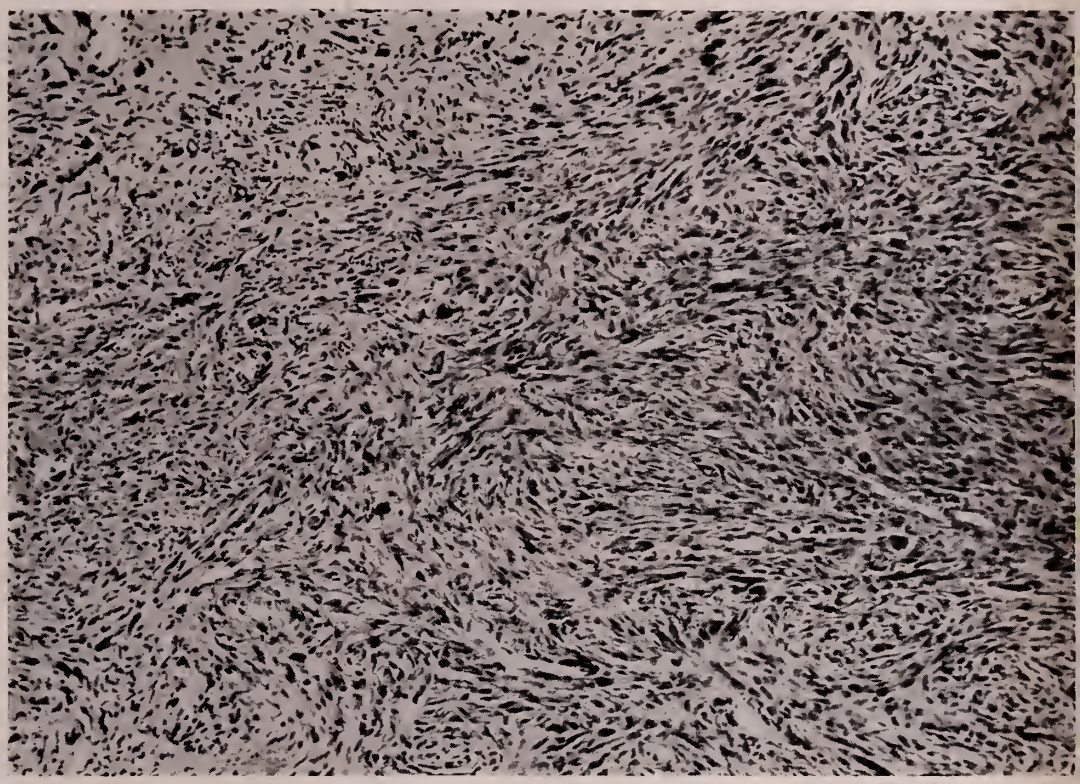

Fig. 11. Case 8. One of the distant metastases shows general anaplasia of the tumor cells. The pattern of interlacing bundles is preserved. Hematoxylin and eosin, X 120.

noted earlier that in many areas even in the original tumor the amount of collagen was minimal. We have recently been alerted to the potential malignant behavior of tumors of the fibroxanthoma group. Dr. A. P. Stout has observed some cases in his material, and after he had pointed out that possibility we have been able to identify a few other cases. Whether we classify the present case as a dermatofibroma which became a metastasizing dermatofibrosarcoma, or as a fibrous xanthoma which became a malignant metastasizing fibrous xanthoma, might be open to argument. We prefer now to use the term malignant fibrous xanthoma for these lesions which, in our opinion, are not pure fibroblastic growths and which, on occasional study in tissue culture done by Drs. Margaret Murray and Luciano Ozzello in our department, have shown a predominant histiocytic type of growth. In the literature there are occasional references to the metastasizing potentiality of dermatofibrosarcoma protuberans, but it certainly is a most rare occurrence. Lastly, one could ask one's self: Could this sequence of events have been predicted and avoided by a more accurate study of the original specimen? I believe that one can honestly say that this sequence of events was unpredictable. In retrospect, to be sure, there were some atypical features in the first specimen but I am sure that I would miss them again if I saw another case of this type.

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Case 9

Diagnosis.—Liposarcoma, partly undifferentiated, of shoulder.

Contributors.—Col. M. W. Bayliss, MC, and Maj. R. L. Creveling, MC, Brooke General Hospital, Fort Sam Houston.

History.—A 28-year-old Negro man noted a small nodule in the right deltoid region shortly after an influenza immunization. This nodule slowly increased in size and was excised. It recurred and a tumor nodule measuring 4 by 2.5 by 2 cm. was removed. Cut section revealed gray-pink soft tissue.

Submitted Diagnoses.—Liposarcoma, 25; fasciitis-fibromatosis group, 18; fibrosarcoma, 10; rhabdomyosarcoma, 7; angiosarcoma

group, 3; other sarcomas, 3; hemangiopericytoma, 3; ganglioneuroma, 1; sclerosing hemangioma, 1.

DR. LATTES: This soft tissue mass which grew rapidly in the region of the shoulder in a 28-year-old man is only apparently well-circumscribed; obviously it grows by infiltration. It is interesting to study different sections because its pattern varies considerably in different microscopic fields. I hope that the sections made available for the seminar were representative of these different fields. I shall describe first the findings which in my opinion are most important for a diagnostic appraisal of the tumor. In most areas this tumor shows a rich and fairly regularly distributed vascular supply, and the tissue situated in the meshes of these blood vessels is often myxoid in type. It features elongated and spindle-shaped cells mixed with pleomorphic elements, some of which are multinucleated. With careful study it is possible to see that some of these pleomorphic cells contain intracytoplasmic vacuoles, and furthermore, occasional "signet ring" cells are seen which are acceptable as lipoblasts. Unfortunately I have no fat stains available but I feel confident that they would be positive in areas such as the one I just described. In other sections, the tumor is extremely anaplastic and undifferentiated. It has lost almost entirely the rich vascular network, and is composed essentially of predominantly elongated and spindle cells with occasional bizarre multinucleated cells. All these cells have hyperchromatic nuclei, few of them are vacuolated, and mitoses are fairly frequent. In some rare places these bizarre cells have an abundant eosinophilic cytoplasm and one might think of the possibility that these areas are rhabdomyosarcomatous in nature. In my opinion this cannot be proven. There are no cross-striations nor evidence of myofibrils or of intracytoplasmic granules, and of course I am heavily influenced by the typical liposarcomatous pattern elsewhere. Therefore, I do not think it is justified to classify this tumor as a malignant mesenchymoma but only as a liposarcoma with extremely undifferentiated areas. An analogous comment could be made for other microscopic findings which one might interpret as suggestive of osteogenic sarcoma. There are in fact microscopic fields in which the ground substance surrounding the tumor cells is homogeneous and hyaline. I do not think that we can prove that this is real osteoid, and therefore, the same comments apply as those I made for the possible rhabdomyosarcomatous components. I be-

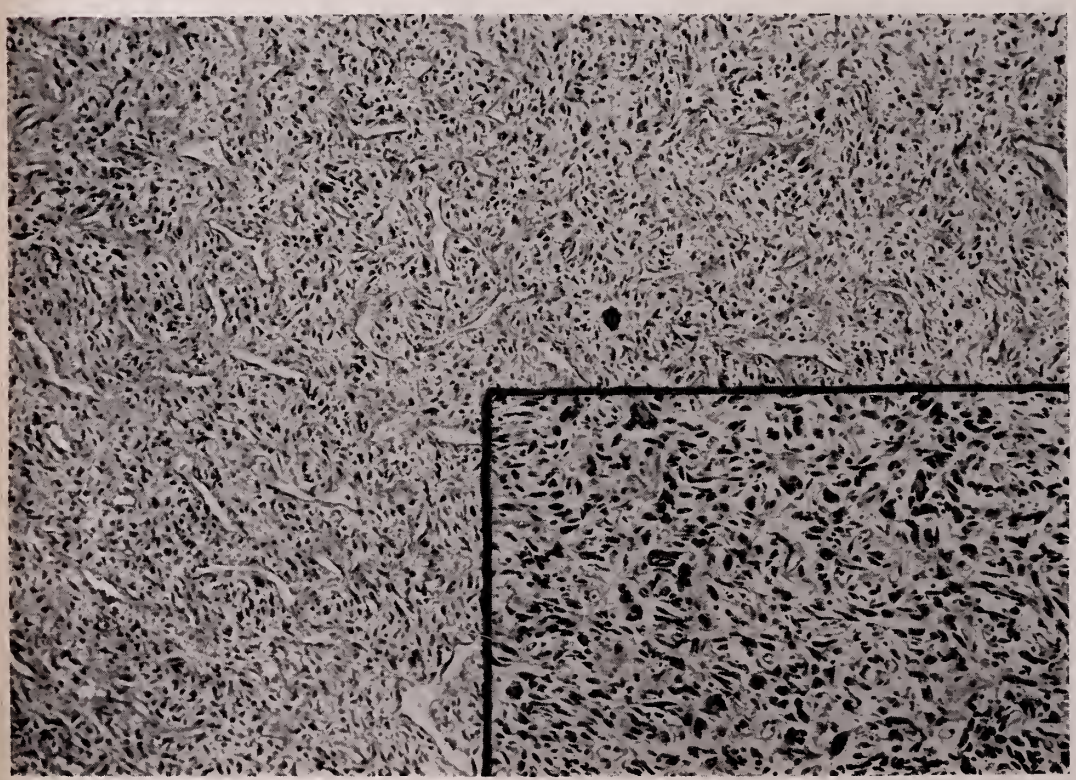

Fig. 12. Case 9. Partly undifferentiated liposarcoma. Note the characteristic vascular pattern. Some of the cells contained distinct vacuoles. The insert at lower right shows a more anaplastic area. Hematoxylin and eosin stain, X 120.

lieve the tumor is highly malignant and able to metastasize to distant areas.

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Case 10

Diagnosis.—Leiomyosarcoma of retroperitoneal region.

Contributors.—Col. M. W. Bayliss, MC, and Maj. R. L. Creveling, MC, Brooke General Hospital, Fort Sam Houston.

History.—A 78-year-old white woman was hospitalized with a pelvic mass. For two years she had noted a large abdominal mass which gave her no difficulty until two weeks before operation, when she noted pain and tenderness over the mass and the sides of her abdomen. Intravenous pyelogram revealed minimal dilation of the right ureter. Grossly the tumor measured 18 by 15 by 15 cm. and was composed of firm tissue interspersed with softer, gelatinous areas, and hemorrhage.

Submitted Diagnoses.—Leiomyosarcoma, 46; leiomyoma, 7; liposarcoma, 6; mixed mesodermal tumor, 3; myosarcoma, 2; fibrosarcoma, 2; other sarcomas, 4; chordoma, 2.

DR. LATTES: This is a tumor composed of elongated spindle cells with nuclei which have blunted ends and with eosinophilic cytoplasm which contains intracytoplasmic fibrils. These cells are arranged in bundles which curve around and interlace with each other. In most places where the tumor is well preserved, special stains show a minimal amount of intercellular collagen substance, and they also show that the reticulum fibers have a striking tendency to run parallel to the long axis of the bundles without wrapping themselves around the individual cells. In addition, trichrome stains show recognizable myofibrils.

The nuclei of the tumor cells vary somewhat in size. In occasional focal areas they are hyperchromatic. In these areas there is approximately an average of one mitosis per high power field. Bizarre nuclei are seen occasionally in these fields. There are foci of necrosis. In the sections taken it is not possible to evaluate invasiveness. Around several blood vessels, tumor cells are seen making cuffs just outside the endothelial lining.

I believe that this is obviously a tumor of smooth muscle and in addition I believe it is malignant. I think there will be little

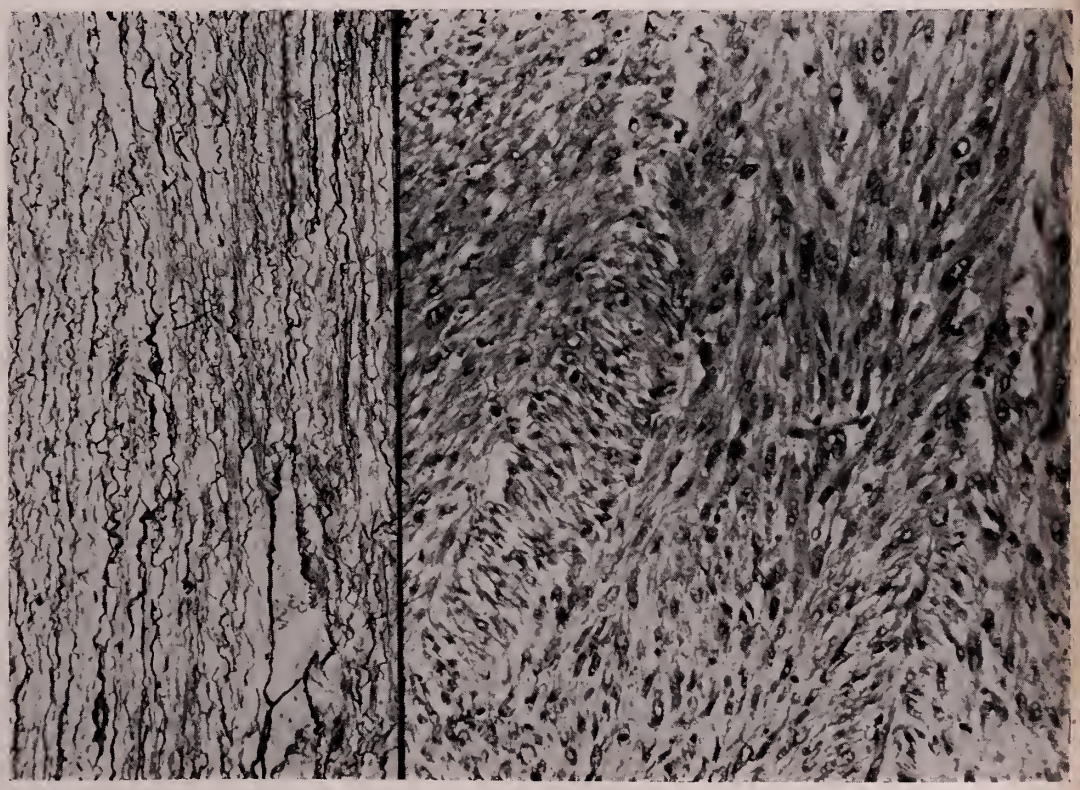

Fig. 13. Case 10. Leiomyosarcoma of retroperitoneum. At left, the reticulin stain shows clearly the parallel arrangement of the fibrils. Hematoxylin and eosin and Laidlaw stains, X 180 (insert: X 120).

controversy about the interpretation of this tumor. However, it might be worthwhile discussing briefly the histological criteria that one may use to differentiate benign from malignant smooth muscle tumors. Some of them are obviously malignant; namely, those which grow rapidly, infiltrate widely the surrounding tissues, and show nuclear anaplasia and a high mitotic rate. Others appear to be well differentiated and apparently encapsulated; mitotic activity may be quite low, and of course one would then conclude that the tumor belongs to the benign group. Some of these, however, later prove to be definitely malignant in that they give rise to distant metastases. I have had that experience a few times with smooth muscle tumors of the gastrointestinal tract. In these instances I do not know of any completely reliable histological criterion for the differentiation between benign and malignant. Other difficulties of course arise when myofibrils are not easily recognizable. The criterion mentioned above of the parallel arrangement of the reticulum fibrils and the paucity of intercellular collagen in the individual bundles has been of great help in many occasions in differentiating smooth muscle tumors from fibrosarcomas or other spindle cell tumors. Lastly, it is worth mentioning here the great frequency of leiomyosarcoma in the retroperitoneal and mesenteric area as contrasted to all other soft tissue regions (exclusive of gastrointestinal tract and female genital systems). Out of 105 leiomyosarcomas of soft tissues, Dr. Stout found that 61 occurred in the retroperitoneal or peritoneal areas. A last comment is that since a radical surgical excision is practically impossible in the retroperitoneal area, the incidence of recurrences and death from tumors in this region is higher than elsewhere in the soft tissues.

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Case 11

Diagnosis.—Fibrous xanthoma (histiocytoma) of orbit, locally invasive.

Contributor.—Dr. Lattes.

History.—A 62 year-old white woman first noted swelling in her left eye in 1949. A mass was partially removed. In 1955 the swelling recurred and a mass in the region of the left lacrimal gland was removed. In 1958, a wider operation was done for a new recurrence, but the globe was preserved. The tumor recurred again and, in order to remove it completely, the eye was enucleated in 1961. There were no systemic symptoms at any time.

Submitted Diagnoses.—Fibrous xanthoma-hemangioma-histiocytoma group, 20; vascular sarcoma (various types), 17; hemangiopericytoma, 15; others, benign, 8; fibrosarcoma, 6; myosarcoma, 3; other sarcomas, 2.

DR. LATTES: This is an unusual case. This 62-year-old woman in 1949 had a nodule removed from her orbit in another hospital. At that time this was classified as a "benign blood vessel tumor." A recurrence was operated on in December, 1958, and since a new recurrence with extensive involvement of the orbit took place after this, it was felt necessary to perform a complete clean-up of the orbit with enucleation of the eye in November, 1961.

On review of all the histological material available, it is now felt that the essential microscopic features of this tumor have not changed during the three years in which the patient was followed. The description here will then be a comprehensive description, and not refer only to the last operation.

On low power this is a poorly circumscribed tumor which sends extensions in the surrounding tissues, namely towards the conjunctiva and into the muscular and fatty tissue of the orbit. On higher power the tumor is found to consist of a highly vascular tissue. The vessels are generally quite inconspicuous and in some places special stains are necessary to bring out this vascularity. Between the vessels, which consist only of endothelium-lined spaces, are collections of spindle-shaped, oval-shaped, and sometimes comma-shaped cells with vesicular nuclei which vary in size and have eosinophilic, sometimes granular, cytoplasm. Some of the cells have a clear, faintly foamy cytoplasm and, with special stains from these areas, fatty droplets can be demonstrated in the tumor cells. Mitoses are quite rare. Trichrome stains show a feature which is of importance; namely, an almost absolute absence of collagen framework in the most cellular portions of the tumor. This seems to militate

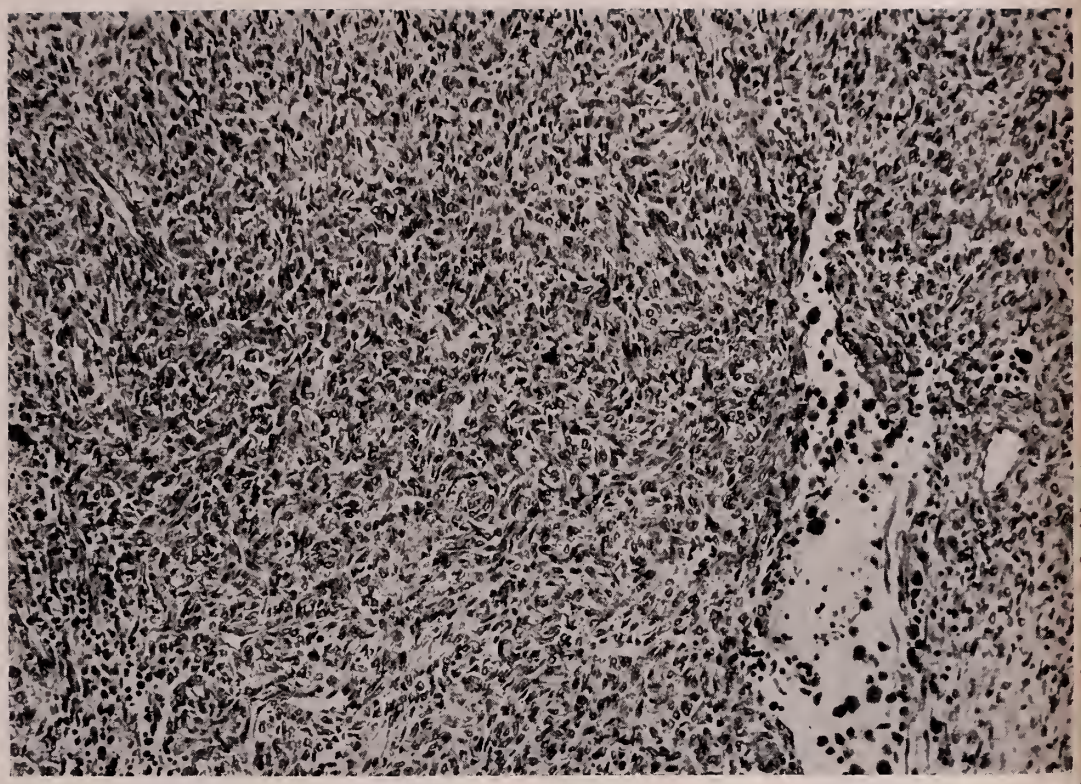

Fig. 14. Case 11. Fibrous xanthoma of orbit. Hematoxylin and eosin, X 175.

against a fibroblastic nature for this growth. On the other hand there is a very abundant reticulum feltwork which outlines the small blood vessels but which sometimes does not penetrate within the individual nests of tumor cells.

At the time of the last operation a portion of this tumor was studied with the tissue culture method. Dr. Ozzello reported that there was an outgrowth of cells which had the features of histiocytes. They showed an ameboid pattern of growth, had branching cytoplasmic processes, and active phagocytosis of lithium carmine.

We decided to classify this tumor as a fibroxanthoma or histiocytoma, with local invasive potentialities. This was also shown by the gross appearance of the tumor that presented as at least two main tumor masses of yellowish color, extending superiorly toward the dura in the supra-orbital region and anteriorly toward the conjunctival surface above the eyeball. The largest mass measured 2.5 cm. in diameter and near it was a separate mass 1.5 cm. in diameter.

This case was of particular interest because of the unusual location of the tumor and because even though it probably will not behave as a metastasizing lesion, locally it had to be handled as a malignant tumor and a total exenteration of the orbit was performed.

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Case 12

Diagnosis.—Chondroblastoma, benign, of rib.

Contributors.—Drs. N. H. Jacob and G. C. Mani, Santa Rosa Medical Center, San Antonio.

History.—A 46-year-old man complained of pain in the right side of his chest for five years. Radiographs revealed a mass in the sixth rib. The resected rib contained a 7 by 5 by 3.5 cm., moderately firm, tumor mass. The mass cut with a gritty sensation but the entire specimen could be transected with a knife. The cut surface was gray to yellow-gray, with scattered firmer white areas up to 1 mm. in diameter. The cortical portion of the bone was thinned and in some areas completely absent.

Submitted Diagnoses.—Chrondroblastoma, 62; chondrosarcoma, 4; myeloma, 2; chloroma, 1; aneurysmal bone cyst, 1; osteosarcoma, 1; reticulum cell sarcoma, 1.

DR. LATTES: This is a tumor of the sixth rib in a 46-year-old man. The tumor mass was large; it measured 7 by 5 by 3.5 cm.

Histologically, the findings consist of a highly cellular pattern with foci of amorphous calcification, apparent encrustation of intercellular fibers with calcium salts, definite chondroid and cartilaginous areas, and occasional areas of osteoid appearance. In some places the calcification presents itself as a fine lacework in which are scattered tumor cells. These foci sometimes merge with areas with cartilaginous ground substance. There are multinucleated giant cells similar to those seen in giant cell tumors of bone. The individual tumor cells vary somewhat in size and shape. Generally, they are irregularly polygonal and show eosinophilic cytoplasm. Also, their nuclei vary in size and shape—some of them are round and vesicular, others are indented or irregularly lobate. Mitoses are extremely rare. When the chondroid ground substance is present these cells are easily recognizable as chondrocytes. The transition between osteoid and chondroid is seldom sharp, so there is gradual merging from one to another tissue type. However, mucicarmine stains show that mucicarminophilia is present only in the more definitely cartilaginous areas.

The above description is in perfect keeping with the tumor which is now called benign chondroblastoma. It is well known that originally these tumors were considered part of the giant cell tumor group and classified as "chondromatous giant cell tumor." The unusual feature here is that it did not occur at or near the epiphysis of long bones as apparently the absolute majority of these tumors have occurred so far. Jaffe, Lichtenstein[4] and Coley are not familiar with any cases occurring away from the epiphyseal cartilages. In our files there are 17 cases of benign chondroblastoma. Most of these are outside cases submitted for consultation. Among them there is one lesion in a rib, one in the sternum, one in the metacarpal, and one in the calcaneus bone. All the others occurred in the epiphysis of long bones. In addition to the case of chondroblastoma of the rib that exists in our files, the rare occurrence of this tumor in ribs is mentioned by Ackerman[1] in his recent fascicle on bone tumors. Dahlin,[3] in his book on bone tumors, mentions having seen two chondroblastomas occurring in ribs.

I believe that this lesion is benign. Of course the important contribution that was given by Codman[2] and by other authors has

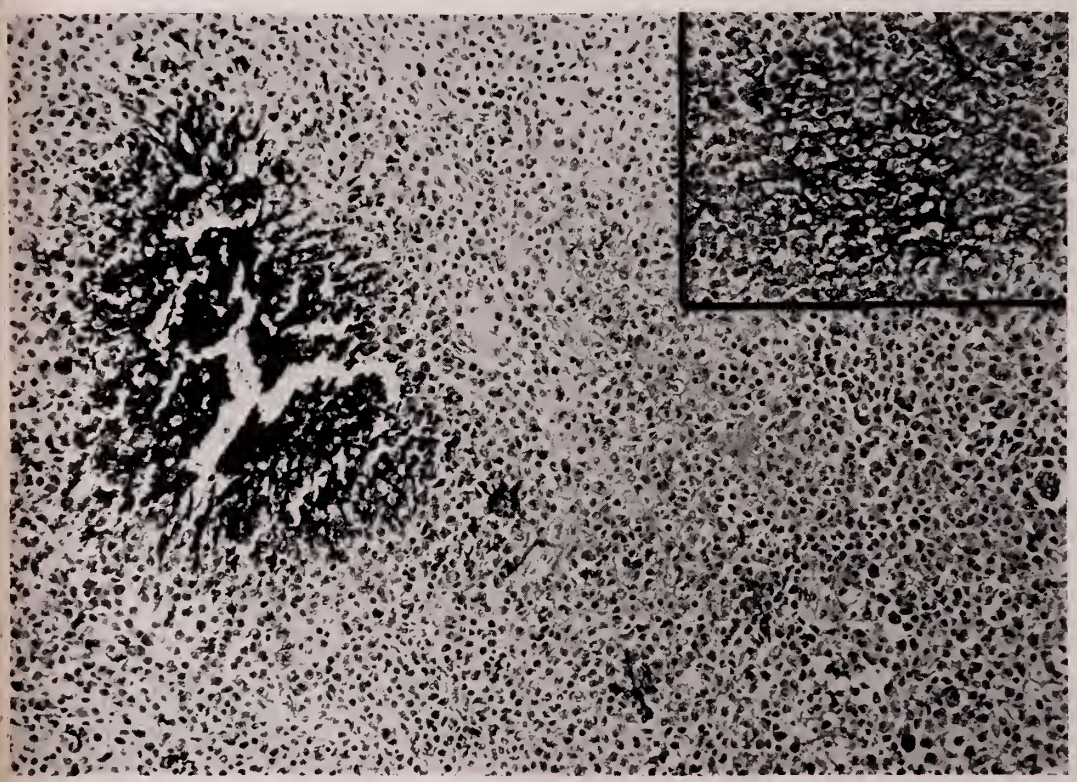

Fig. 15. Case 12. Chondroblastoma of rib. Chondroid ground substance is seen in the central area. Some giant cells can be seen at the right. A lacework of calcified fibrils is present at left and in the insert at upper right. Hematoxylin and eosin, X 120.

been to point out that these tumors should not be confused with chondrosarcomas.

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Case 13

Diagnosis.—Fibrous xanthoma (dermatofibroma) of skin and subcutaneous tissue of mammary region.

Contributors.—Drs. A. O. Severance and H. A. Van Auken, Baptist Memorial Hospital, San Antonio.

History.—A 44-year-old Negro woman had a mass in the right breast, thought to be a lipoma, of four years' duration with onset following an injury. It was excised from over the right pectoralis muscle. The specimen measured 6.5 by 5.5 by 5.5 cm. The cut surface was homogeneous grayish yellow.

Submitted Diagnoses.—Dermatofibrosarcoma, 34; desmoid-fasciitis-fibroxanthoma group, 14; fibrosarcoma, 9; neural tumors (benign), 8; neural tumors (malignant), 4; leiomyosarcoma, 1; pericytoma, 1.

DR. LATTES: This tumor has a beautiful, regular, so-called storiform pattern. It consists of fibroblast-like cells with abundant cytoplasm arranged in small whorls or "pinwheel" pattern. Mitoses are extremely rare. The tumor is fairly vascular and its vascularity is demonstrated better with trichrome stains, with which the erythrocyte-containing narrow blood vessels stand out more conspicuously; they are often located in the center of the pinwheels. That the tumor is not an encapsulated one is shown by the fact that, here and there, remnants of fat lobules can be seen surrounded entirely by neoplastic cells. Occasional cells are larger than others due to more abundant clear cytoplasm. However, no satisfactory foam cells are seen with certainty. It is unfortunate that no tissue was available for fat stains, because very probably there would have been groups of spindle-shaped elements containing lipid droplets. The tumor is not really a tumor of the breast, but rather a tumor of the skin and subcutaneous tissue of the mammary region. This is shown by the fact that pigmented epidermis overlies directly the neoplastic

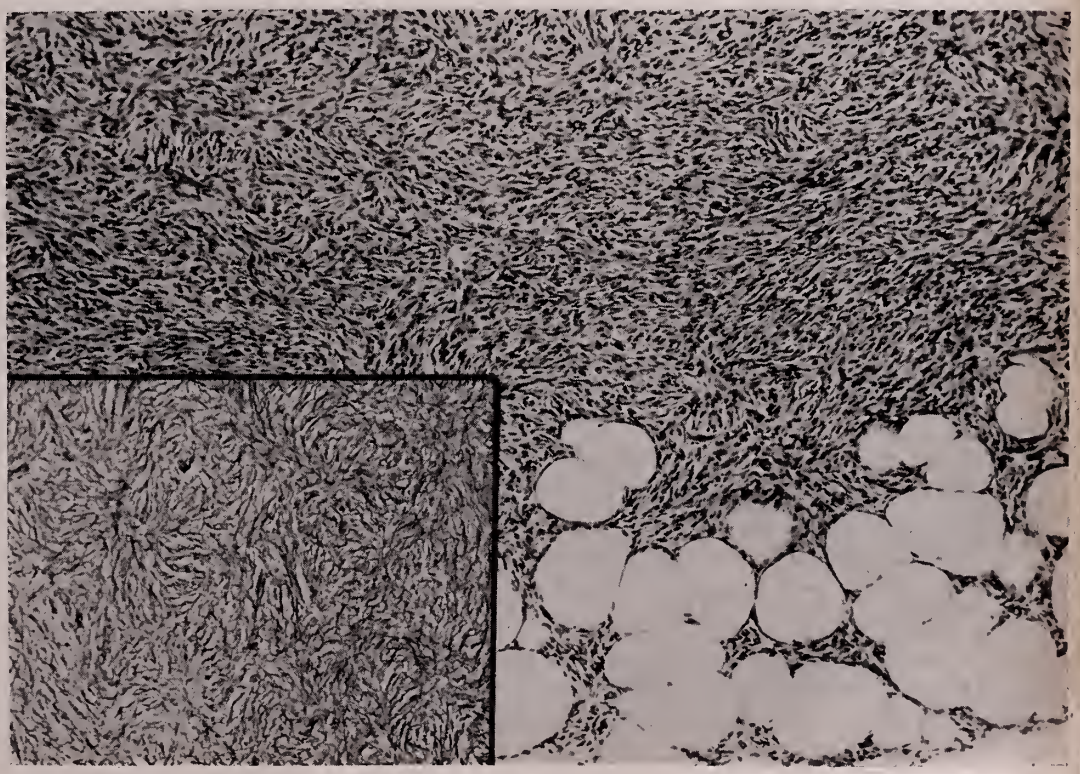

Fig. 16. Case 13. Fibrous xanthoma of mammary region. The tumor is locally invasive. Insert shows well the "pin wheel" pattern. Hematoxylin and eosin and Laidlaw stains, X 120.

mass. There is a moderate diffuse collagen production.

This is a tumor which, in my opinion, belongs to the group of so-called fibrous xanthomas or histiocytomas. When they occur in the skin, the term dermatofibroma is the one used most commonly. The name dermatofibrosarcoma has been applied to those tumors which show rapid and more definitely invasive growth. The majority of these lesions are benign or at most locally infiltrating problems. Distant metastases are exceptional. This point is discussed in connection with case 8 of this seminar.

This lesion had already been submitted for consultation to Dr. Stout in 1950. At that time he had classified it as a well differentiated fibrosarcoma. Because of this I took the liberty of asking his opinion now, and he agrees that at this time he would have classified it as a fibrous xanthoma and not as a fibrosarcoma.

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See also: References listed under Case 8.

Case 14

Diagnosis.—Stromal endometriosis (? endometrial sarcoma) of uterus.

Contributors.—Drs. C. L. Green and M. L. Hart, Hotel Dieu, El Paso.

History.—A 54-year-old woman underwent hysterectomy for sudden profuse postmenopausal bleeding. The cut surface of the globular uterus showed diffuse infiltration of the myometrium with tumor which blended with an intracavitary mass. The serosa was spared but tumor extended into the blood vessels of the broad ligament. The atrophic ovaries contained cysts filled with hemorrhagic fluid and having a light yellow lining. There had been no hormone therapy prior to surgery.

Submitted Diagnoses.—Endometrial stromal sarcoma group, 45; stromal endometriosis group, 13; hemangiopericytoma, 7; leiomyosarcoma, 4; angiosarcoma, 1; intravascular leiomyoma, 1.

DR. LATTES: This partly polypoid, partly invasive intra-uterine tumor in a 54-year-old postmenopausal woman shows histologically a highly cellular tissue with little recognizable pattern of growth. The majority of the tumor cells have either round or oval vesicular nuclei and poorly outlined, scanty eosinophilic cytoplasm. They are closely packed together and they show some tendency to grow in whorls and bundles. Mitoses are extremely rare. In some places the tumor cells form small rosette-like nests. The neoplasm has a definitely infiltrative pattern of growth as shown by remnants of bundles of smooth muscle, probably from the myometrium, which can be found here and there in the neoplastic masses. Reticulin stains show abundant reticulin production which penetrates practically between cell and cell. This pattern does not suggest definitely an epithelial tissue; on the other hand, trichrome stains show no recognizable collagen except for the perivascular adventitia in the trabeculae. No myofibrils could be seen in the tumor cells.

It is to be emphasized again that this is essentially a tumor composed of one cell type, and because of that it does not belong in the group of so-called mixed mesodermal tumors of the uterus. According to Ober's classification,[5] this is a "pure, homologous" type of mesenchymal tumor of the uterus.

The differential diagnosis here is between so-called stromal endometriosis, endometrial sarcoma, and leiomyosarcoma. I believe that leiomyosarcoma is the least likely, since the tumor cells are quite uniform in appearance and much smaller than those usually seen in either benign or malignant smooth muscle tumors. No myofibrils were discovered and the pattern of reticulum fibers is not that seen in smooth muscle tumors. As for endometrial sarcoma versus stromal endometriosis, perhaps the difference is more one of semantics than a real one. We know that cases classified as stromal endometriosis have recurred locally, invaded vaginal wall or urinary bladder or peritoneal structures in general, or have recurred in laparotomy wounds. On the other hand we know that cases classified as endometrial sarcomas have seldom given distant metastases; more frequently they have shown their malignant behavior by extensive local infiltration. The lack of mitotic activity, the fairly monotonous cell type, and the lack of evidence of local destruction and necrosis would suggest a classification of stromal endometriosis.

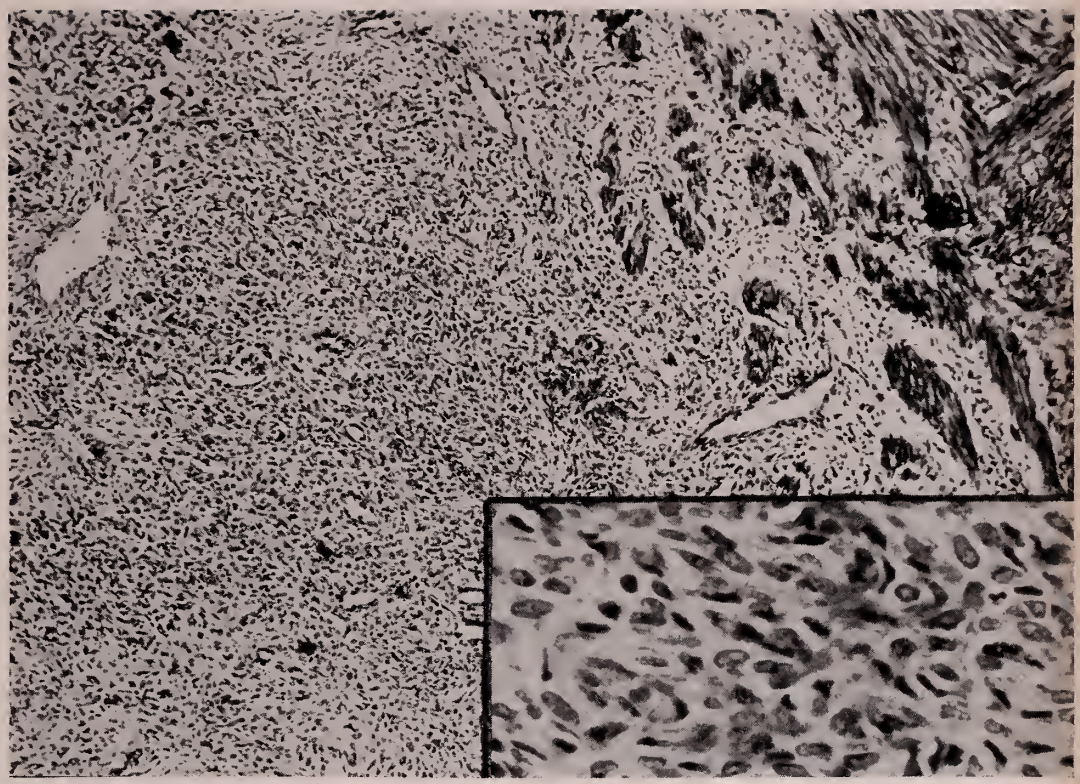

Fig. 17. Case 14. Endometrial sarcoma. The photomicrograph shows invasion of myometrium; the insert shows cell type. Hematoxylin and eosin stain, X 120 (insert: X 610).

Fig. 18. Case 14. Reticulin stain shows the rich and regular reticulin meshwork. Laidlaw stain, X 180.

However, from the information given we know that veins in the broad ligament were invaded by tumor. In the sections available to me I have not seen any endolymphatic or any endovascular permeation. Whether blood vessel invasion necessarily leads to distant embolic metastasis is, of course, unsettled. One point in favor of classifying this case as endometrial sarcoma is its occurrence in a postmenopausal woman. The majority of the reported cases of stromal endometriosis occurred in a younger age group (35 to 50).

Personally, I believe that the terms endometrial sarcoma and stromal endometriosis are often interchangeable, and that in this particular case we are dealing with a lesion that is probably of slow and essentially local invasive growth. As everybody knows there is still considerable controversy on the question of histogenesis of the disease called stromal endometriosis and/or endometrial sarcoma. Good discussions can be found in articles by Park[6] and in an excellent article by Hunter and associates.[4]

Park was not even sure that the cells of the lesion can be definitely identified as endometrial stroma cells, and suggested that they may represent a transition between stroma cells and muscle cells. Hunter, on the other hand, is in favor of identifying this tissue as definitely consistent with endometrial stroma. Hunter also states in another paper[3] that "the course and outcome of uterine stromal endometriosis are unpredictable."

Lastly, I would like to mention that occasionally cases which have the morphological features of hemangiopericytoma have been labeled endometrial sarcoma or stromal endometriosis. This case does not belong in that group.

Summing up, I believe we have here a potentially malignant tumor which morphologically has some resemblance to the endometrial stroma.

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Case 15

Diagnosis.—Rhabdomyosarcoma (sarcoma botryoide) of prostate.

Contributors.—Col. M. W. Bayliss, MC, and Col. J. L. Hansen, MC, Brooke General Hospital, Fort Sam Houston.

History.—A 37-year-old white man had urinary obstruction. Transurethral resection yielded 26 gm. of firm to gelatinous tissue. The prostate, urinary bladder, seminal vesicles, and adjacent lymph nodes were then removed. A 9 by 5 by 5 cm., lobulated, glistening, gray-tan tumor with minute areas of hemorrhage replaced most of the right lobe of the prostate and protruded into the lumen of the bladder.

Submitted Diagnoses.—Rhabdomyosarcoma, 45; undifferentiated carcinoma, 11; carcinosarcoma, 6; malignant mesenchymoma, 5; teratoma, 3; other, 2.

DR. LATTES: The histological findings in this tumor, which grossly presented as polypoid masses protruding into the lumen of the bladder and replacing most of the substance of the right lobe of the prostate, are quite characteristic and interesting. Most of the tumor is undifferentiated and consists of diffuse collections and poorly outlined cords of cells, the majority of which have irregularly shaped hyperchromatic nuclei and scanty cytoplasm. Among them, however, there are other cells containing more abundant strongly acidophilic cytoplasm. Some of these are elongated and occasionally ribbon-shaped. In addition, multinucleated giant cells, again with strongly acidophilic cytoplasm, are seen and also cells which have multiple vacuoles. More infrequently, easily recognizable rhabdomyoblasts with beautiful cross-striations can be observed. They resemble the skeletal muscle fibers seen in the early fetal development. These nests and collections of cells referred to above are distributed in a somewhat myxoid supportive ground substance. The tumor infiltrates diffusely the prostatic gland and the smooth muscle of the prostatic urethra. In one of the sections it is also seen to form a polypoid mass protruding in the lumen of a seminal vesicle. Here and there, the collections of neoplastic rhabdomyoblasts are arranged in a so-called alveolar fashion. Mitotic activity varies in the different areas but is generally moderate to low. Another interesting finding is shown by the reticulin stains. They show little or no reticulum fibers within the individual tu-

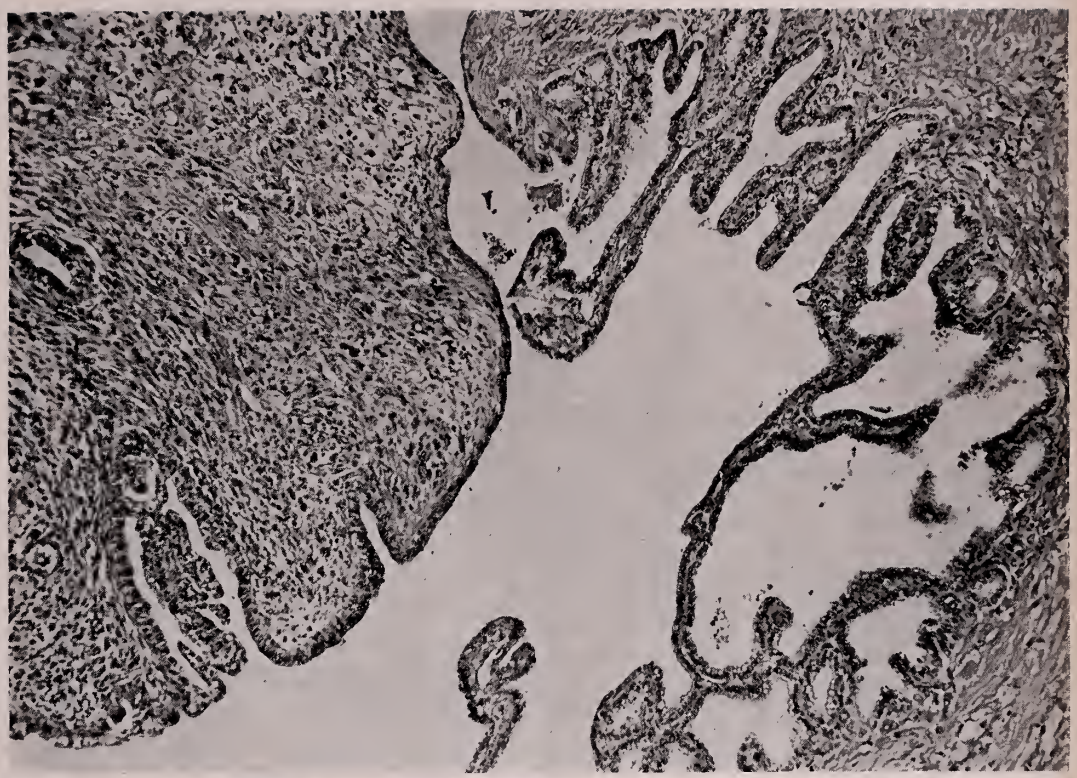

Fig. 19. Case 15. Embryonal rhabdomyosarcoma of prostate and seminal vesicles. The photomicrograph shows a polypoid (botryoid) portion of the tumor projecting in the lumen of a seminal vesicle. Hematoxylin and eosin stain, X 105.

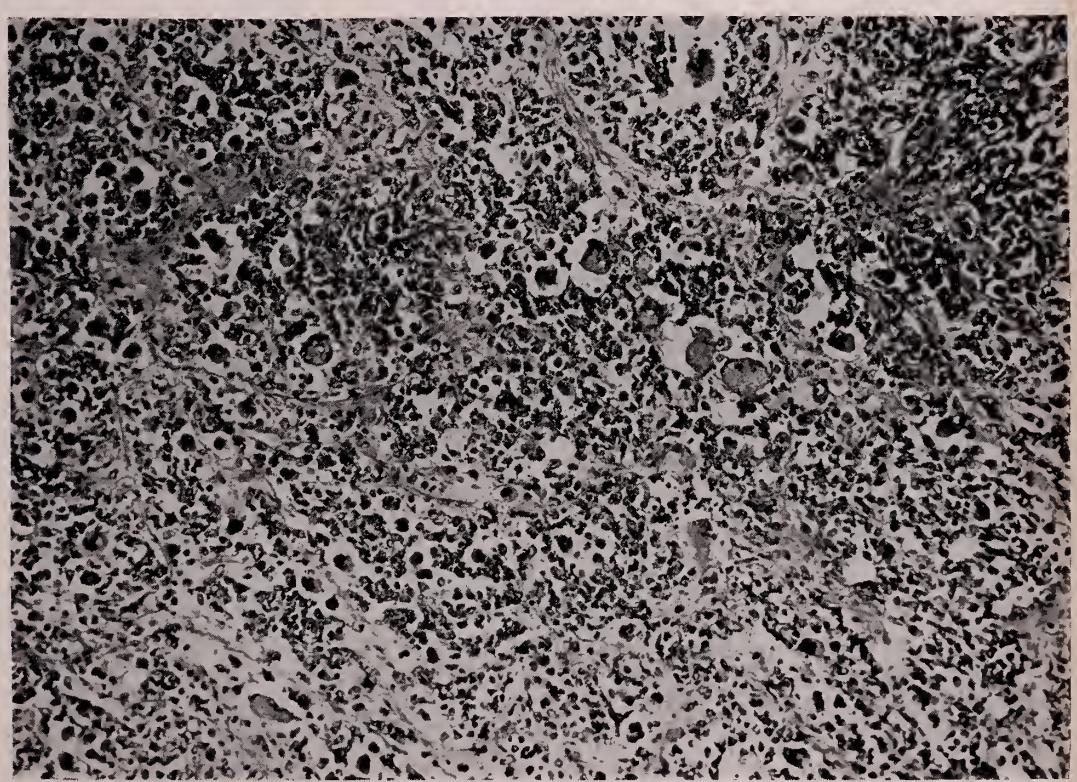

Fig. 20. Case 15. Pleomorphic cell population, with large, rounded rhabdomyoblasts, and alveolar pattern is seen. Hematoxylin and eosin, X 120.

mor nests described above, emphasizing thus the "alveolar" pattern of the tumor. PAS stain was not very informative except that it showed that the cytoplasm of the more recognizable rhabdomyoblasts, especially those with cross-striation, was faintly PAS positive.

This is a beautiful example of embryonal rhabdomyosarcoma of the botryoid type occurring in an area where skeletal muscle is

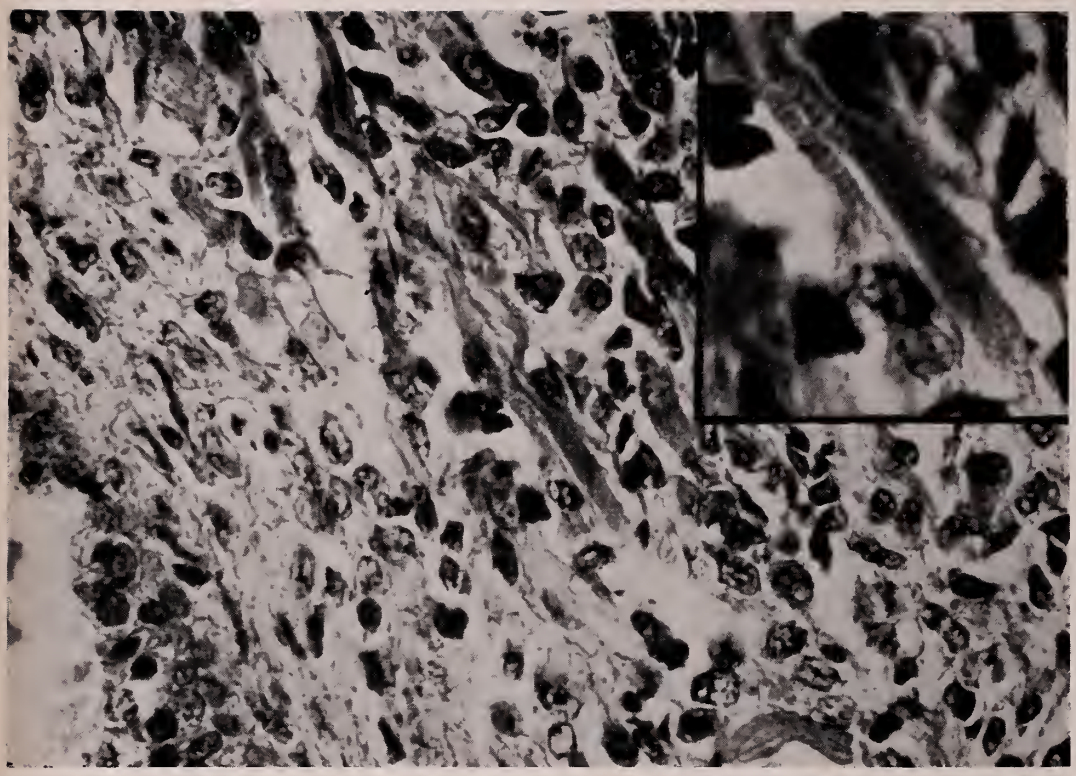

Fig. 21. Case 15. Note the ribbon-shaped rhabdomyoblasts with cross-striations. Insert is a higher magnification of the central area. Hematoxylin and eosin, X 610 (insert: X 1,215).

generally completely absent. The term botryoid should be reserved for those rhabdomyosarcomas which arise in the vicinity or from mucous membrane and produce polypoid masses. In these sections there are areas that would qualify for the term alveolar rhabdomyosarcomas and others that would be better defined as purely embryonal rhabdomyosarcoma, as well as foci acceptable as the pleomorphic, or adult type, or rhabdomyosarcoma. I would also like to point out that in all the sections that I have examined there are several microscopic fields in which the tumor is so undifferentiated that no recognizable rhabdomyoblasts can be seen. In these areas the diagnosis would be either missed or based on previous experience. We have then here an example that these different names actually are labels of different variants of what is basically the same tumor, namely a malignant tumor of cells able to differentiate into rhabdomyoblasts. There are some good recent papers on this tumor in general and some references are appended. One last word is in regard to the involvement of the urogenital system. Embryonal rhabdomyosarcoma with or without botryoid features not infrequently has involved the male and female urogenital system. The majority of patients are children or infants. However, its occurrence in later decades such as in the present case is a well-known possibility. Generally these are tumors that are definitely malignant, but the interesting point is that many of them tend to grow by local extension and infiltration rather than by distant metastases. In our files we have records of 10 rhabdomyosarcomas of the prostate, 14 in the urinary bladder, and 11 others between the spermatic cord and scrotum. The majority of these were in patients age 20 and younger.

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Case 16

Diagnosis.—Carcinoma, muco-epidermoid type, of maxillary sinus following thorotrast.

Contributor.—Dr. Lattes.

History.—A 45-year-old man developed a fistula following dental extractions from the right upper alveolar region. There was progressive loss of teeth from the upper right

side, and the soft palate was swollen. Roentgenographic examination revealed foreign material in the right antrum as well as destruction of the bony wall. The entire antrum was replaced by homogeneous, gritty tissue. Biopsies were taken. A right maxillectomy was done. The specimen was found to emit radiations characteristic of thorium and its by-product.

Submitted Diagnoses.—Squamous cell carcinoma (thorium induced, 22), 66; mucoepidermoid carcinoma (thorium induced, 2), 3; respiratory epithelium carcinoma, 1; tissue reaction to thorium, 1; adamantinoma, 1.

DR. LATTES: I added this case to the seminar not because the sections presented any difficulty or problem in the histological classification of the tumor, but because this was an opportunity to discuss an unusual but well established mechanism in the production of some carcinomas of the paranasal sinuses. This 45-year-old man was first seen in our hospital in May, 1961, because of a so-called fistula which followed two dental extractions in the right upper alveolar region. At that time he had symtoms of what was thought to be a chronic sinusitis which persisted in spite of multiple irrigations, various medications, and antibiotic administration. When seen here he complained of pain in the area of the right cheek, inability to chew, and progressive loss of teeth on the upper right side. The two upper right incisors were loose, the soft palate was swollen on the right side, and there was a fistula through which a probe could be passed upward from the right gingival labial sulcus. Roentgenographic examination showed what appeared to be a foreign body in the right antrum, as well as destruction of the bony wall of the same right antrum. The foreign body was interpreted as being probably a fragment of a tooth or of a dental filling. Because of this the patient was scheduled for a Caldwell-Luc operation. The possibility of a tumor of the maxillary sinus was considered preoperatively. At this operation no calcified bone was encountered in entering the right maxillary sinus. The entire antrum had been replaced by a homogeneous-appearing, gritty tissue. Biopsies were taken. Since the biopsies proved the presence of a carcinoma, it was decided to perform a right maxillectomy, and this was done on Oct. 10, 1961. The sections submitted for this seminar come from this specimen. They showed extensive invasion in the pterygoid area, the infratemporal area, and the hard palate.

Because of previous experience with analogous cases of carcinoma of the maxillary antrum associated with metallic type of foreign body, this specimen was examined and

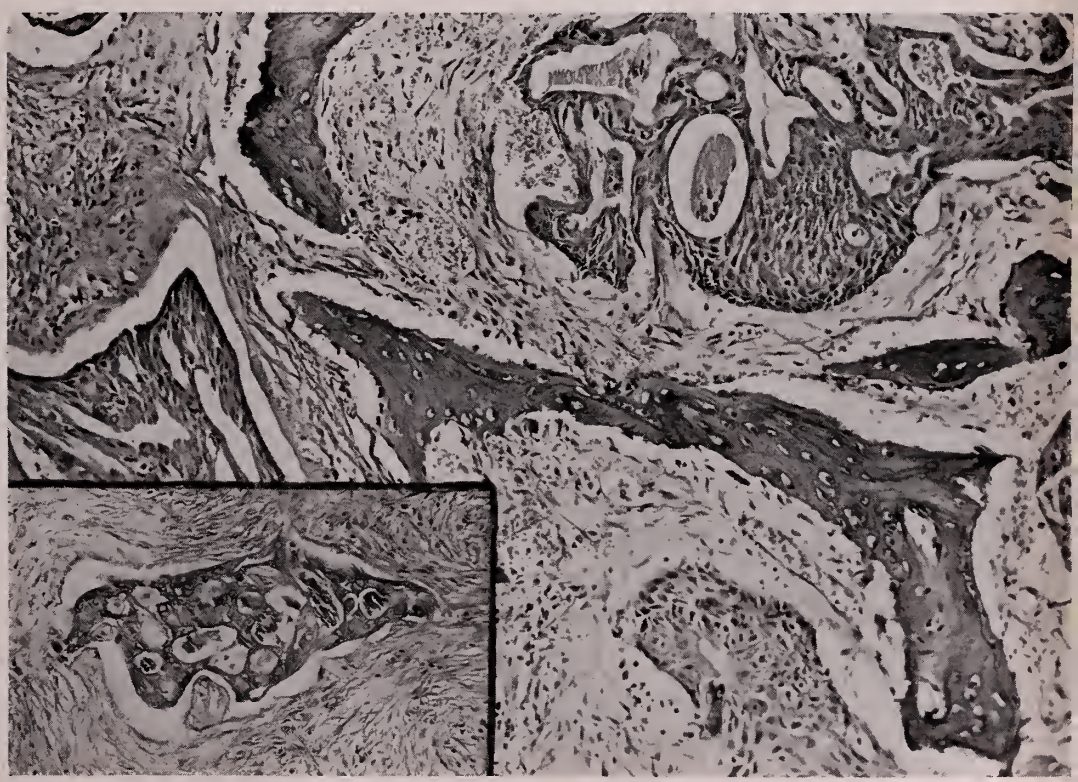

Fig. 22. Case 16. Muco-epidermoid carcinoma of maxillary sinus. Photomicrograph shows invasion of bone by predominantly squamous carcinoma. Insert shows a nest of tumor cells with intra- and extracellular mucicarminophilic secretion. Hematoxylin and eosin and mucicarmine stains, X 120.

studied by the Radiological Research department and found to emit radiation of the gamma ray type with the characteristic pattern of thorium and of its by-products. It was concluded that the specimen contained thorium in minimal amounts, the exact quantity of which could not be determined. This patient had only a vague recollection of having been treated about 15 years ago in another hospital for sinusitis.

Microscopic sections show a carcinoma of a moderate degree of differentiation. In most areas it appears to be squamous, and in fact that was the original diagnosis given by our department. However, it was later realized that this was a muco-epidermoid carcinoma, since multiple areas were found where the neoplasm was glandular in type. This could be demonstrated more satisfactorily with the use of mucicarmine stain, which showed mucicarminophilic material not only in some neoplastic glands, but also intracellularly in tumor cells which at first could be mistaken for squamous tumor cells. As already said, this was a diffusely infiltrating carcinoma which invaded bony and other structures in a fairly extensive way. Histologically we could not demonstrate to our satisfaction any easily recognizable metallic material.

Three months after this operation, the patient was referred to the Department of Radiotherapy for postoperative radiation treatment. He received a total of 4,830 mg. hours with an acrylic mold containing three 37.5 mg. tubes of radium. The depth dose at 1 cm. was calculated to be 5,000 gamma roentgens (R) with maximum surface dose of 11,000. The patient tolerated the procedure well and was discharged to follow-up. In March of 1962 the patient was found to have symptoms of possible recurrence with swelling and redness of the right cheek and the infraorbital area. No biopsy was taken. The patient was referred again for radiation therapy with a plan to deliver 4,000 R tumor dose at a depth of 2 cm. in four weeks. Actually a total dose of 2,500 R was given up to June 1962. There was regression in the size of the swelling, which incidentally was never documented as being a recurrence of the tumor. The patient was seen last in June, 1962, after which he did not keep appointments.

I introduced this case in this seminar because of the apparent definite correlation between the presence of radioactive material in the antrum and the occurrence of a carcinoma. In 1960, a report of three such cases was made from this department. Each one of them had a definite history of thorotrast instillation in the antrum, done for visualization during the study of benign paranasal sinus diseases 10, 15, and 21 years before. Of those tumors, one was an adenocarcinoma, one was a squamous cell carcinoma, and another was a mucoepidermoid carcinoma similar to the present case. We have since seen three other cases including the present one. All of them had the same history of chronic sinusitis, metallic foreign material in the maxillary sinus, and history of introduction of radiopaque material in the investigation of what were thought to be chronic sinusitis or allergic sinusitis.

REFERENCES

1. Kligerman, M., et al.: Carcinoma of Maxillary Sinus Following Thorotrast Instillation, Report of 3 Cases, Cancer 13:967 (Sept.-Oct.) 1960.

See also: References cited by Kligerman, et al.

Clinicopathologic Conference

F. C. Rector, M.D., C. B. Mullins, M.D., M. Ziff, M.D.,
J. D. Wilson, M.D., and R. C. Reynolds, M.D.

An elderly hotel employee had a five-year case history before her death. It started out with pain in her feet when exposed to cold.

Case Presentation

DR. C. B. MULLINS: This 66-year-old white female hotel employee's medical history dates back to July, 1958, when she noticed the occurrence of foot pain when her feet were exposed to cold. In September, 1958, she was abraded and contused in an automobile accident. Following this she developed nausea, vomiting, polyuria, frequency of urination, and polydipsia which lasted 4 to 5 days, and progressed into a comatose state. She was admitted to a local hospital, and was noted to have flaccid paralysis of both extremities, slow respiration, and an irregular slow heart rate. Laboratory findings were reported, a diagnosis of hypokaliemia was made, and a hiatal hernia was found on an upper GI series. She was discharged and referred to Parkland Memorial Hospital where metabolic studies were carried out. These revealed "anemia, cryoglobulinemia, and renal tubular acidosis."

In November, 1958, she was readmitted to Parkland following an acute onset of nausea and vomiting for 24 hours and extreme weakness. She complained of polydipsia and polyuria of one and one-half gallons daily since her last hospitalization. She was again found to have hypokaliemia, lowered plasma CO_2, alkaline urine, and plasma cryoglobulin, which precipitated at room temperature. She was placed on potassium (Shohl's solution). She remained relatively free from weakness from that time, continuing to take Shohl's Solution.

She was again admitted on March 15, 1959, for metabolic studies. The diagnosis of renal tubular acidosis was substantiated by reversing the hypokalemic hyperchloremic acidosis with potassium (Shohl's Solution). Macroglobulinemia and cryoglobulinemia were again found.

In March, 1960, she was admitted again to study the effect of Diamox on renal tubular bicarbonate reabsorption. This study revealed a normal depression of bicarbonate reabsorptive capacity tabular maximum (Tm) with Diamox. Similar results were again obtained in August, 1960.

She was admitted for the last time in May, 1963, with a four-month history of malaise, 13-lb. weight loss, night sweats, anorexia and cough productive of a yellow-white sputum without hemoptysis. These symptoms seemed to have started following a mild exposure to severe cold which precipitated leg and arm pain. She continued to have polyuria and polydipsia, and she had been taking Shohl's solution regularly with no apparent return of weakness.

Physical examination revealed an elderly woman with some evidence of recent weight loss. She complained of being cold and of diffuse pain in her arms and legs. She appeared slightly anemic but not cyanotic. Blood pressure was 110/75, pulse 80, respirations 18, and temperature 100 F. Positive physical findings included a perforated nasal septum; slight dullness to percussion over the right midlung field, but without rales; and a grade 2 apical holosystolic murmur without radiation. The liver was enlarged,

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This is the second in the 1964-1965 series of clinicopathologic conferences to be provided by the University of Texas Southwestern Medical School at Dallas. Dr. C. T. Ashworth, chairman of the Department of Pathology is guest editor.

Case presentation is made by Dr. C. B. Mullins of the Department of Internal Medicine; clinical discussion is by Drs. F. C. Rector, M. Ziff, and J. D. Wilson of the Department of Internal Medicine; and pathological findings are reported by Dr. R. C. Reynolds of the Department of Pathology.

extending below the right costal margin and was nontender. Mild generalized lymphadenopathy was present and brownish pigmentation was noted on both lower legs. Bilateral hallux valgus was present.

A chest x-ray on admission revealed an infiltrate in the superior segment of the right lower lobe, and a suggestion of cavitation. Three sputum smears were positive for acid-fast bacteria. She was then transferred to Woodlawn Hospital for Chest Disease, where she was treated for the remainder of her course. The renal tubular acidosis was well controlled with 25 cc. of potassium (Shohl's solution) three times daily, and never presented a problem in management. She continued a febrile course and produced sputum despite starting isonicotinic acid hydrazide (I.N.H.) and para-aminosalicylic acid (PAS) (Fig. 1). She had some difficulty with PAS due to gastrointestinal intolerance and she continued to lose weight progressively. A urine culture revealed diphtheroids. Tetracycline was instituted and shortly thereafter her fever began to subside, although she never seemed clinically improved. After tetracycline was discontinued, she again became febrile and a urine culture was positive for *Pseudomonas* which were sensitive to colistin and chloramphenicol. At this time the urine contained numerous white blood cells. Colistin and chloramphenicol were then started. Her chest x-rays revealed progression of the pulmonary disease, and sputum production and clinical findings never improved. She suddenly and unexpectedly died on the 69th hospital day.

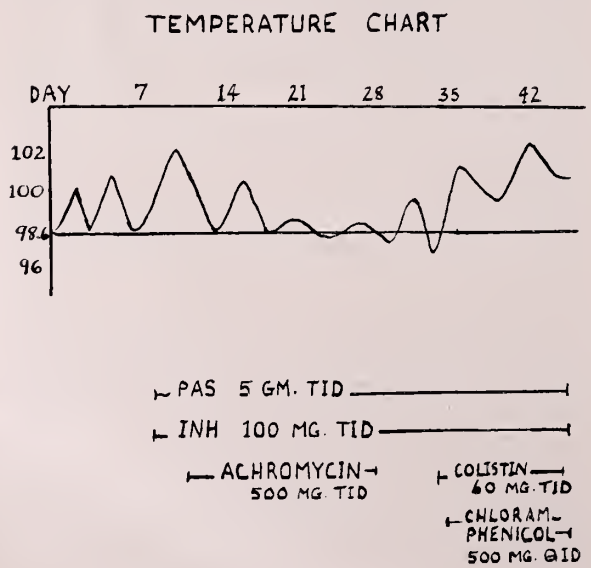

Fig. 1.

The major laboratory findings in this case are shown in Table 1.

Clinical Discussion

DR. F. C. RECTOR: This patient is a unique case. First, I would like to clear up one or two points concerning the history. Although initially the possibility was introduced that the patient's disease was related to the automobile accident, careful questioning established that manifestations of disease were present about three or four weeks before the accident. At that time, she had a petechial rash over the lower extremities followed by increasingly brown pigmentation of the skin; and second, there was the onset of nocturia at the same time.

This woman was admitted with a flaccid paralysis which was proven to be due to hypokaliemia. When she was first seen at another hospital, the presence of hypernatremia obscured the fact that she had hyperchloremic acidosis, and it was believed that the existing hyperchloremia was simply the result of dehydration. When she was admitted to Parkland Hospital about a week later, again she did not have obvious hyperchloremia. She did, however, have acidosis on Nov. 11 as manifested by reduction in the CO_2 content associated with a blood pH of 7.27. The chloride of 108 mEq. per liter does not indicate a severe hyperchloremia, but considering the fact that the serum sodium was only 130 mEq. per liter, this chloride of 108 mEq. per liter becomes significant.

The differential diagnosis of hyperchloremic acidosis with severe hypokaliemia was, then, the original problem. Subsequent studies established without doubt she had renal tubular acidosis. In the differential diagnosis of hyperchloremic acidosis manifested by low CO_2 content and elevated serum chloride it is necessary first to ascertain whether acidosis does in fact exist or whether these laboratory findings are due to respiratory alkalosis. This requires determination of arterial pH. Once acidosis is established, several possibilities must be considered. Although it is usually stated that acidosis due to chronic renal insufficiency is not associated with hyperchloremia, our experience here indicates that this is not the case. In reviewing 56 patients that Dr. Norman Carter has dialyzed for chronic renal

TABLE 1.—*Major Laboratory Findings.*

	9/1/58	11/4	11/11	3/16/59	3/14/60	3/5/62	5/17/63	6/7/63	6/17/63
Hgb. (gm./100 cc.)		7.4	9.1	7.5	10.2	8.3	7.7		
Hct. (%)	41		26.8	24.5	31.7	20	29	28	27
Reticulocytes (%)			2.8						
WBC (1000s/cu. mm.)		7.4		4.1	10.2	8.5	9.6	9.8	6.9
Segs/bands (%)		74/3		68/4	84	76	79	94	84
Lymphocytes (%)		20		27	13	14	18	5	14
Eosinophils (%)		3		1	2	2	1	1	0
Urine:									
pH		7.5	6.0	7.5	7.5	7.5	7.5	7.5	7.5
sp. gr.	1.004	1.002	1.010	1.010		1.010	1.010	1.010	1.012
albumin	1+	N		N	N	N	N	5 mg.	5 mg.
sugar		N		N	N	N	N	N	N
WBC		10-25		Rare	N	N	N	8-10	200
Bence-Jones				N	N				
BUN (mg./100 cc.)	8	18		16	19	20	17	14	19
Creatinine (mg./100 cc.)		3.5					1.3	1.2	
Glucose (mg./100 cc.)	235	102							
CO_2 (mEq./liter)	20	19.3	10.4 (arterial)	25	24	15	26	27	27
Cl (mEq./liter)	118	102	108		102		99	92	99
Na (mEq./liter)	159	140	130		135	135	138	132	138
K (mEq./liter)	2.5	2.7	4.0		4.9	3.3	4.2	3.6	4.0
Proteins:									
Albumin (gm./100 cc.)	3.8	4.2		2.9	4.1		3.1	3.0	
Globulin (gm./100 cc.)	3.3	3.0		4.0	4.2		3.8	3.4	
Ca (mg./100 cc.)		9.5		9.6	10.8		9.4		
P (mg./100 cc.)		3.3		3.8	4.4		3.3	4.3	
Blood pH			7.27						
Cholesterol (mg./100 cc.)		258							
Bilirubin									
1 min. (mg./100 cc.)		0.6							
Total (mg./100 cc.)		2	0.5		0.3		0.3	0.1	
Cephalin flocculation		2+	4+		4+		4+	3+	
Thymol turbidity		26.7					25.5	23.3	
Alkaline phosphatase (King-Armstrong Units)		4.1	2.1	2			2.3	2.1	
Total serum iron (μg./100 cc.)			18				18		
Total iron in binding capacity (μg./100 cc.)			160				200		
Cryoglobulins			+	3+				+	
Serology		1+		Neg.					
Amylase		320							
LE			Neg.						
Macroglobulins				+					
Latex fixation							+		

Additional Laboratory Studies:

Cultures: Urine: 11/15/58, *Aerobacter* x2; 6/8/63; *Pseudomonas*, sensitive to colistin and chloramphenicol. Sputum: 5/15/63, typical tuberculosis; 5/27/63, typical tuberculosis; 5/29/63, typical tuberculosis; 5/20/63, routine, mixed flora; 5/23/63, routine, *Klebsiella* and *Pseudomonas*.

Protein Electrophoresis: 12/5/58, before and after precipitated cryoglobulins, cryoglobulins in gamma, beta and alpha 2 with majority in beta. 3/15/59, Broad-based gamma globulin spike, ultracentrifuge, small amounts in 19S and 22S macroglobulins.

Complement Fixation: 5/20/63, histoplasmosis, coccidioidomycosis, and yeast, negative.

Skin Tests: 5/20/63, photochromogens, 2 cm.; scotochromogens; 1.5 cm.; PPD #1, 2 cm.

insufficiency during the past two or three years, 31 of these have at one time or another had definite hyperchloremic acidosis. Whether hyperchloremic acidosis is due to chronic renal insufficiency or represents true renal tubular acidosis may be a very

difficult question to answer. The distinction can best be made on the basis of urine pH, or the response of urine pH to an acid load. The patient with renal tubular acidosis cannot acidify the urine maximally. In most instances, the urine pH will not fall below a pH of 6 despite the presence of severe acidosis. In addition, hypokaliemia usually coexists with acidosis. In contrast to this pattern in renal tubular acidosis, the patient with renal insufficiency can acidify the urine quite significantly. Usually at a time when acidosis is present, the urine pH would be less than 5.5. In addition, serum potassium is almost invariably elevated in renal insufficiency, and hypokaliemia in chronic renal insufficiency with hyperchloremic acidosis is very unusual unless the patient has had severe vomiting.

We occasionally see hyperchloremic acidosis in patients with phenacetin nephritis. Characteristic of this disorder is the fact that acidosis is disproportionately greater than the degree of renal insufficiency. Ingestion of other drugs also must be considered as a cause of hyperchloremic acidosis. Occasionally ammonium chloride produces it, and it may also occur during acetazolamide therapy for glaucoma. True renal tubular acidosis has been reported in sulfonamide intoxication and after the administration of paraldehyde. In addition to these causes, hyperchloremic acidosis may result from the loss of alkaline gastrointestinal contents through draining fistulae or severe acute diarrhea, and in ureterosigmoidostomy.

The criteria for establishing the diagnosis of renal tubular acidosis in a given patient depends on demonstrating the presence of hypokaliemia, hyperchloremia, acidosis, and inability to acidify the urine below pH 6. It helps if nephrocalcinosis is demonstrable by radiologic examination.

Patients with renal tubular acidosis develop several complications. A very common complication is muscular paralysis which may closely resemble either poliomyelitis or Guillain-Barré syndrome. Patients with renal tubular acidosis seem to have a higher incidence of severe muscle weakness and paralysis at a given serum potassium than other patients. The three patients that we have seen have been paralyzed with serum potassiums in a range of 1.5 to 2 mEq. per liter. In part, this may be due to the fact that most patients with hypokaliemia have an associated metabolic alkalosis, whereas in renal tubular acidosis there is a metabolic acidosis.

Another complication that is frequently seen in patients with renal tubular acidosis is nephrocalcinosis. It has been present in approximately 75 per cent of the cases reported in the literature. Our patient did not have radiologic evidence of nephrocalcinosis and the question arises as to why not. The exact cause of nephrocalcinosis in these patients is not known. They all have hypercalcinuria; calcium excretion is increased by metabolic acidosis and is decreased by metabolic alkalosis. A second factor that may play an important role in the development of nephrocalcinosis in renal tubular acidosis is decreased citrate excretion. In alkaline urine, citrate helps maintain urine calcium in a soluble state. If a normal subject is given $NaHCO_3$ to alkalinize the urine, citrate excretion increases; if a patient is acidified, citrate excretion decreases. Acetazolamide, on the other hand, blocks carbonic anhydrase and the urine becomes alkaline, but citrate excretion falls practically to zero. This has been suggested by Harrison and Harrison[3] as the cause of the nephrocalcinosis in acetazolamide therapy. In patients with renal tubular acidosis, citrate excretion is very low despite the fact that the urine is relatively alkaline. Therefore, calcium would tend to precipitate and produce nephrocalcinosis.

Approximately 80 per cent of patients with nephrocalcinosis due to renal tubular acidosis eventually develop superimposed pyelonephritis. It has been suggested that in adult patients renal tubular acidosis develops because of chronic pyelonephritis, but the evidence indicates that renal tubular acidosis occurs first and only later does pyelonephritis develop.

This patient was studied extensively on the metabolic ward in an attempt to identify some of the factors that might impair acid excretion in renal tubular acidosis. First, there might be a defect in the reabsorption of filtered bicarbonate, so that bicarbonate is continually spilled into the urine, resulting in a relatively alkaline urine and an acidosis. This might also stimulate potassium secretion. Several observers have suggested that such a defect in the reabsorption of bicarbonate might be due to carbonic anhydrase deficiency. Second, there might be a reduction of the capacity of renal tubules to produce

titratable acid. Third, there might be a defect in ammonia production. Finally, the distal portion of the nephron might not be able to establish maximum pH gradients between blood and urine; such a defect would secondarily impair titratable acid and ammonia excretion.

The results of our studies in this patient can be summarized as follows: First, the capacity of the renal tubule to reabsorb filtered bicarbonate was perfectly normal. In addition, her response to acetazolamide was also normal, indicating that there was no deficit in renal carbonic anhydrase. Second, although titratable acid excretion was below normal it could be greatly increased by the administration of buffer salt, sodium phosphate. Third, ammonia excretion in this patient was also low, but when compared with that in normal subjects with comparable urine pH values (6.5-7.0) it was greater than normal. This would indicate that the relatively alkaline urine, rather than any enzymatic defect in ammonia production, was responsible for the limitation in ammonia excretion. Finally, it was found that the patient could not maximally acidify her urine. At a time when she was severely acidotic with a CO_2 content of 12 mEq. per liter, the lowest urine pH was 6.4.

The fact that a patient with renal tubular acidosis cannot lower urine pH below 6.0 to 6.5 means that there will always be some bicarbonate in the urine. This might be due either to a slight leak of bicarbonate out of the proximal tubule, flooding the distal tubule with alkaline fluid, or to a specific defect in acidification in the distal tubule. These two possibilities can be distinguished by examining the effects of water diuresis on bicarbonate excretion. If the defect is due to inability of the distal tubule to lower urine pH below some fixed minimal value, then urine pH will remain constant as urine flow is increased during water diuresis. Since the urine will be in equilibrium with the CO_2 tension of plasma, the concentration of bicarbonate in the urine will remain constant, and consequently bicarbonate excretion will increase as urine flow is accelerated. In contrast, if there is spillage of a fixed amount of bicarbonate out of the proximal tubule, bicarbonate excretion will not change as urine flow is increased. Several studies in this patient reveal that as urine flow was progressively increased from 1 to 12 ml. per min. the urine pH and urine bicarbonate concentration remained constant and bicarbonate increased proportionately. This indicates that the defect is not due to spillage of bicarbonate out of the proximal tubule but rather is due to a specific defect in the distal tubule impairing its ability to lower urine pH below a fixed minimal value.

The pathogenesis of renal tubular acidosis is summarized in Fig. 2. The primary defect is an inability of the tubule to elaborate a maximally acid urine; consequently there is decreased excretion of ammonia and titratable acid. Systemic acidosis thereby ensues. Since the defect in acidification impairs the NA^+–H^+ exchange process when the limiting pH is approached sodium will be lost into the urine along with the nonreabsorbable acid anions. This slight sodium wastage will tend to contract the extracellular volume, which in turn will stimulate the reabsorption of sodium chloride. The reexpansion of the extracellular fluid volume with sodium chloride will result in hyperchloremia. In addition, the tendency to waste sodium will also stimulate potassium secretion resulting in severe hypokaliemia.

The etiology of the renal tubular acidosis is not completely clear. Many of the cases have been reported as familial. Perhaps Dr. Wilson who has recently reviewed the literature on the genetics of renal tubular acidosis will comment about the genetics of this disorder. In some instances the disorder has resulted from drug intoxication, including sulfonamides and paraldehyde. Chronic pyelonephritis, hyperthyroidism, and hyperparathyroidism have also been implicated in isolated cases, but the evidence for a causal role is not convincing.

This patient presents the very interesting combination of renal tubular acidosis and cryoglobulinemia. The possibility that the dysproteinenia might in some way have caused the renal tubular acidosis makes interesting speculation. Cases of Fanconi's syndrome associated with multiple myeloma have been reported by Engle and Wallis.[2] Our patient was carefully checked for proximal renal tubular defects such as occur in Fanconi's syndrome. She did not have hypophosphatemia, glycosuria, or aminoaciduria. Thus, this patient did not have Fanconi's syndrome, but rather pure renal tubular acidosis. Sanchez and Domz[10] reported a man with multiple myeloma who had hyperchloremic acidosis. The urine pH's were not reported and I suspect this was simply a case of chronic renal insufficiency with

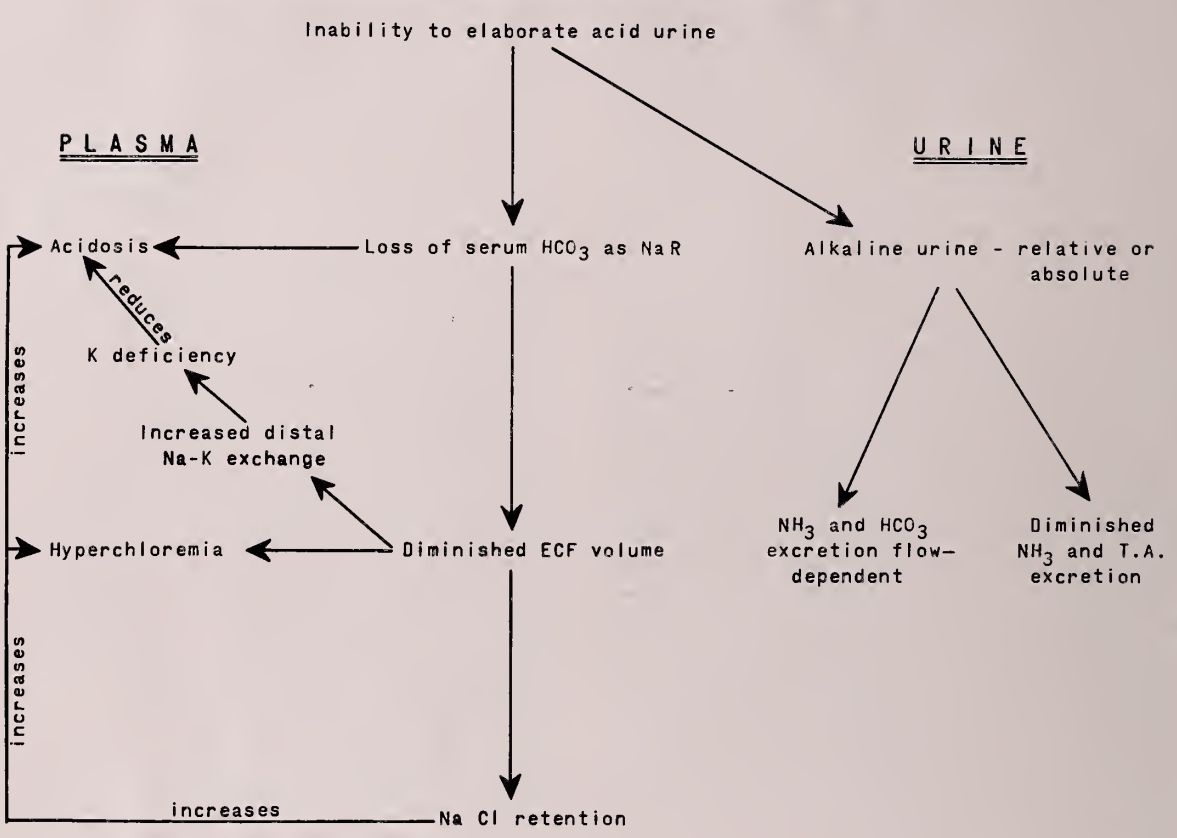

Fig. 2.

hyperchloremic acidosis and not renal tubular acidosis.

In 1962 Cohen and Way[1] in Australia reported three cases of hyperglobulinemia associated with renal tubular acidosis. They were all in young women, and resembled the present case in many respects: hypokaliemic paralysis, hyperchloremic acidosis, petechial rash and skin pigmentation, and hyperglobulinemia. In another patient followed here at Parkland Memorial Hospital we have seen renal tubular acidosis associated with hyperglobulinemia. This woman has been known to have nephrocalcinosis since 1945. In 1957 she was found to have renal tubular acidosis with slightly elevated serum proteins. Recently, her total protein was 9.5 gm. per cent globulin, mainly gamma globulin. In one of the three cases in Australia and in one of the two cases here at Parkland, renal tubular acidosis clearly antedated known dysproteinemia. In one of the Australian cases and in the case under discussion today these two features appeared to develop simultaneously. The relationship between renal tubular acidosis and dysproteinemia is not known. Whether these are coincidental genetic defects appearing at different times, or whether dysproteinemia in some of these patients may actually produce a tubular defect, perhaps a reaction between the abnormal protein and kidney tissue, I don't know. Perhaps Dr. Ziff can comment on this possibility.

DR. MORRIS ZIFF: This patient was first referred to us in the rheumatology clinic in 1958 because she had a positive latex fixation test. It turned out that her pain was in the arm and leg, and was not joint pain. However, she had rather interesting gamma globulin abnormalities. Our records indicate she had weakly positive Kline and Wasserman reactions and a negative *Treponema* immobilization test. The total globulin was 6.0 gm., and thymol turbidity, as you know, was 27. Cryoglobulins were present; the gamma globulin was 3.7 gm. per cent, and as I looked at the patterns just now, the forward edge of the gamma globulin appeared sharp, as though representing a discrete component, while the slow portion was broad based as though repre-

senting a diffuse component. Plasmacytosis of 5 per cent was found in the marrow. The latex fixation titer was 1:2560 in our laboratory, and by the tanned cell hemagglutination tests, in which one coats human gamma globulin onto tannic acid-treated red cells, the titer was 1:56,000. She reacted well to aggregated Cohn FII. However, the reaction to sensitized sheep cells was negative, as is to be expected when one sees positive latex tests in the absence of rheumatoid arthritis. This happens in liver disease, and other hyperglobulinemic states. When Dr. LoSpalluto[5] ultracentrifuged the serum, he noted a 22S component, as occurs in rheumatoid arthritis, liver disease, or sarcoidosis —that is, where there are rheumatoid or rheumatoid-like factors in the serum.

Cryoglobulinemia was also present in this patient. Most cryoglobulins are 7S gamma globulin, that is, ordinary lightweight gamma globulins. Occasionally they are of the 19S or heavy variety, but this is much less common. Rarely one may see a mixture of 19S and 7S cryoglobulins. In this patient the amount of cryoglobulin, measured as 1 gm. per cent, was within the symptomatic range. About 300 mg. per cent is clinically significant. There were certain unusual features about the cryoglobulin. On ultracentrifugation, the serum showed the 22S component mentioned previously, but when it was separated in the cold, redissolved in warm saline, and ultracentrifuged, the 22S component had dissociated into 7S and 19S proteins. Thus, the 22S component was stable in the serum, which was much like the 22S form of the rheumatoid factor as found in rheumatoid serum, but when it was redissolved in saline and ultracentrifuged, this complex dissociated into 7S and 19S components in approximately equal amounts. Since the patient had 1 gm. per cent of cryoglobulin, of which half was 19S, it appears that 500 mg. per cent or about 5 per cent of her total protein was in the form of 19S macroglobulin. One could not say on this basis that she has a macroglobulinemia in the usual sense of the term. The macroglobulin was peculiar in that it was able to react with 7S gamma globulin and form a material which precipitated in the cold, yielding a good deal of cryoglobulin, unusual as it was.

I might summarize what I have been saying in the following way: Her 22S component was like other 22S rheumatoid factor-like components, as seen in patients with collagen disease, liver disease, and other hyperglobulinemic states, in that it reacted with Cohn Fraction II to give a precipitate and with latex particles to agglutinate them. It was soluble and nonprecipitating at normal temperatures as in sera of rheumatoid arthritis or liver disease, but it had the peculiar property of being insoluble in the cold, and in this respect, it was unique.

The cryoglobulin of this patient was described during life in a report in the literature.[5]

DR. JEAN WILSON: That many cases of renal tubular acidosis are due to a genetic defect was not recognized until many years after the entity was first described. In a review in 1957 by Piel,[7] 56 of the 60 cases were said to have a negative family history. Since that time, however, a familial incidence of this disease has been described with greater frequency, and 13 different families involving the 36 affected individuals have now been reported to have some combination of the triad of persistent hyperchloremic acidosis, nephrocalcinosis, and nephrolithiasis. The best studied of these families is the one that has been reported first by Pitts and others,[8] and subsequently by Randall and Targgart.[9] In this family, renal tubular acidosis has been transmitted as a dominant trait over three successive generations and has involved six individuals. Schreiner and others[11] have reported another family involving three generations. There have been six families with involvement of two generations and five families in which 12 out of 20 individuals of one generation only were affected. Consequently, it seems safe to conclude that renal tubular acidosis does occur as a simple Mendelian dominant with variable expressivity. This formulation does not, however, answer all the questions, for even though it has been established that renal tubular acidosis may be secondary to a genetic defect, a large number of cases appear to occur sporadically; this is true even when the known causes of secondary renal tubular acidosis have been excluded. Such sporadic cases do not necessarily disprove the genetic nature of the disorder since in a disease which affects both survival rate and reproductivity, the deletion rate of the abnormality must be very high. It is consequently very likely that a large percentage of the sporadic cases are due to new mutation and that the mutation rate equals the deletion

rate. This would make the situation in renal tubular acidosis similar to that in polycystic disease of the kidneys, which is a common enough defect that the mutation rate can be calculated with some degree of accuracy; in this condition 40-50 per cent of the cases seen at random are due to new mutations. Thus, at the present time it does not seem too far-fetched to say that a genetic defect may play a role in most cases of renal tubular acidosis. The degree to which a genetic defect may be involved in the secondary cases is unknown.

Finally, in addition to the types of adult renal tubular acidosis, there is a very interesting transient form of the disease which occurs in infancy. There are two theories as to its etiology. One possibility is that it represents a very mild form of adult renal tubular acidosis in which the individuals recover from the basic defect. Huth and others[4] have argued, however, that this is actually a different disease; the rationale for this thesis is that no child with this form of the disease has ever been reported with a positive family history (with one possible exception), and they suggest that the transient renal tubular acidosis of infants is either a nongenetic developmental defect or a recessive abnormality and not related to the adult form of the disease.

Pathologic Findings

DR. R. C. REYNOLDS: Autopsy revealed extensive deposits of amyloid in the pulmonary blood vessels (Fig. 3) and occasional focal deposits of amyloid in the kidneys, heart, and thyroid. The subendothelial spaces were filled with an eosinophilic, granular, and waxy material which frequently replaced the entire medial wall of the blood vessel. The medium-sized and small blood vessels were predominantly involved, but deposits were also seen in the adventitial tissue of the blood vessels and in alveolar walls. There was a foreign-body giant cell reaction to the amyloid material where it had extended into the adventitia of blood vessels and supporting connective tissue of the lung (Fig. 4). No foreign-body giant cell reaction was seen in the kidneys, heart, or thyroid.

Sections of lung, adrenal and kidney were stained with Congo red, methyl violet, and PAS. The amyloid deposits were PAS positive and showed the typical violet color produced by methyl violet in amyloid. The uptake of Congo red was variable. Amyloid associated with dysproteinemia frequently demonstrates such a variability in its reaction to the amyloid stains.

The pattern of amyloid deposition (mesenchymal and vascular tissues) was that of primary amyloidosis and its distribution was similar to the amyloidosis of multiple myeloma and other dysproteinemias. Secondary amyloidosis, on the other hand, is generally deposited in organs rich in reticuloendothelial tissue such as the spleen, liver, and lymph nodes.

A second vascular lesion was found extensively in the lungs and to a lesser extent in the kidneys (Fig. 5). This lesion consisted of multiple hyaline thrombi in the small arteries and arterioles. In many pul-

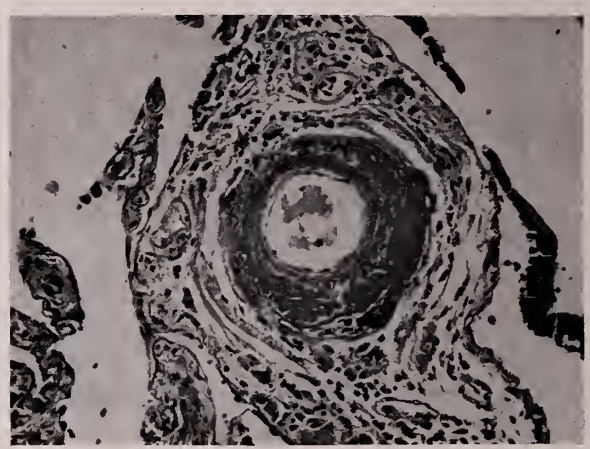

Fig. 3. Low power photomicrograph of lung, showing amyloidosis of pulmonary artery. The deposits in the wall stained positively with Congo red, and metachromatically with methyl-violet.

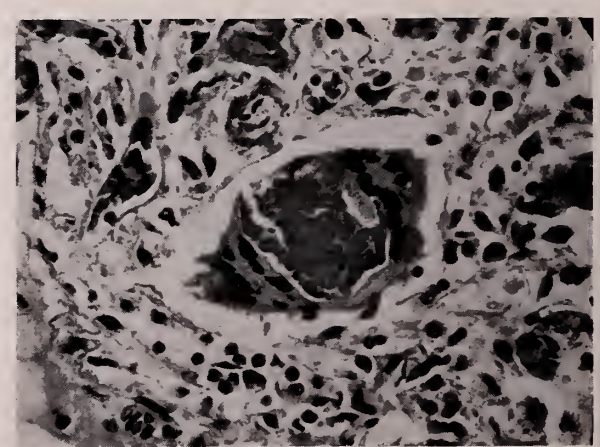

Fig. 4. High power photomicrograph of interstitial tissue of the lung containing a heavy deposit of amyloid. Foreign body giant cells are noted around the deposit.

monary blood vessels, and in a rare renal blood vessel, the hyaline thrombi had become organized. Recanalization of the organized hyaline thrombi was frequently seen in the lungs.

The kidneys weighed approximately 120 gm. each, and the renal surfaces were finely granular. On microscopic examination, the kidneys contained multiple small, wedge-shaped foci of cortical atrophy and fibrosis (Fig. 6), in addition to the occasional amyloid deposits and hyaline thrombi noted previously. These areas of atrophy were most prominent beneath the capsule, and their distribution was similar to that occurring in nephrosclerosis. In these areas there was moderately severe glomerulosclerosis (Fig. 7) and varying degrees of tubular atrophy. A prominent infiltrate of lymphocytes was present in the areas of atrophy. In the deeper portions of the cortex, similar foci of moderate tubular atrophy associated with dense infiltrates of lymphocytes were noted. Hyaline casts were frequently found in the tubular system (Fig. 8), but they were not surrounded by foreign body giant cell reaction as is commonly seen in multiple myeloma kidney.

The relationship between this patient's renal pathology and the two outstanding clinical disease processes, renal tubular acidosis and cryoglobulinemia, is not clear. In the adult type of renal tubular acidosis the commonest associated clinical condition is pyelonephritis. In ten cases of renal tubular acidosis reported by Owen and Verner,[6] eight were found to have clinical pyelonephritis or there was a history of pyelonephritis; but the histopathology of the renal lesions was not studied.

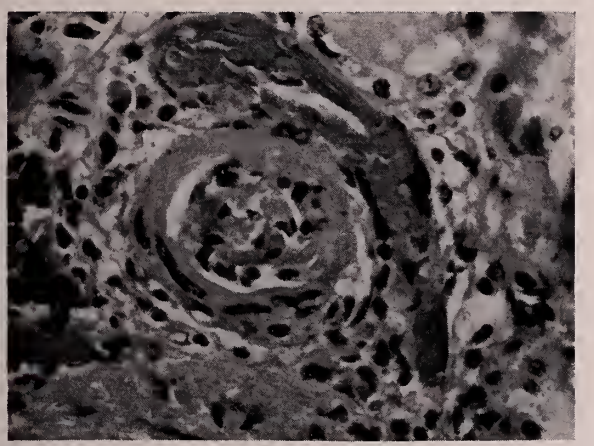

Fig. 5. High power photomicrograph of small renal artery showing thrombosis with early organization.

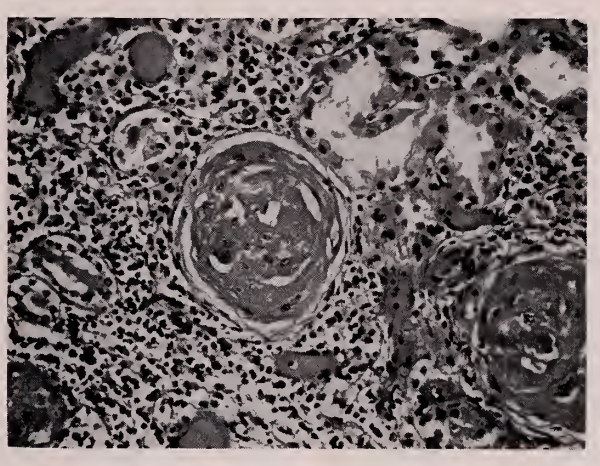

Fig. 7. High power photomicrograph of kidney showing hyalinization and obliteration of glomeruli in areas of ischemic atrophy.

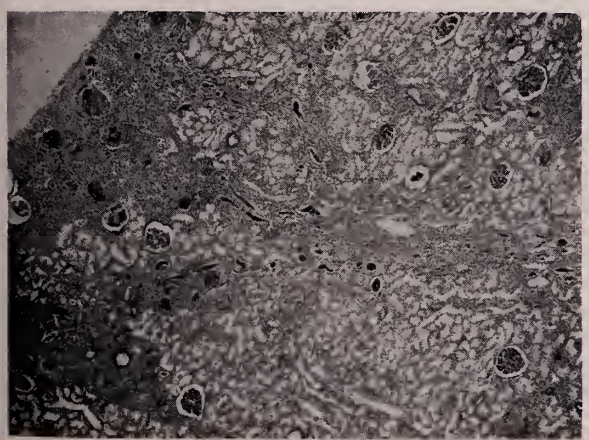

Fig. 6. Low power photomicrograph of kidney cortex. The areas of ischemic atrophy are shown.

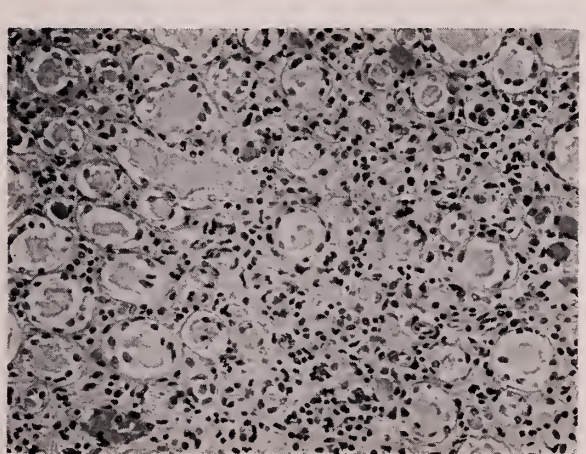

Fig. 8. High power photomicrograph of corticomedullary zone of kidney. The tubules contain numerous casts, but no foreign body reaction around these has occurred as is usual in myeloma kidney.

Cohen and Way[1] reported three patients having renal tubular acidosis. Renal biopsies were performed on two of these patients. On one patient the biopsy was reported as normal; the other patient showed vacuolated tubular epithelium, some infiltration of lymphocytes in the interstitial tissue, and an occasional hyalinized glomerulus. These findings, with the exception of the vacuolated tubular epithelium, partially resemble those found in the kidneys of the patient presently under consideration.

The histopathologic alterations in our patient's kidneys are essentially those of ischemic atrophy with an unusually prominent lymphocytic infiltrate. These changes could result from arteriolar nephrosclerosis; from vascular occlusion by hyaline thrombi or amyloid secondary to cryoglobulinemia. A 73-year-old woman might be expected to have a considerable amount of arteriolar nephrosclerosis independently. Furthermore, severe infections, such as this woman had, frequently result in dense foci of lymphocytic infiltration in the kidneys, that is, an interstitial nephritis. Another possibility is that the patient's dysproteinemia and associated amyloid disease were responsible for the areas of renal cortical atrophy found in the kidneys. A rare blood vessel in the kidneys demonstrated amyloid deposits and a few hyaline thrombi were found in renal blood vessels. Occlusion of blood vessels due to such a process could lead to the same general type of morphologic alteration as is seen in nephrosclerosis. Moderate hyaline arteriolosclerosis was present in the kidneys. It appeared likely that the focal ischemia resulted from arteriolosclerosis, or cryoglobulinemia, or both. The dense infiltrations of lymphocytes could be a local reaction to ischemic atrophy or they might represent an interstitial nephritis associated with severe infection. While the renal lesion that accounts for renal tubular acidosis has not been defined histologically, the atrophic and retrogressive changes present in the numerous patchy areas of cortical and corticomedullary fibrotic atrophy might very well be a cause of this defect.

Grossly, the apex of the lower lobe of the right lung and the adjacent segment of the right upper lobe revealed extensive caseous necrosis and several areas of liquefactive necrosis suggesting abscesses, rather than tuberculous cavities. Microscopically, two distinct lesions were found in these tissues. The first consisted of suppurative inflammation with many polymorphonuclear leukocytes and colonies of gram-positive cocci in clusters. Coagulase positive staphylococci were grown from the lung and from postmortem heart and blood cultures.

The second lesion found microscopically was the caseous necrosis which had been noted grossly. There was a relatively sparse epithelioid and giant-cell inflammatory response around the margins of caseous material. Acid-fast stains revealed innumerable acid-fast bacilli. Typical *Mycobacterium tuberculosis* was cultured from the lungs. The disease appeared to result from an endobronchial spread of tubercle bacilli from a caseous hilar lymph node. It is considered most likely that this pulmonary tuberculosis is a reactivation or reinfection which developed during the course of (and may have been predisposed to by) existing dysproteinemia and other metabolic disorders.

The major pathological findings were (1) nephropathy associated with dysproteinemia (cryoglobulinemia) and renal tubular acidosis, (2) amyloidosis (dysproteinemic type), with involvement of pulmonary arteries, heart, kidneys, and thyroid, and (3) nodular caseous tuberculosis and pyogenic (staphylococcal) abscesses of upper lobe of right lung.

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◗ Drs. Mullins, Rector, Ziff, Wilson, and Reynolds, University of Texas Southwestern Medical School, 5323 Harry Hines Blvd., Dallas.

NEWS

AMA Expects 16,000 Physicians At San Francisco Convention

AN ATTENDANCE of between 15,000 and 16,000 physicians is anticipated for the American Medical Association's 113th annual convention in San Francisco June 21-25. When the AMA held its last convention in San Francisco in June, 1958, the total physician registration was 13,997.

The entire Texas delegation will be there and will present three resolutions from the Texas Medical Association. These regard recognition of cigarette smoking as a health hazard, opposition to the nationalization of medicine, and opposition to compulsory area-wide planning for health facilities.

Dr. J. Arnold Bargen, Temple, chairman of the AMA Council on Postgraduate Programs which plans the scientific programs for the two AMA conventions, the annual and clinical, said that the San Francisco program will be most comprehensive, including lectures, scientific exhibits, preview showings of medical films, and color television.

Dr. John Hickam, Indianapolis, chairman of the program planning committee of the Council, said that the following general scientific meetings have been coordinated by section secretaries: differential diagnosis of the liver and pancreas; hyperbaric oxygen phenomena; computers in medicine; autoimmune mechanisms and disease, cardiovascular opacification, and tumors of the endocrine function.

In addition, a special half-day program on various aspects of heart disease will be offered by the American College of Cardiology and the American Heart Association.

The popular and interesting research forum program, under the chairmanship of Dr. Edwin H. Ellison, Milwaukee, will be offered again at the San Francisco meeting. Sixty papers, based on new and original work being done in the nation's medical schools, will be delivered by young, outstanding researchers. In contrast to previous forums, one or two major areas of research, possibly organ transplantation and hyperbaric oxygen in the treatment of disease, will be covered in a symposium presentation.

A general scientific meeting program that has already elicited considerable interest is the half-day session on hyperbaric oxygen phenomena—the science of administering oxygen at super atmospheric pressure.

San Francisco's Civic Auditorium has been completely modernized at a cost of more than $7,000,000 and will be ready for the AMA convention. Scientific exhibits will be set up there.

San Francisco, in keeping with its growing attractiveness as a vacation and convention center, has increased its housing capacity since the AMA last met in the Pacific coast city six years ago. Several major new hotels have been constructed, existing ones have greatly expanded their accommodations, and hundreds of fine motels now encircle the entire bay area. Complete forms for hotel reservations, as well as for advance convention registration, appear periodically in all AMA publications. The entire scientific program and all essential information for attendance was published in the Convention Issue of *The Journal of the American Medical Association* on May 9.

Three presidents together are Dr. Max Johnson, left, new TMA President, Dr. Edward R. Annis, center, AMA President, and Dr. Robert Mayo Tenery, immediate Past President of TMA.

Members of the Reference Committee on Miscellaneous Business hear debate.

Selected for Distinguished Service Awards by House of Delegates are Dr. Milford O. Rouse, left, of Dallas and Dr. George R. Herrmann of Galveston.

Annual

WITHOUT DEBATE on the floor, the House of Delegates of Texas Medical Association approved properly controlled fluoridation as a safe public health measure. The action followed a unanimous recommendation from the Reference Committee on Legislation and Public Relations and an appeal from President Dr. Robert Mayo Tenery for delegates to take a stand either for or against fluoridation as a safe measure.

In other action, the House turned down the recommendation of the Council on Constitution and By-Laws and the Reference Committee on Constitution and By-Laws which called for a nominations board to be established to nominate one or more candidates for the elective offices of president-elect, vice-president, secretary, treasurer, speaker of the House, vice-speaker of the House, trustees, delegates, and alternate delegates to the American Medical Association. Instead, in an unusual move, the House accepted the minority report of the Reference Committee and voted against such a nominations board.

In addition to fluoridation, delegates approved a number of public health measures including a recommendation of the Reference Committee on Scientific Work urging that everyone should be properly immunized, with emphasis being placed on the tetanus immunization program. The Committees on Disaster Medical Care, Industrial Health, and Public Health had urged such approval.

Delegates also endorsed the use of seat belts in automobiles, commended agencies which are encouraging such use, and recommended compulsory legislation in Texas

Dr. James D. Murphy, speaker of the House of Delegates, Dr. Charles Max Cole, vice speaker, and Mrs. Hamilton F. Ford, outgoing president of the Woman's Auxiliary, await opening of the House of Delegates.

"We can produce new elements through nuclear explosions that can be used for treating disease," Edward Teller, Ph.D., seated center, told physicians at a lecture-luncheon on the Session's last day. Sponsors of the affair were members of the Committee on Nuclear Medicine including, standing, Dr. Elbert DeCoursey, Dr. Ernest E. Anthony, Dr. J. Allen Chamberlin; seated left, Dr. Herbert C. Allen, Jr., and right, Dr. J. R. Maxfield, Jr.

Session Wrap-up

requiring the installation of seat belts in all new automobiles.

Delegates also approved several recommendations from committees and councils, including a resolution from the Committee on Cancer, which pointed to the dangers of smoking, and agreed to a proposal that the Council on Scientific Advancement explore various means of helping people who wish to quit smoking.

Continued use of tests to determine the presence of PKU and the administration of Sabin and Salk vaccines were also recommended. Physicians were called upon to cooperate fully in reporting venereal diseases to public health officials. City ordinances were recommended for inspection of poultry and rabbits used in food stores or where meals are sold.

Approved also were plans for introducing in the 1965 Legislature a child abuse law which would make it mandatory for physicians and hospitals to report any cases of child abuse to the appropriate police authority. The bill would include immunity from liability in such reporting.

Delegates also approved plans to introduce a thallium sulfate law which would make it unlawful for compounds containing thallium to be sold. This is designed to prevent deaths in children.

Another measure which kept the public in mind was the approval of the recommendation by the Reference Committee on Medical Service and Insurance for passage of the resolution from Harris County calling for physicians to continue to refer specimens for laboratory analyses to laboratories supervised by fully qualified and licensed doctors

of medicine.

Also approved was a statement by the Reference Committee on Legislation and Public Relations which declared that if physicians did not support lay-owned laboratories, the problems associated with such laboratories would disappear.

Of further importance to public health was the approval of a recommendation to the State Department of Health asking that when it receives a death certificate of a woman between 10 and 50 years of age, it send to the physician signing this certificate a form requesting information concerning whether the woman was pregnant or had been pregnant within the last 90 days before her death. This recommendation was designed to aid in the compilation of statistics on maternal mortality. Delegates also agreed with the stand of the Council on Scientific Advancement and the Committee on Nutrition against subscribing to ideas and concepts of nutrition which are not based upon sound scientific principles.

Dues Increase Voted

Delegates voted to accept the recommendation of the Board of Trustees, a resolution from Bexar County, and the recommendation of the Reference Committee on Constitution and By-Laws calling for a dues increase which authorized the raising of dues from the present $45 up to a maximum of $55.

The Board of Trustees previously had reported that its operating funds were fully committed and that it would not be possible to underwrite new programs in the future. The Board offered the alternatives of curtailing some existing programs, invading Association reserves, or securing additional revenues through a modest increase in dues.

Two standing committees were approved by delegates, including the Committees on Medicine and Religion and a committee on nursing which would have responsibility for liaison with the Texas Graduate Nurses Association and other recognized nursing groups.

Also approved was a clarification of the Constitution and By-Laws on the authority and responsibility of the Board of Councilors in the administration of certain aspects of multi-county medical societies.

In other action, delegates approved the intent of a resolution from Navarro County Medical Society which called for the formation of a policy committee to accumulate, document, and disseminate the policies of Texas Medical Association to all levels. Delegates approved the recommendation of the Reference Committee on Legislation and Public Relations that the Council on Public Relations and Public Service undertake this function instead of a specially formed committee.

Resolutions from the Committee on Nuclear Medicine commending Attorney General Waggoner Carr, the Texas State Department of Health, and the American Medical Association for their support in opposition to U.S. Department of Labor-proposed changes in the Walsh-Healey Act (1936) were approved. The resolution opposing such changes was passed as part of the report of the Committee on Nuclear Medicine. The resolution pointed out that the proposed regulations were in conflict with the state-federal agreement on licensing regulations.

Delegates passed a substitute resolution which opposed compulsory area-wide planning for health facilities. The resolution also requested that the Council on Medical Jurisprudence alert the corresponding committees of the various county medical societies so that they in turn may warn the governing boards of the hospitals in their areas of the dangers inherent in such compulsory planning.

A resolution in opposition to nationalization of medicine was approved. Other resolutions approved included one endorsing and urging county societies to exert their influence to get people to vote favorably on the amendment to the Texas Constitution enacting the Medical Aid for the Aged portion of the Kerr-Mills Law, one citing the need for more family physicians and asking medical school deans to assist with this problem, and one commending Representative W. H. Miller for his assistance to Texas physicians for better patient care in this state.

Legislation Urged

In other legislative proposals, the delegates recommended the appropriation of enabling funds by the Legislature to permit the Texas State Department of Health to enforce the present licensing standards of nursing homes. They also commended the efforts to promote a voluntary accreditation program and the principle of raising standards of nursing home care. Also approved were recommendations from the Committee on Mental Health that a privileged communications bill be passed in Texas and that opposition to legislative certifi-

Dr. Jasper Arnold, third from right, chairman of TMA Committee on Scientific Exhibits, poses with a group who won prizes for outstanding exhibits.

Members of the Fifty Year Club share a reserved table and recognition from their colleagues at the Annual Luncheon.

Speaker at a General Meeting Luncheon was Dr. Fredrick J. Stare of Harvard whose topic was "Nutritional Nonsense."

Canadian guest speaker, Dr. R. G. Bell, discusses defensive thinking at Symposium on Alcoholism.

Dr. John N. Hunt, Department of Physiology, Guy's Hospital Medical School, London, ponders a question at a refresher course on testing gastric function.

Proved popular for the second year were the Curbstone Consultations. Immunization outranked other tables in attendance. Many physicians remained at the tables still in discussion after the session had ended. There were ten tables in all.

cation of psychologists be continued.

Delegates agreed with the Committee on Industrial Health that medical supervision of industrial nurses should be encouraged where possible. They also agreed that county societies should be made aware of information collected by the Committee on Aging on problems and opportunities for the aged of Texas. This is available in the Memorial Library.

Delegates commended the Board of Trustees for the cost study of the *Journal* and for its efforts to reduce expenditures by recommending separate publication of the transactions of the House of Delegates. In the past, the transactions have been published normally in the June *Texas State Journal of Medicine*, but delegates' action now will allow the transactions to be published separately. Summaries will be published in the *TMA Daily Bulletin* issued on the final day of the Annual Session and later in the *Journal*.

It was also recommended that orientation lectures be augmented into job-descriptive-type handbooks for officers of county societies, including especially descriptions for members of the Board of Censors and the grievance committees.

Delegates agreed with the Board of Trustees by turning down a request from the Committee on Cardiovascular Diseases which called for a page in the *Journal* to be designated each month to the Committee. Instead the trustees felt that such action would establish a precedent and that the Association's 51 other committees might logically request equal space. Each contribution will be evaluated on the basis of merit.

Delegates also approved a number of recommendations from the President of Texas Medical Association, Dr. Tenery, including a recommendation that a study be made of improving the functions of district societies, a study by committees at their next meeting to explore the possibility of combining duties with those of other related committees and to suggest changes in reports to their councils at the September and January meetings of TMA.

Endorsed also was the need for a hospital in Galveston for patients from the Texas Department of Corrections, a suggestion by a special committee and the President.

Delegates rose to a moment of silence at Dr. Tenery's suggestion to observe the deaths of Dr. George Waldron, immediate past president, and Dr. Troy Shafer, the late chairman of the Board of Trustees.

AMA-ERF Gifts Encouraged

Delegates approved actions of the Committee for American Medical Association Education and Research Foundation encouraging the donations to that fund. Included in this recommendation was one calling for annual voluntary contributions of $25 to be included in invoices with a statement for AMA, TMA, and county medical society dues. A notice would be included advising the physician that the contribution to AMA-ERF was voluntary.

Delegates also strongly endorsed the preceptor training program and encouraged deans of medical schools to assist in promoting the program among their students.

Receiving a commendation from the delegates for the Shriners Burns Institute in Galveston was the Ancient Arabic Order, Nobles of the Mystic Shrine of North America (A.A.O.N.M.S.) Delegates also commended TMA's General Counsel Philip R. Overton for his 25 years of distinguished service to the Association and presented him with a plaque.

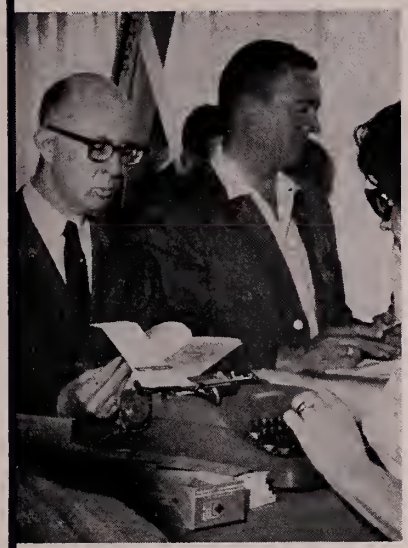
Judges Award Prizes To Scientific Exhibitors

Out of the 54 scientific exhibits entered in this Annual Session, 12 were judged award-winning efforts by a committee of physicians.

Winning first place in Group I, Individual Exhibits, was "Contrast Arthrography of the Knee Joint," prepared by Dr. William P. Price and Dr. Neill B. Longley, both of Houston. Second place winner was "Mammography in Private Practice," exhibited by Dr. Charles W. Yates, Dr. Leslie L. Lemak, Dr. John E. Martin, and James M. Keegan Associates, Houston. Third place went to "Aorto-Iliac Surgery" entered by Dr. Jesse E. Thompson, Dr. Dale J. Austin, and Dr. C. Gene Wheeler, Dallas.

In the Group II, Group Exhibits, division, "Study of Chronic Lung Disease by Inflation-Fixation Techniques" won first place. Exhibitors of this display were Dr. S. Donald Greenberg, Dr. Daniel E. Jenkins, Dr. Robert M. O'Neal, Dr. David Bahar, and Dr. H. Irving Schweppe, Jr., Houston.

Second place, Group II, went to "Use of Low Molecular Dextran in Acute Myocardial Infarction" exhibited by Dr. Per H. Langsjoen, Temple. "Open Heart Surgery Using Dispos-



Registering at Annual Session

Total Attendance Highest Ever

Registration at the Texas Medical Association Annual Session totaled the highest ever—4,093. Registration of members dropped a little under the 2,168 physicians attending last year's meeting in Dallas. A breakdown follows:

Members	2,106
Guests	855
Exhibitors	592
Auxiliary	540
Total	4,093

able Oxygenators and 5 Per Cent Dextrose Prime at Normothermia," an exhibit by Dr. Denton A. Cooley, and Dr. Arthur C. Beall, Jr., Houston, won third place.

In Group III, Institutional Exhibits, first place, was awarded to "Otolaryngologist and the Hearing Aid Selection," prepared by Dr. H. E. Maddox, III, of Galveston. "Hernia Repair with Polypropylene Mesh," the entry of Dr. Francis C. Usher, Houston, placed second. Third place went to "Complications of Biliary Tract Surgery," an exhibit by Dr. Gerald S. Dowdy, Jr., Dr. James L. Moore, Dr. Alfred E. Smith, and the late Dr. George W. Waldron, Houston.

First place in Group IV, Educational Exhibits, went to "Syphilis Is Where You Find It," exhibited by the City of Houston Health Department. Second and third places went to Texas Political Action Committee and the Vocational Rehabilitation Division of Texas Education Agency.

Research Foundation Aids Three Schools

Checks of $7,500 each were presented to the three Texas medical schools in initial payment of $15,000 grants from the Medical Research Foundation of Texas.

Given for unrestricted research during the next year, the grants were presented during the Annual Session in Houston.

Sloan Wingert of Houston, executive vice president of MRFT, made the presentations to the deans of the schools: Dr. A. J. Gill, University of Texas Southwestern Medical School; Dr. Stanley Olson, Baylor University College of Medicine; and Dr. John B. Truslow, University of Texas Medical Branch in Galveston.

TMA Delegates Honor 25 Medical Colleagues

Twenty-five Texas physicians were elected to Honorary Membership in Texas Medical Association by their colleagues in the House of Delegates Friday night. Two of the 25 were elected posthumously.

New Honorary Members include three physicians from Houston, Drs. Thomas H. Compere, F. Hartman Kilgore, and James M. Mitchner; three from Fort Worth, Drs. Charles P. Hawkins, Hub E. Isaacks, and Richard J. White; and two from Dallas, Drs. William H. Potts, Jr., and Wayne T. Robinson.

Other doctors receiving this honor were William C. Barnard, Corpus Christi; Arthur W. C. Bergfeld, New Braunfels; William A. Chernosky, Temple; Ben H. Griffin, Frost; Joseph E. Kanatser, Wichita Falls; Bedford A. Kirkpatrick, Taylor; Matthew F. Kreisle, Sr., Austin (posthumously); and William K. Logsdon, Corsicana.

Drs. Robert E. Mann, Pleasanton; Frank P. Miller; Dewey W. Peace, Sr., Bishop; Elisha P. Powell; Fred L. Story, Ennis; T. Edgar Tabb, Waco; William C. Tenery, Waxahachie; E. R. Townsend, Eastland (posthumously); and William C. Windham, Center, complete the list.

Ten Physicians Named Golf Tourney Winners

A Houston physician, Dr. Fred P. Thomas, won the medalist trophy as low gross winner in the Championship Flight in the golf tournament held during the Annual Session at the Westwood Country Club.

Low net winner in the Championship Flight was Dr. L. E. Ramey, Kingsville, who was awarded a gift certificate.

Winners of four other flights were announced.

The champions are listed

below by flights.

First Flight: Dr. Malcolm D. Dinges, Jr., Houston, net; Dr. G. I. Anderson, Waco, gross.

Second Flight: Dr. W. R. Whitehouse, Cleburne, net; Dr. Homer A. Taylor, Jr., Houston, gross.

Third Flight: Dr. Louis R. Robey, Houston, net; Dr. Rodney Rodgers, Houston, gross.

Fourth Flight: Dr. Frank H. Kidd, Jr., Dallas, net; Dr. Arthur C. Beall, Jr., Houston, gross.

Memorial Services Eulogize 179

Memorial Services for deceased members of the Texas Medical Association and the Woman's Auxiliary held at the Annual Session honored a total of 179 deceased physicians and Auxiliary members.

Dr. Jared E. Clarke of Houston gave a memorial address for 140 deceased physicians, and Mrs. G. Bedford Brown of Angleton spoke in memory of the 39 deceased members of the Auxiliary.

541 Auxiliary Members Register at Convention

Five hundred forty-one members of the Woman's Auxiliary were registered for the 46th Annual Convention in Houston, April 23-26.

Mrs. Hamilton Ford, La Marque, outgoing president, reported that the Woman's Auxiliary to TMA continues to rank second in size in the nation, with a current membership of 6,242.

Contributions made throughout the year to American Medical Association Education and Research Foundation total over $19,000, and members voted to contribute the interest from the Special and General Funds, amounting to $895.76, to AMA-ERF this year.

A contribution of $1,000 from the Special Fund is being given this year to the Physicians Benevolent Fund in memory of the deceased Auxiliary members.

Mrs. William B. Allensworth of Mineral Wells was named "Outstanding Council Woman —1964" and was presented an Auxiliary charm bearing this inscription at the Council Woman's Meeting and Tea.

The silver cup for largest percentage increase in membership was presented to the Woman's Auxiliary to Palo Pinto - Parker - Young - Jack - Archer Counties Medical Society.

Mrs. Ford, other 1963-1964 officers, and past presidents were honored at a tea at the Doctor's Club.

Mrs. W. D. Nicholson New Auxiliary Head

Mrs. W. D. Nicholson of Lake Jackson was installed as 1964-1965 president of the Woman's Auxiliary to the Texas Medical Association at a luncheon in Houston during Annual Session. Installation ceremonies were conducted by Mrs. Mark H. Latimer of Houston. The luncheon, followed by a style show, closed the regular sessions of the Auxiliary's 46th annual convention.

Mrs. Nicholson

Other officers installed were Mmes. Russell L. Deter of El Paso, president-elect; James A. Hallmark of Fort Worth, first vice president; MacField McDaniel of Pampa, Western regional vice president; James C. Terrell of Stephenville, Northern regional vice president; Frank M. Posey, Jr., San Antonio, Southern regional vice president; William M. Palm of Houston, Eastern regional vice president, A. O. McCary of Freeport, corresponding secretary; Scott Haggard of Denton, recording secretary; Ralph C. Patrick of Houston, treasurer; Tom Oliver of Waco, publicity secretary; Richard C. Bellamy of Liberty, parliamentarian.

Mrs. Nicholson is a member of the Woman's Auxiliary to Brazoria County Medical Society and a past president of this auxiliary. At the state level, she has held the positions of president-elect, council woman, regional vice president; first vice president, chairmanships of committees on the school of instruction, science fair, historical-awards, voluntary health plans, and nurse recruitment. While serving as Recruitment Chairman, Mrs. Nicholson was instrumental in the writing and distribution of the Manual for Future Nurses Clubs and in arranging the first Statewide meeting of the Future Nurses Clubs in Texas.

In addition to her Auxiliary work, Mrs. Nicholson has been active in youth organizations, garden club, and church work. She has been organist for her church for more than ten years.

Dr. Byron York presents TMA's General Counsel Philip R. Overton with a plaque commemorating his 25 years of distinguished service to the Association.

Harris County Medical Society's "Moonmaid" has a strange anatomy which includes a Geiger counter instead of a heart. Checking the level of radioactivity through his stethoscope is Dr. Charles Berry, NASA, chief of the Center Medical Operations Office.

A ribbon cutting ceremony officially opens the technical and scientific exhibits. Wielding the shears is Dr. R. M. Tenery. Watching is Dr. Jasper H. Arnold, chairman of TMA Committee on Scientific Exhibits.

Arriving at their Houston hotel for Annual Session are Dr. and Mrs. Charles B. Sadler who drove down from Amarillo.

Dr. William M. Crawford, chairman of the Committee on Medical History, and Dr. Hampton C. Robinson examine an anatomy book written by Andreas Vesalius and published in 1555. Dr. Robinson donated the book to the TMA Memorial Library in memory of the late Dr. George Waldron, Past President of TMA.

No. 100

AUSTIN PSYCHIATRIST NAMED PRESIDENT-ELECT

THE 100th President of Texas Medical Association will be Dr. David Wade, Austin psychiatrist. He was named President-elect at the 97th Annual Session in Houston April 26.

The new Vice President of Texas Medical Association is Dr. Travis Smith, a former member of the Board of Councilors and former president of Taylor-Jones Counties Medical Society. The Abilene internist had practiced in Temple and served in the Navy during World War II before moving to Abilene in 1948.

New members elected to TMA councils are Dr. Alice Lorraine Smith, Dallas, Council on Annual Session; Dr. Jack B. Lee, San Antonio, Council on Constitution and By-laws; Dr. Bryan V. Williams, Houston, Council on Medical Education and Hospitals; Dr. Doyle W. Ferguson, Dallas, Council on Medical Jurisprudence; Dr. Robert F. Gossett and Dr. Jack M. Partain, both of San Antonio, Council on Medical Service and Insurance; and Dr. O. E. Harper, Abilene, and Dr. Benjamin M. Primer, Austin, Council on Public Relations and Public Service.

Dr. Harvey Renger, Hallettsville, was elected delegate to the AMA, and Dr. James H. Sammons of Highlands was elected alternate delegate. Delegates reelected were Dr. John K. Glen, Houston; Dr. G.W. Cleveland, Austin; and Dr. John L. Otto; Galveston. Alternate delegates reelected were Dr. Nym L. Barker, Paris; Dr. Ridings E. Lee, Dallas; and Dr. Edward P. Hall, Jr., Fort Worth.

Next year will be the first time in 50 years that the Association will be headed by a psychiatrist. Two other Presidents, Dr. G. H. Moody in 1915 and Dr. Marvin L. Graves in 1913, specialized in the treatment of mental and nervous disorders.

Dr. David Wade

Dr. Wade has been in private practice in Austin since January, 1945, and has operated a hospital there since 1947 when he established Oak Ridge Sanitarium.

Before moving to Austin, Dr. Wade had been superintendent of the State Psychopathic Hospital in Galveston, the Austin State School for Mental Defectives, and Rusk State Hospital. During the Korean Conflict (1951-1952), he was stationed in San Antonio as a psychiatric consultant in charge of psychiatric facilities for the Fourth Army.

He received most of his preliminary education in Jacksonville, Ark., where he was born in 1910. He attended Little Rock Junior College (now Little Rock University) and the University of Arkansas in Fayetteville, graduating with a degree in business in 1930. He received his M. D. degree from the University of Arkansas School of Medicine in Little Rock, interned at St. Vincent Infirmary, and served his residency at Galveston and a visiting residency in 1944 at New York Psychiatric Institute.

His medical affiliations include the Travis County Medical Society, American Medical Association, American Psychiatric Association, and Texas Neuropsychiatric Association. He has served as president of the latter and also the Seventh District Medical Society. For nine years, he was councilor for the Texas Medical Association, and last year he was on the Council on Constitution and By-laws. Currently, he is president of the Texas Rehabilitation Association and medical administrative consultant to the Texas Education Agency.

Pharmacy Board Seeks Cooperation of Physicians

The Texas State Board of Pharmacy is seeking the cooperation of Texas physicians in assisting pharmacists in each community to abide by state laws and statutes which govern the practice of pharmacy.

The Board is seeking to stop substitution of drugs without the authorization of physicians, to stop the over-the-counter sale of legend item pharmaceuticals without a prescription, to curtail refilling of prescriptions without the physician's authorization,

and to stop the filling of prescriptions written by physicians who subsequently have died.

The Texas State Board of Pharmacy presently is embarking upon an educational program to inform all pharmacists of their responsibilities. The Board has set forth 15 questions on the back of an application which all pharmacists must submit each year for a store permit. The questions remind pharmacists of their responsibilities to their profession, to doctors of medicine, and to the public.

Stopping Smoking Lowers Patient Death Rate

A lower mortality rate has been found among bronchitis and emphysema patients who stopped smoking compared with similar patients who continued to smoke, three physician-researchers report.

A correlation between the amount of smoking and the presence of chronic bronchitis and emphysema also was found, according to Drs. Roger S. Mitchell, Thomas N. Vincent, and Giles F. Filley, Denver.

The physicians reported on a study of 150 patients with chronic lung disease conducted between July, 1956, and July, 1961, in the April 6 *Journal of the American Medical Association.*

Smoking histories were obtained at the start of the study and all 144 smokers were warned to stop, the authors said. However, only 42 per cent, or 60 patients, were able to do so.

Of the 60, 44 noted complete or almost complete relief from coughing, according to the report.

These 44 experienced a lower mortality rate during the 20 years following onset of their disease than did the 84 who continued to smoke. The mortality rate at the end of 20 years was about 50 per cent among those who gave up smoking and nearly 100 per cent among those who did not.

The expected frequency of smoking in this study group composed predominantly of men, aged 5 to 75, according to a 1958 U.S. Public Health Service survey, would have been about 75 to 80 per cent, it was also pointed out. However, the observed frequency was 95 per cent. In addition, these smokers were more apt to be heavy smokers than an average U.S. population.

A postmortem study of a lung from each of 125 patients who died with or without apparent signs of chronic lung disease was also made to determine the amount of lung damage due to emphysema and the amount of black pigment deposited throughout the lung, the authors reported. This group included 18 patients in the previous study.

Of 40 patients with more than 25 per cent lung destruction, all except two were regularly moderate to heavy cigarette smokers. Of 28 patients with definite diagnoses of chronic bronchitis, all except one were again regularly moderate to heavy cigarette smokers.

The nonsmokers and light smokers tended to have light lung pigmentation and postmortem examination tended not to reveal chronic bronchitis or emphysema.

A light smoker was defined as one who smoked up to five cigarettes a day, a moderate smoker 15 to 25 cigarettes a day, and a heavy smoker more than 30 a day.

The black pigment which is found in all adult human lungs has been labelled anthracosis, the physicians explained. It could be harmless carbon or soot, they said, but on the other hand could be a tar from petroleum and/or cigarette smoke or some other biologically active and potentially harmful substance.

"It is very likely to be a mixture of materials, depending upon the individual's past environment," they said.

The correlation between the amount of smoking and the presence and degree of chronic bronchitis and emphysema and of lung pigmentation by no means proves that cigarette smoke is a cause of emphysema, a disease clearly described in the 19th century before cigarettes were generally available, the physicians stressed.

However, they said, accumulating evidence indicates that cigarette smoking is one of the major causes of chronic bronchitis, outweighing in importance the effect of community air pollution.

"Very little evidence has been accumulated regarding the importance of inhaling tobacco smoke as an influence on its ability to do harm," they commented. "The nose is a remarkably effective filter, humidifier, and warmer of the air we breathe. Industrial and community air pollutants are inhaled mostly through the nose; this is not true of tobacco smoke."

Camp Opportunities for the Handicapped Child

IN A FEW WEEKS, school will be out and children will be heading for summer camp. For children whose handicaps will not permit them to participate in camps for normal boys and girls, Texas offers several camps specifically designed for their needs.

Camp Manison in Friendswood operates a diabetic camp session June 7-19 for children age 6 to 16. Campers are accepted only upon recommendations from physicians, according to Thomas A. Manison, director.

Fees for this session are $130, but campers may receive aid to pay a portion or all of the fee. "Normally the camperships are furnished by a local civic or service club," Manison explains.

In its private camp sessions, a coed session from June 21 to July 4 and a boys camp session from July 5 to 31, Camp Manison accepts asthmatic children in addition to those with diabetes. All buildings are air-conditioned and a medical staff is in residence.

Camp Manison is located on a wooded tract two miles east of Friendswood between League City and Pearland.

Some of the activities at camp include swimming, riding, fishing, athletics, Indian lore, nature study, archery, crafts, and marksmanship.

Campers are given the opportunity to learn to collect their urine specimens, give themselves insulin injections, and manage their diets with supervision.

Registrations are handled by Miss Gene Christie, executive secretary, Houston Area Diabetes Association, Her-

mann Professional Building, Houston.

Camp Sweeney Diabetic Health Center, located nine miles northeast of Gainesville, will provide three terms of three weeks each during the summer. The first session will be from May 31 to June 20; second session, June 21 to July 11; and third session, July 12 to Aug. 1.

Children from 6 to 16 years of age are accepted and divided into junior, intermediate, and senior divisions, each with separate living quarters, classes, and special events.

A counselor is assigned to every four boys and girls, and each camper with the supervision of a counselor selects the activities in which he or she wishes to participate, such as crafts, art, music, square and folk dancing, and horseback riding. Afternoons are devoted to team play and development of group spirit with baseball games, swimming, boat races, and hikes.

Dr. J. Shirley Sweeney and the staff doctor hold medical forums for each camping group.

The camp medical director makes daily visits, and two resident physicians are on duty at all times. A complete health chart is kept on each camper and filed in one of the infirmaries which are situated in the barracks. Each camper is weighed and examined daily, and all insulin injections are given by or under the supervision of a doctor.

Children are admitted to the camp on the basis of what their family can afford to pay. This amount may be paid in the manner preferred by the parents.

Information may be obtained from James V. Campbell, 911 Lindsay Street, Gainesville.

THE DEVEREAUX Schools sponsor camps in connection with their school in Victoria for all age groups of children. Children may attend the camp only if they are enrolled in the Devereaux Schools or if they plan to enter the coming year.

This is a camp primarily for the emotionally disturbed, but mental retardation and all types of handicaps are considered on an individual basis. Only ambulatory patients without severe involvement are accepted.

Camp sites are maintained at Victoria and in the hill country in the Marble Falls-Burnet area, and plans are being considered to establish camps at Tyler and Austin.

The fee is $500 to $700 per

month based on individual needs, and the camp starts at the end of the first week in June, continuing for ten weeks.

Services are available in physical, occupational, and speech therapy; remedial work; individual counseling; and therapy and training to meet problems of retardation or emotional disturbance. The program for each child is determined by psychiatric, psychological, educational, medical, vocational, and physical education departments.

Twenty-six activities were offered last year including canoeing, boating, hiking, arts and crafts, swimming, and diving.

Information may be obtained from Richard G. Danko, chief administrator, Devereaux Schools in Texas, Box 2269, Victoria.

HOUSTON COUNCIL for Retarded Children Camp Program operates a two-week camp Aug. 1-14. The residential camping program is held in Baytown at Camp Allen, located on a site overlooking Trinity Bay.

"Housing facilities are sufficient to accommodate 100 to 150 persons," according to Frank A. Borreca, executive director, and the staff consists of a trained dietitian, nurse, swimming instructor, arts and crafts instructor, 14 to 20 trained counselors, and a camp director.

Total fee for the camp period is $70, and scholarships are provided for those children who are not able to pay the fee. "No child is turned away because of lack of finances," Mr. Borreca says.

The program is open to both educable (I.Q. 50-75) and trainable (I.Q. 30-50) mentally retarded children from the age of ten and up. They are required to be toilet trained and capable of feeding themselves.

The program consists of swimming, arts and crafts, physical activities, group games, square dancing, hiking, crabbing, music, story telling, movies, and cook-outs.

Information may be obtained through the Houston Council for Retarded Children Camp Program, Opportunity Center, 3410 Montrose Blvd., Houston.

SOROPTIMIST CLUB of Dallas Crippled Children's Camp, a member of the American Camping Association, is located in Argyle. Children with orthopedic and neuromuscular handicaps, mental retardation, and others are admitted.

Seven sessions are held from June to August, and the capacity is 72 per period. The first session is June 7-13 for physically handicapped children age 6 to 10. June 14-27, the second session, will be for physically handicapped young adults 18 and up. The third session, June 28 to July 4, is for the young mentally retarded group, and from July 5 to 18 intermediate mentally retarded children will be accepted. The fifth session, from July 19 to Aug. 1, is for physically handicapped children age 11 to 13, and children age 14 to 16 with physical handicaps will be accepted Aug. 2-15. The seventh session will be for the older group of mentally retarded persons and will be held Aug. 16-25.

Fees for the short sessions are $30 and for the long sessions, $60. Dallas County children are admitted free.

The usual camping activities are available including swimming, riflery, badminton, and other games.

Applications may be made to Camp Soroptomist, 3415 Milton, Dallas. All applications should include the name, age, handicap, parent's name, address, phone number, and person or institution who referred the child.

THE TEXAS LIONS Camp for Crippled Children is located three miles southeast of Kerrville, high in the hills overlooking the Guadalupe river valley.

Crippled, blind, deaf, and mute children may receive supervised camping and rehabilitation during the months of June, July, and August without charge. Children from 7 through 16 may participate in the program if they can participate fully in the modified camping program offered.

The camp operates five summer sessions starting the first Sunday in June. Each camping period is 13 days.

Program activities include swimming under supervision of a Red Cross water safety instructor, arts and crafts, camp-outs, horseshoes, chess, shuffleboard, Ping-Pong, bowling, baseball, volleyball, golf, archery, journalism in which campers write articles for the camp newspaper, music, dramatics, and special programs.

More information about this camp may be obtained from Frank Robertson, executive director, Texas Lions Camp for Crippled Children, Box 247, Kerrville.

In addition to these camps, the Texas Association for Retarded Children sponsors day camps for mentally retarded children. Approximately 20 such camps were held last summer, and information about them may be obtained by writing local councils of the Texas Association for Retarded Children.

Personals

Dr. J. H. Caton Honored

Dr. J. H. Caton, retired Eastland physician, recently was presented a 50-year membership certificate from the Order of the Eastern Star.

Dr. John Kinross-Wright, director of clinics for the Houston State Psychiatric Institute and professor of psychiatry at Baylor University College of Medicine, Houston, and *Dr. Robert W. Johnson*, practicing psychiatrist in Corpus Christi, have been appointed by Gov. John Connally to the Committee to Study Sociopathic Personalities.

New officers of the Texas Association of Obstetricians and Gynecologists elected in Corpus Christi are president, *Dr. John A. Wall*, Houston; vice president, *Dr. Jack A. Pritchard*, Dallas; secretary-treasurer, *Dr. James T. Downs, III*, Dallas; and president elect, *Dr. Hugh W. Savage* of Fort Worth.

Dr. Edmund Fountain of Houston has been appointed to the third rotation team of the teaching - training hospital ship S.S. HOPE, now anchored at Guayaquil, Ecuador.

Dr. J. Richard Crout, assistant professor of pharmacology and internal medicine, Southwestern Medical School, spoke at the March meeting of the Tarrant County Medical Society on the subject of "Modern Concepts in Uses of Digitalis."

Dr. Eric Anderson is leaving Groveton to practice in New Hampshire. His practice has been taken over by *Dr. Neil Bowie* of Glasgow, Scotland. Dr. Bowie was attracted to Texas by an ad placed by Dr. Anderson in the *British Medical Journal*.

IRS Amendments Opposed by AMA

The American Medical Association, along with a number of professional persons and representatives of national organizations at a public hearing in Washington, protested against amendments proposed by the Treasury Department to Internal Revenue Service regulations concerning professional service groups.

The amendments would neutralize the professional corporation and association laws passed by more than 30 states. These state laws have allowed professional men to incorporate and enjoy corporation tax treatment, beneficial in that it allows the privilege of postponing federal tax on income set aside in retirement plans until it is paid out as benefits. Usually, the beneficiary by this time is in a lower tax bracket because of his age and status.

There is no such law in Texas. The Texas Professional Corporation bill, introduced and defeated in the Texas Legislature last year, was opposed by Texas Medical Association. TMA opposed the bill because it is opposed to the corporate practice of medicine. (See January issue of *Texas State Journal of Medicine*, p. 78).

New Books in TMA Library

Academy of Science U.S.S.R., Works of the Institute of Higher Nervous Activity, Pathophysiological Series, vol. 7 and vol. 8, Washington, D. C., the Israel Program for Scientific Translations, Jerusalem, published for the National Science Foundation.

Adriani, J., ed.: Appraisal of Current Concepts in Anesthesiology, vol. 1, St. Louis, C. V. Mosby Co., 1961. Proceedings of the 19th Annual Sessions of the Forum on Fundamental Surgical Problems, 49th Clinical Congress of the American College of Surgeons, 1963, Surgical Forum, vol. 14, Chicago, American College of Surgeons, 1963.

Bauer, D. D.: Lumbar Discography and Low Back Pain, Springfield, Ill., Charles C Thomas, 1960.

Bockus, H. L.: Gastroenterology, 2nd ed., Philadelphia, W. B. Saunders Co., 1964, vol. 2.

Breed, R. S., et al.: Bergey's Manual of Determinative Bacteriology, 7th ed., Baltimore, Williams & Wilkins Co., 1957.

deReuck, A. V. S., and Cameron, M. P., eds.: Lysosomes, Ciba Foundation Symposium, Boston, Little, Brown and Co., 1963.

Conn, H. F., ed.: Current Therapy 1964, Philadelphia, W. B. Saunders Co., 1964.

Cooley, D. G.: The Science Book of Modern Medicines, New York, Pocket Books, Inc., 1963.

Crews, E. R.: A Practical Manual for the Treatment of Burns, Springfield, Ill., Charles C Thomas, 1964.

Ellis, P. P., and Smith, D. L.: Handbook of Ocular Therapeutics and Pharmacology, St. Louis, C. V. Mosby Co., 1963.

Greifer, E.: How I Cured My Incurable Ailments; A Plea for Doctor-Guarded Self-Research, New York, Author, 1958.

Hoffman, W. S.: The Biochemistry of Clinical Medicine, 3rd ed., Chicago, Year Book Medical Publishers, 1964.

Kalb, S. W.: Your Future as a Physician, New York, Richards Rosen Press, Inc., 1963.

Klein, K. K., and Hall, W. L.: The Knee in Athletics, Washington, D. C., American Association for

Health, Physical Education, and Recreation, 1963.

Klosovskii, B. N.: Blood Circulation in the Brain, Washington, D. C., Israel Program for Scientific Translations, Published for the National Science Foundation, 1963.

Ladimer, I., and Newman, R. W., eds.: Clinical Investigation in Medicine: Legal, Ethical, and Moral Aspects, Boston, University Law-Medicine Research Institute, 1964.

Love, A., and Childers, J. S.: Listen to Leaders in Medicine, New York, Holt, Rinehart and Winston, 1963.

Metcoff, J., ed.: Hereditary, Developmental, and Immunologic Aspects of Kidney Disease, Evanston, Ill., Northwestern University Press, 1962.

Mollison, P. L.: Blood Transfusion in Clinical Medicine, 3rd ed., Oxford, Blackwell Scientific Publications, 1961.

Moritz, A. R., and Stetler, C. J.: Handbook of Legal Medicine, 2nd ed., St. Louis, C. V. Mosby Co., 1964.

Moser, R. H., ed.: Diseases of Medical Progress, 2nd ed., Springfield, Ill., Charles C Thomas, 1964.

Nourse, E. S., ed.: American Medical Colleges, 1963-4 Admission Requirements, Evanston, Ill. Association of American Medical Colleges, 1963.

1964 Physicians' Desk Reference to Pharmaceutical Specialties and Biologicals, 18th ed., Oradell, N. J., Medical Economics, Inc., 1963.

Pavlovskii, E. N., ed.: Natural Foci of Human Infections, Washington, D. C., Israel Program for Scientific Translations, Published for the National Science Foundation, 1963.

Race, R. R., and Sanger, R.: Blood Groups in Man, 4th ed., Oxford, Blackwell Scientific Publications, 1962.

ReMine, W. H., et al.: Cancer of the Stomach, Philadelphia, W. B. Saunders Co., 1964.

Stanisavljevic, S.: Diagnosis and Treatment of Congenital Hip Pathology in the Newborn, Baltimore, Williams and Wilkins Co., 1964.

Slaughter, F. G.: A Savage Place, Garden City, N. Y., Doubleday & Co., Inc., 1964.

Stoddard, F. J.: Case Studies in Obstetrics and Gynecology, Philadelphia, W. B. Saunders Co., 1964.

Linsay, J. R., et al., eds.: Year Book of Ear, Nose and Throat, 1963-1964 Series, Chicago, Year Book Medical Publishers, Chicago, 1964.

Hughes, W. F., ed.; Year Book of Ophthalmology, 1963-1964 Series, Chicago, Year Book Medical Publishers, 1964.

New Films in TMA Library

Epileptic Seizures — 1963, sound, color, 24 minutes.

THE FILM shows some historical and geographical aspects of epilepsy. It shows the electroencephalograph and how it is used and interpreted. It pictures actual patients in a state of seizure, along with corresponding EEG tracings, exemplifying various types of seizures, including grand mal, petit mal, automatism, myoclonic, psychomotor, and mixed.

Sterilization Procedures for the Medical Office—1962, sound, color, 29 minutes.

THE FILM defines the processes by which sterilization should be accomplished, and demonstrates how to apply such processes. It includes explanations of proper cleaning and wrapping, length of exposure to sterilizing agents, and conditions for storage of sterile goods. It discusses disinfection procedures and defines the types of materials to which they should be applied.

Book Notes

These books are available from TMA Library.

Organization and Administration in World War II. Col. John Boyd Coates, Jr., MC, USA, ed. 613 pp. Washington, D. C. U. S. Government Printing Office, $6.25.

THIS volume on administra-

"It's another teenager who's very sensitive about her complexion problems."

"I must admit this is the first time I've been asked to help research on piles."

tion and organization emphasizes the explosive increase of the armed services in all directions and the lack of flexibility in organization and in skilled people to administer all facets of the services to the best advantage. In reviewing this book, one has an awareness of the rapidity of expansion of the medical service.

Developing almost from scratch was an enormous air force with new medical problems coupled with a demand for their solution by forces outside the medical profession. It was necessary to expand training facilities to meet this emergency and was probably the primary reason for the development of autonomy and the status of a new service to the Air Force.

It reflected the lack of being ready to delegate authority to take care of developing needs and situations demanding solution. The reviewer is of the opinion that in the future there must be a pooling of medical resources by all services for both economy and adequate coverage. Cross training of medical careers for all eventualities is essential, and one views with some apprehension the open end type of checking account in the missile and space programs since it may prove detrimental to research, development, and progress in our conventional medical services.

—Joe C. Rude, M.D., Austin.

Rhinoscleroma. Francisco Kerdel-Vegas, M.D., Jacinto Convit, M.D., Bernard Gordon, M.D., Mauricio Goihman, M.D., and Marion B. Sulzberger, M.D. 111 pp. Springfield, Ill. Charles C Thomas, 1963. $9.75.

THIS BOOK was written for those dermatologists, otolaryngologists, internists, and epidemiologists who are interested in what is now a worldwide problem—rhinoscleroma.

Treatises on tropical and exotic diseases will be needed in increasing numbers as the United States strives to meet its obligations in many parts of the world.

The present study is the first in a planned series by the newly formed research center in tropical dermatoses established by the dematology department of the Vargas Hospital in Caracas, Venezuela.

Childbirth with Hypnosis. William S. Kroger, M.D., and edited by Jules Steinberg. 216 pp. Garden City, N. Y., Doubleday & Company, Inc., 1961. $3.95.

"CHILDBIRTH WITH HYPNOSIS" is recommended for every expectant mother who wants to know everything science is doing to protect her health and the health of her baby during childbirth.

Dr. Kroger discusses the background, technique, and advantages of using hypnosis in childbirth. He shows how the patient is taught to relax so that the pains of childbirth are decreased and often disappear, yet the full awareness of birth remains.

Chemistry of Chelation in Cancer. Arthur Furst, Ph.D. 143 pp. Springfield, Ill., Charles C Thomas, 1963. $7.50.

THIS COMPREHENSIVE REVIEW (over 600 references are cited) brings to the foreground the relationships between trace metals, the phenomenon of chelation, and cancer.

Correlations of action of most anticancer drugs are possible if these compounds are viewed as chelating agents, rather than as antimetabolites.

Recommendations for the design of new anticancer compounds complete the monograph.

Surgical Treatment of Unequal Extremities. Charles Weer Goff, M.D., Ed. 184 pp. Springfield, Ill., Charles C Thomas, 1960. $11.50.

THE EFFECTS of fractures within youthful age groups on growth are documented by accurate anthropologic measurements. These and other techniques of great practicality with reference to future long bone lengths after trauma are included.

Emphasis is on surgical methods of controlling growth by osteotomies, stimulation by drilling, dissimilar screws, effects of heat; lengthening and shortening osteotomies, and use of stapling to decelerate growth on the long side.

Self-Hypnosis: A Conditioned-Response Technique. Laurance Sparks. 255 pp. New York, Grune & Stratton, Inc., 1962. $5.75.

TAKING PRINCIPLES with which all are familiar in daily life, and, through scientific organization and intelligent application, this book puts them at the disposal of any person seriously interested in increased efficiency in concentration, relaxation, self-control and learning capacity, and in alleviation of pain, correction of self-damaging habits, and control of psychic and psychosomatic symptoms.

Writing for both laymen and interested professionals, the author sums up a wide and successful experience in this manual. Seriously applied, it should increase the intellectual, emotional, and physical potential of those who use it.

Recent Deaths

DR. A. D. CALDEIRA, SR.

Dr. Antonio Dias Caldeira, Sr., who had practiced medicine in Mercedes for more than 25 years, died of a heart attack at his home on Feb. 20. He was 73.

He was born in Port of Spain, Trinidad, British West Indies, on April 17, 1890. He came to the United States to attend the University of Chicago and Loyola University from which he received a bachelor of science degree in 1919. He was graduated in medicine from Loyola University School of Medicine, Chicago, in 1923. He interned at St. Mary of Nazareth Hospital, Chicago.

Dr. Caldeira practiced medicine in British West Indies, Chicago, and Mercedes. His specialty in recent years was geriatrics.

He was a member of the Hidalgo-Starr Counties Medical Society, Texas Medical Association, American Medical Association, American Academy of General Practice, and the Knights of Columbus. He was a Roman Catholic.

He was city housing commissioner and a member of many civic and charitable groups. He was a staff member of the Knapp Memorial Methodist Hospital.

Survivors include his wife; two daughters, Mrs. J. R. Wade, Jr., of Mercedes and Mrs. Dan Love of Houston; two sons, Noel Caldeira of Long Beach, Calif., and Anthony Caldeira, Jr., of Houston; three brothers, Dr. Frederick Caldeira of Harlingen, Manuel Caldeira and John Caldeira of New York; one sister, Mrs. Virginia Rezende of San Diego, Calif., and 17 grandchildren.

DR. J. C. LOCKE

Dr. John C. Locke, 39, a Slaton physician since 1956, died of a heart attack after making his hospital rounds on the morning of Jan. 30.

Dr. Locke, who was born Dec. 2, 1924, in Luling, was a graduate of Austin High School in Austin. He received three degrees from the University of Texas: his B. A. in 1948, his M. A. in 1953, and his M. D. from the Medical Branch in 1955.

He served his internship at King's Daughters Hospital in Temple.

The immediate past vice president of the Panhandle District Medical Society, he was a member of Texas Medical Association, American Medical Association, American Academy of General Practice, American Association of Railway Surgeons, Mu Delta Honorary Medical Service Society, Phi Chi, and Association of American Physicians and Surgeons, Inc. He was the recipient of the Hildebrand Award in 1955.

He was an elder of the Christian church, Khiva Temple Shrine in Amarillo, and the Slaton Masonic Lodge.

Dr. Locke's research activity had centered on physiology and circulation. He was the author of several published medical papers.

DR. A. D. CALDEIRA, SR.

DR. J. C. LOCKE

He was chief of staff at Mercy Hospital in Slaton.

During World War II he served in Europe in the Third Army under Gen. George S. Patton.

His hobbies were golf and playing the organ.

He was married Sept. 2, 1946, in Austin to Constance Shirley Henry.

He is survived by his wife and three children, Cynthia Gail Locke, Melissa Ann Locke, and John Childress Locke, Jr., of Slaton; his parents, Mr. and Mrs. Will H. Locke of San Antonio; two sisters, Mrs. Wallace R. Langston of Dallas and Mrs. A. F. Thompson of Carlisle Barracks, Pa.; and a brother, Bruce M. Locke of El Paso.

DR. M. YEN

Dr. Moore Yen, 48, assistant professor in the Department of Anesthesiology at the University of Texas Medical Branch, died in a Galveston hospital on Feb. 29 after a brief illness.

Dr. Yen, born in Tientsin, China, had been associated with the Medical Branch since 1948. He received his education at Tientsin Nankai Middle (high) School in Tientsin, Cheeloo University in Shangtung, China, and West China University Medical School, Chengtu, China, from which he received his M. D. degree in 1942. He interned at the teaching hospital near this medical school. He served his residency in anesthesiology at the University of Texas Medical Branch when he first arrived there.

He was a member of the Galveston County Medical Society, Texas Medical Association, American Medical Association, Texas Society of Anesthesiologists, and American Society of Anesthesiologists. In addition to being an active member of the Medical Branch hospital staff, he was also a consultant member of the staff of St. Mary's Infirmary. Dr. Yen was certified by the American Board of Anesthesiology and was a fellow in the American College of Anesthesiologists.

His wife whom he married in Galveston on Aug. 21, 1954, is the former Helen Hing-Ng Siu of Hong Kong.

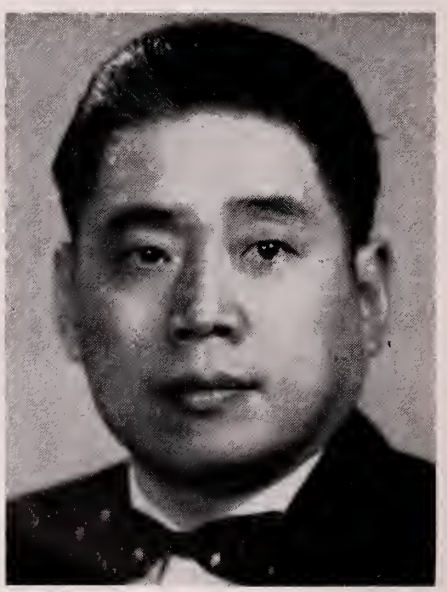
He is survived by his wife and one son, Harry.

DR. M. F. KREISLE, SR.

Dr. Matthew Ferdinand Kreisle, Sr., 77, Austin internist, died in an Austin hospital Feb. 25.

He was a lifetime resident of Austin and the first physician there to emphasize and use electrocardiography. One of the five children of Louis Henry and Lottie (Shipman) Kreisle, he was born in Austin Dec. 8, 1886, and attended the schools there including the University of Texas.

He was a graduate of the University of Texas Medical Branch in Galveston and interned at John Sealy Hospital. He remained in the island city in the capacity of instructor in pathology at the medical school until 1915 when he returned to Austin to begin



DR. M. YEN

DR. M. F. KREISLE, SR.

practice. For several years after his return, he was the Travis County physician.

Dr. Kreisle was a veteran of World War I in which he served as a first lieutenant in the Army Medical Corps. He was awarded the Congressional Selective Service Medal for his work in connection with the draft during World War II.

He was twice president of the Travis County Medical Society and was also a member of Texas Medical Association and American Medical Association. He belonged to two medical fraternities, Alpha Omega Alpha and Phi Chi.

A 32nd degree Mason, he was a member of the Ben Hur Shrine and Scottish Rite Bodies. He was a member and deacon of the First Southern Presbyterian Church of Austin.

Dr. Kreisle was a bibliophile of wide interests, especially in the fields of history and Texana.

He and Mrs. Kreisle, the former Margaret Hanna of Galveston, were married on Sept. 5, 1917. She survives him as do three children, Dr. James E. Kreisle, Mrs. Margaret Straw, and Dr. Matthew F. Kreisle, Jr.; one brother, Edwin C. Kreisle; three sisters, Mrs. Latrelle Thompson, Mrs. Bernard Werkenthin, and Mrs. William S. Gatewood; and 12 grandchildren. All are from Austin.

DR. J. W. WOODWARD

Dr. Joseph Weldon Woodward, 46, of Waco died Feb. 11 in Houston of Hodgkin's disease.

Dr. Woodward designed a special operation, muscle origin release operation for congenital elevation of scapula, to correct Sprengel's deformity in children. The first operation of this kind was performed in June, 1956, and in 1960 Dr. Woodward presented his paper to the American Academy of Orthopaedic Surgeons in Chicago and to the Society of Orthopedic Surgery and Traumatology (SICOT) in New York.

Born in Roscoe, Tex., in 1917, his parents were E. J. and Ivah Mae (Barkley) Woodward. On graduating from Brownwood High School, he attended Baylor University and received a B. A. degree in 1939. He graduated from the University of Texas Medical Branch in Galveston in 1942 and served his internship in Philadelphia at Graduate Hospital of the University of Pennsylvania.

Dr. Woodward served as a Navy lieutenant aboard a destroyer from 1943 to 1946.

DR. J. W. WOODWARD

He was released from active service in the Navy in 1946.

In 1946 also he married Miss Jean Armor in Sweetwater.

After practicing in Waco and Thornton, Dr. Woodward went to Dallas in 1948 to serve a rotating orthopedic residency at Parkland and Baylor Hospitals and Scottish Rite Hospital for Crippled Children. He returned to practice in Waco in 1951.

He was the author of two papers, "Treatment of Osteomyelitis" and "A Different Operation for Congenital Elevation of the Scapula," which were published in the *Journal of Bone and Joint Surgery* in 1950 and 1960.

His medical affiliations included the McLennan County Medical Society, Central Texas Journal Club, American Academy of Orthopaedic Surgery, Western Orthopedic Society, Texas Surgical Society, Texas Medical Association, and the American Medical Association. He served on the staffs of both Hillcrest Baptist Hospital and Providence Hospital and acted as consultant to James Connally Air Force Base and the Veterans Hospital.

He was director of Lake Air National Bank, a member of the board of directors of the Baylor Ex-students' Association, of the Ridgewood Country Club, of the committee "Operation Second Century" for looking into the future needs of Baylor University, and of St. Paul's Episcopal Church.

Survivors include his wife; three children, Kay, Lynn, and Joe Woodward, Jr., of Waco;

parents of Sweetwater; a brother, Jack S. Woodward of Dallas; and two sisters, Mrs. Smith Bell of Brownwood and Mrs. Robert Faver of Sweetwater.

DR. J. W. JERMYN

Dr. John W. Jermyn, 42, died Jan. 13 in a Dallas hospital.

A Korean War veteran, Dr. Jermyn received the Bronze Star for risking his life in treating wounded soldiers in October, 1951.

He was born in Everett, Mass., in 1921 and attended school at Northeastern University in Boston, Texas A&M University, and Baylor University. He was graduated from Southwestern Medical School in 1950, and interned at Baltimore City Hospitals from 1950 to 1951.

He was a member of the Tarrant County Medical Society, the Texas Medical Association, and the American Medical Association.

Before moving to Fort Worth in 1955, Dr. Jermyn had practiced in Pampa and Dumas.

He was a member of the Glenview Hospital board of directors and a trustee for the Birdville Independent School District.

He had served as team physician to the Haltom High School athletic teams since 1955. He was a member of St. Nicholas Episcopal Church, Moslah Temple Shrine, and the Diamond Oaks Country Club.

He is survived by his wife, Patricia, two children, John William, III, and Deborah Kay, all of Fort Worth; and his mother, Mrs. Margaret K. Jermyn of Boston.

DR. J. W. JERMYN

DR. E. R. TOWNSEND

Dr. Edwin R. Townsend, 77, a practicing physician in Eastland since 1919, died Feb. 29 in an Eastland hospital where he had been a patient for two weeks.

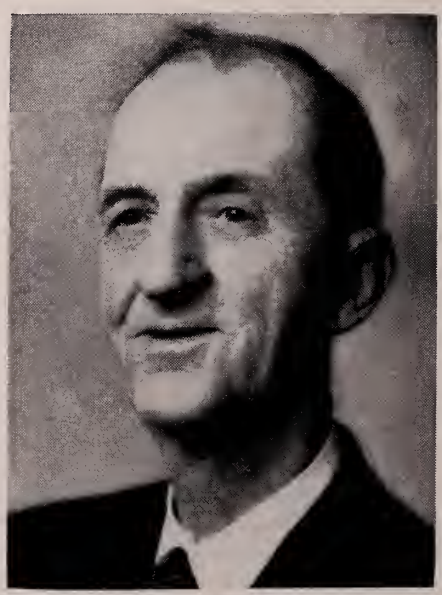
He was a native of Eastland, born Aug. 22, 1886, the son of Dr. and Mrs. E. D. Townsend. His grandparents were among the caravan of six covered wagons bringing the original settlers to that area in 1869.

Dr. Townsend was one of the youngest physicians ever to start medical practice; he began at 21. He was also a registered pharmacist.

His early education was in the schools in Llano where his family had moved from Eastland when he was ten years old. Following graduation from Tulane University School of Medicine, he interned at Physicians and Surgeons Hospital in St. Louis, Mo., and practiced in Fredericksburg before entering the Army in 1917.

He served two years as flight surgeon under Gen. Carl Spaatz in the Army Air Corps during World War I. During the war he married an Army nurse, Edith Helen Shaw of Philadelphia, in Paris, France. Following their tour of military duty, the couple came to Eastland where they have since lived.

Dr. Townsend was a charter member of Eastland Presbyterian Church and of Lone Cedar Country Club. His other memberships included the American Legion which he served as president at one time, Veterans of



DR. E. R. TOWNSEND

Foreign Wars for which he has served as district surgeon, Eastland County Medical Society which he had served as president, Texas Medical Association, and American Medical Association.

He had served for the past ten years as Eastland County health officer and had assisted in civic and health programs in the county.

Survivors are his wife and one sister, Mrs. John Kinberg of Eastland.

DR. M. H. JENSEN

Dr. Martin Hans Jensen, a physician and surgeon of San Antonio since 1941, died Feb. 25. Death was attributed to pulmonary insufficiency due to bronchogenic carcinoma of the lung.

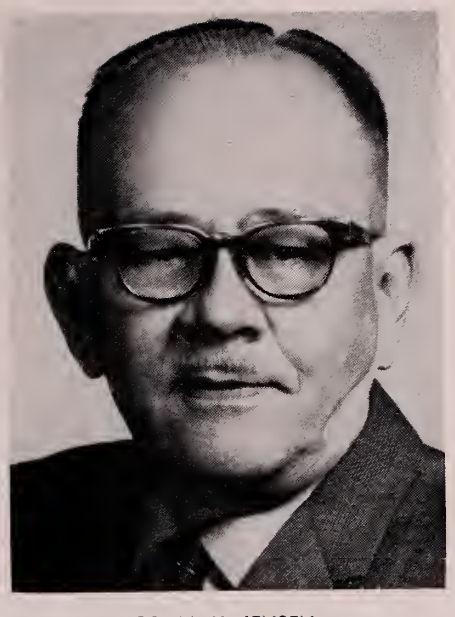
Prior to going to San Antonio, Dr. Jensen had been in private practice in Sweetwater from 1935 to 1940, and between 1927 and 1935, he worked in public health in Louisiana, Texas, Washington, D. C., Boston, and Kentucky. He had served as director of the public health unit for Madison and East Carroll Parishes in Louisiana and field director of the state health units and director of the State Bureau of Epidemiology in Kentucky.

Born in 1902 in San Antonio, his parents were Andrew and Dusine Steffensen Jensen. After receiving his preliminary schooling in San Antonio, he attended the University of Texas, Texas Agricultural and Mechanical University, and Baylor University from 1919 to 1921. He received his M. D. degree from the University of Texas Medical Branch in Galveston in 1925 and interned at Scott and White Memorial Hospital in Temple the following year. In 1931 he received a fellowship from the Rockefeller Foundation to study at Harvard, from which he received a master's degree in public health.

He married Hattie Rose Proctor of Merkel in 1926 in Sweetwater.

Dr. Jensen was a Baptist, Mason, and member of the Bexar County Medical Society, Texas Medical Association, American Medical Association, American Academy of General Practitioners, Delta Omega honorary public health society at Harvard, and Theta Kappa Psi medical fraternity at the University of Texas Medical Branch.

Survivors include his wife and daughter, Mrs. Arthur L. Walker of San Jose, Calif.; son, Martin H. Jensen, Jr., of San Antonio; brother, Dr. Andrew M. Jensen, and stepmother, Mrs. Andrew Jensen, both of San Antonio; and five grandchildren. A daughter, Shirley Louise Probst, died in 1959.



DR. M. H. JENSEN

DR. C. A. HOLSHOUSER

Dr. Charles A. Holshouser, physician and surgeon, died March 10 in San Antonio. Death was attributed to pulmonary complications following surgery for carcinoma of the mouth.

Dr. Holshouser was born in Rowan County, N. C., on June 19, 1903. He attended schools in Salisbury, N. C., the University of North Carolina, and Johns Hopkins University School of Medicine, from which he received his M.D. in 1928. He was a member of Pi Kappa Phi and Alpha Kappa Kappa fraternities. He served his internship at Fort Sam Houston Station Hospital (now Brooke General Hospital) in San Antonio in 1928 and 1929. He then entered practice in San Antonio. During World War II he reentered the U. S. Army Medical Corps serving as Chief of General Surgery with the 94th General Hospital near Bristol, England. He participated in the invasion of France in June, 1944, serving the wounded evacuated from Omaha Beach in Normandy. He received the Certificate of Merit and a battle star for his service to the invading troops.

He was a Fellow of the American College of Surgeons, and a member of the Bexar County Medical Society, the Texas Medical Association, American Medical Association, and the International Postgraduate Medical Assembly.

Membership in other organizations in-

DR. C. A. HOLSHOUSER

DR. R. N. KRAUS

cluded The Order of the Alamo, San Antonio German Club, Texas Cavaliers, San Antonio Country Club, San Antonio Rotary Club, and American Legion Post No. 10. He was a member of St. Mark's Episcopal Church.

Survivors are his wife, the former Ella Savage; two daughters, Joanne, a member of the 1963-1964 debutante coterie, and Louise, a student at Trinity University; two sons, Charles, junior ROTC Cadet Colonel at Thomas Jefferson High School, and John, an eighth-grade student. Also surviving are two sisters, Mrs. Harry L. Cunningham, Greer, S. C., and Mrs. George Fink, Salisbury, N. C.

DR. R. N. KRAUS

Col. Ralph N. Kraus, USAF, MC, who was assigned overseas as Chief, Department of Air Force Clinics, stationed at Wiesbaden, Germany, died suddenly of cardiac failure while acting as a consultant on temporary duty assignment in Athens, Greece, on Jan. 29.

The former Chief, Department of Clinical Medicine, School of Aerospace Medicine, Brooks Air Force Base, had been overseas since June, 1961. Colonel Kraus, 43, an otolaryngologist, was a member of Texas Medical Association through the Bexar County Medical Society and a military member of the American Medical Association.

He was born May 15, 1921, at Farley, Iowa, the son of Leo J. and Alvina (Schmitt) Kraus. He attended St. Joseph's School in Farley and received his bachelor of arts degree from Loras College in Dubuque, Iowa, in 1942. His M. D. degree was earned at the State University of Iowa College of Medicine, Iowa City, in 1945.

He interned at St. Mary's Hospital, Detroit, and served a residency at Letterman Army Hospital, San Francisco. He spent six weeks in postgraduate study at Lempert Institute of Otology in New York.

Colonel Kraus, a Senior Flight Surgeon, was commissioned in the Army in 1946.

He was certified by the American Board of Otolaryngology in 1951 and was a member of the American Academy of Ophthalmology and Otolaryngology, Society of Military Otolaryngologists, Society of Air Force Clinical Surgeons, Aerospace Medical Association, and Texas Air-Medics Association. He authored several medical publications.

His hobbies were golf and fishing. He was a Roman Catholic.

He was married to Elizabeth Ann Kennedy in Placid, Iowa, on May 22, 1943. She survives and lives in Dubuque, Iowa, as do three daughters, Judith Kraus, Jane Kraus, and Linda Kraus; a fourth daughter, Kay Kraus, lives in Paris, France; one son, Leo Kraus, Iowa City, Iowa; his mother, Mrs. Alvina Kraus, Farley; two brothers, James Kraus, Farley, and Bill Kraus, Dubuque; four sisters, Mrs. Kenneth Kemp, Dubuque, Mrs. John O'Neil, Marion, Iowa, Mrs. Carlton Westemeyer, Dyersville, Iowa, and Mrs. Lawrence Scherrman, Farley.

COMING MEETINGS

American Medical Association, Annual Meeting, San Francisco, Calif., June 21-25, 1964. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

MAY

American Association of Plastic Surgeons, Chicago May 13-16, 1964. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American College of Obstetricians and Gynecologists, Miami Beach, Fla., May 17-22, 1964. Mr. D. F. Richardson 79 W. Monroe, Chicago 3, Exec. Sec.

American Gynecological Society, Hot Springs, Va., May 25-27, 1964. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Medical Women's Association, Roanoke, Va., May 31-June 2, 1964. Dr. Jessie Brodie, 1790 Broadway, New York 19, Exec. Sec.

American Proctologic Society, Philadelphia, May 10-14, 1964. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

International College of Surgeons, Vienna, Austria, May 11-16, 1964. Dr. W. F. James, 1615 Lake Shore Dr., Chicago, Sec.

South Texas District, American College of Surgeons, Corpus Christi, May 15-16, 1964. Dr. Sanford Glanz 2425 Medical Center, Corpus Christi, Sec.

JUNE

American College of Chest Physicians, San Francisco, Calif., June 18-22, 1964. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American Dermatological Association, Hawaii, June 27-July 2, 1964. Dr. Wiley M. Sams, 808 Ingraham Bldg., Miami 32, Sec.

American Neurological Association, Atlantic City, June 15-17, 1964. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

Texas State Board of Medical Examiners, Fort Worth, June 22-24, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice. Mr. M. F. Cahal, Volker Blvd., at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Association for Thoracic Surgery. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Physicians. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Oct. 2-3, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Gastroenterological Association. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., New Orleans, La., Oct. 8-10, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Medical Society of United States & Mexico. Dr. C. Kalil, 200 East Monterey Way, Phoenix, Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwest Regional Cancer Conference, Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, San Antonio, April 29-May 2, 1965. Dr. W. S. Brumage, 2600 Westover Rd., Austin, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, San Antonio, April 29-May 2, 1965. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. John W. Chriss, 2436 Morgan, Corpus Christi, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, San Antonio, April 29-May 2, 1965. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. E. I. Bruce, Jr., 1014 Strand, Galveston, Sec.

Texas Ophthalmological Association, San Antonio, April 29-May 2, 1965. Dr. Harold Hunt, 150 Eighth St., S.E., Paris, Sec.

Texas Orthopaedic Association, San Antonio, April 29-May 2, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-May 2, 1965. Dr. J. P. Moran, 3154 Reid Dr., Corpus Christi, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. D. Hoehn, Doctor's Bldg., Fort Worth, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, San Antonio, April 29-May 2, 1965. Dr. L. Duflot, 173 San Marino, Galveston, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section, San Antonio, April 29-May 2, 1965. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc., San Antonio, April 29-May 2, 1965. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29-May 2, 1965. Dr. W. C. Sellman, Jr., 610 Medical Arts Bldg., Dallas, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Maria P. Hathaway, 1012 State Office Bldg., Austin, Exec. Sec.

Texas Surgical Society, San Antonio, Oct. 4-6, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. B. C. Bates, 1108 Nueces, Austin, Sec.

United States-Mexico Border Public Health Association, Monterrey, Mexico. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society. Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals. Mr. R. L. Heath, Malone-Hogan Clinic, Big Spring, Sec.

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Prepaid Health Insurance With Service Benefits

PRESSURES ARE being exerted by certain groups to extend the principle of voluntary prepaid health insurance with service benefits and to insure their success to all states and to all income levels. Too, there is evidence of increasing desire by individuals to obtain fully paid medical care through a method of prepayment or budgeting.

In January, 1963, General Motors employees and Ford Motor Company employees of Texas became insured under a new contract with Metropolitan Life Insurance Company and Health Service, Inc., respectively. In negotiation of these contracts a new and far-reaching principle was demanded and obtained; this principle called for the fee for professional service to be paid to the physician if his charges equaled or were less than the fee schedule but to the patient if the fee exceeded the schedule. This, in an area where service contracts had been denied, appeared to be a method of obtaining a service contract.

In December, 1963, the method of payment of Michigan Medical Service was altered by a court judgment which grew out of the legal action initiated by the Insurance Commission of the State of Michigan. The alteration in payment method was as follows: "If the physician charges a subscriber who is eligible for service benefits an amount which is in excess of Blue Shield's scheduled fee, Blue Shield will make its payment to the subscriber rather than to the physician and under such circumstances Blue Shield will not honor the subscribers assignment to the physician."

In Michigan, a service contract is in operation with Michigan Medical Service, but more than 40 per cent of those so insured are securing the services of nonparticipating physicians so that it appears that this new principle of payment is an effort to insure the operation of a service contract.

In Pennsylvania, the steel industry has service

benefits through Medical Service Association of Pennsylvania, whereas steel groups in some other areas, including Texas, do not. Pressure is being exerted on Medical Service Association of Pennsylvania to force indemnity plans in indemnity areas to arrange to provide service benefits based upon local fee schedules. In placing this pressure there is intimation that if such indemnity programs do not cooperate they will be dropped as well as those who have agreed to provide service contracts.

In April, 1964, the director of the U. S. Civil Service Commission Bureau of Retirement and Insurance requested that Blue Shield raise income levels applicable to the Federal Employees Program Service Plans with Blue Shield from the present $6,000 to $7,500. Insured under various Blue Shield plans are 3,600,000 federal subscribers and their dependents. With such an influential number it is no wonder that Blue Shield executives have endorsed the director's request.

The Texas Medical Association, along with 17 other state medical associations, has not seen fit to participate in full payment or service benefits for professional services. Rather, indemnity plans are favored feeling that the charge for professional service is a matter between the physician and his patient and that usual and customary charges for the area will prevail.

The Texas Medical Association through the Hospitals-Insurance-Physicians Joint Advisory Committee of Texas and liaison with Blue Shield is cooperating with the Texas insurance industry, Texas Hospital Association, and Blue Shield of Texas in matters of voluntary health insurance. Much has been accomplished through education and cooperation between these groups to insure the success of the indemnity principle.

The continued success of the indemnity principle in health insurance in Texas is dependent upon three factors:

1. Provision of realistic coverage in terms of fees for professional service schedules by Blue Shield and commercial insurance companies.

2. Willingness of the Texas physician in a high percentage of cases to accept these payments, for full payment or nearly full payment, dependent upon family income and responsibility.

3. Willingness of the Texas physician to assume his full responsibility as the director of the spending of the health dollar, since it is he who sends the patient to the hospital, orders the studies while there, requests the consultations, determines the length of hospital stay, and finally, renders a fee for professional services.

—CHARLES D. BUSSEY, M.D., Dallas,
Chairman, Council on Medical
Service and Insurance,
Texas Medical Association.

Extradietary Fluoride Supplementation

CONSIDERABLE information has been channeled to dentists and physicians regarding the various dietary routes for the administration of fluoride compounds. There have also been increasing numbers of commercial preparations whose promotional literature presents a highly confusing picture regarding the possible beneficial results of the dietary administration route.

Both dentist and physician are concerned with prevention of any disease involving any organ system during childhood. Dental caries is the most common chronic and progressive disease prevailing throughout childhood and adolescence. Since there are effective preventive measures available to eliminate a substantial percentage of the cases, and since 50 per cent of all two year olds have dental decay, it is advisable to implement at an early age all preventive procedures known to be effective.

If dental caries progresses without necessary attention, it will lead to pain, inability to masticate properly, gingivitis, osteomyelitis of the jaws, and unnecessary loss of teeth. Neglected caries and/or premature loss of the primary or permanent teeth is the major cause of malocclusion or "crooked teeth." For these reasons early dental treatment, especially dental treatment of a preventive nature, is most urgently recommended.

The exact mechanism of how fluoride systemically ingested prevents caries is not fully understood; however, it is thought that fluoride makes the tooth less soluble to the acid.

The most effective preventive procedure that can be practiced daily without conscious

effort is the consumption of communal fluoridated water. However, because only 60 per cent of the United States' population lives in areas supplied by a public water system, still to be considered are the remaining 40 per cent who use private wells.

A preventive measure that has been suggested is extradietary fluoride supplementation. Hypothetical projections regarding the benefits of this method of fluoride administration are available, but there have been no long-term studies reported. Furthermore, studies reported have been limited in scope, or were performed on relatively small groups of individuals for short time periods. In March, 1962, D. K. Hennon and J. C. Muhler released data which revealed that no clinical studies in the United States were available to illustrate the effectiveness in children ingesting fluoride supplement during the period of tooth calcification (birth to nine years) and for which adequate controls were available. Until such studies are available and corroborated, this method of administering fluoride must be considered still in the experimental stage. Similarly, since there are no differences in the caries prevalence rate for children born from mothers living in optimal fluoride areas as compared to similar children born from mothers living in fluoride deficient areas, one may severely question the effect of placental transfer of fluorides.

Muhler and Hennon also noted that one must be extremely cautious in anticipating results to be obtained from extradietary fluoride administration when comparing such administration to a fluoridated public water supply since the two methods are so drastically different. Even though it is possible to ingest the same total amount of fluoride each day from either source, the duration of blood availability and the peaking of fluoride ions in the blood are undoubtedly different. For example, in a quart of drinking water fluoridated to 1.0 mg. per Liter, a child would consume 1 mg. of the fluoride ion in small doses over a long period. If 1 mg. of fluoride were ingested in a single dose it would be rapidly eliminated via the kidneys with only a transitory amount available to the developing teeth.

Consumption of fluoride from a fluoridated water source supplies fluoride to the body during various periods of the day and maintains a more constant blood level. Also, it increases the food fluoride content since the meals are prepared from fluoridated water. Studies on both animals and humans have shown that the fluoride tablet taken once daily does not duplicate the blood picture of a person drinking fluoridated water, and as a result may not duplicate the decay preventive effect in the teeth.

The acceptable range of fluoride ions present in drinking water is between 0.7 ppm. and 1.5 ppm. dependent upon climatic conditions influencing water consumption. If the fluoride level is below the minimal figure, the Council on Dental Therapeutics of the American Dental Association reports that the daily addition of sodium fluoride solution to the drinking water *may* be desirable for children under nine years of age *where community fluoride programs are not feasible.* However, such administration must take into consideration the child's normal fluoride intake and also be consistent and continuous from birth to nine years of age (or during the tooth formative years) for substantial benefits. Because of the obvious difficulties of its appropriate control, it is recommended that this material be prescribed only under professional supervision.

There has also been a recent trend to combine vitamins and sodium fluoride in fixed combinations for infants and very young children. These products are not accepted by the Council as rational combinations because of the increased difficulties involved in the proper dosage determinations, and the Council concluded that fluoride supplements combined with vitamins does not represent a logical type of product for prescription by the dentist.

The effect of a fluoride-multivitamin preparation on tooth decay or on the appearance of enamel has not been investigated in children. However, in several other species, including the monkey, there have been indications that vitamins A, C, and D influence fluoride metabolism. Thus the physician who prescribes vitamin preparations containing fluoride cannot be assured that the fluoride will be effective nor can he predict the effect on caries or enamel appearance (mottling). The problem of controlling the number of vitamin capsules ingested per day also becomes more important when fluoride is involved since, unlike the vitamins, a factor of two or three times more fluoride may materially alter the enamel appearance. Further, any fixed combinations of fluoride with other nutrients increase the difficulty of adjust-

ing the prescribed fluoride to allow for fluoride levels already in the child's drinking water, since alterations of the dose will also alter the vitamin intake.

Preparations composed of calcium fluoride or bone meal are totally unacceptable to the Council because it is impossible to provide any accurately controlled level of fluoride ion in the patient. The fluoride in these preparations is practically insoluble.

In summary, it should be pointed out that the effect of supplemental fluoride in combination with other compounds on either caries or enamel mottling cannot be predicted. Additionally, strong motivation on behalf of both parents is necessary to properly dispense this drug over a long period of time as well as to prevent excessive consumption. Controlling the patient's blood level is problematic, and the fact that a single dose is rapidly excreted suggests that the benefits would not be the same as small repeated doses in the drinking water. In light of these facts, plus the lack of sufficient clinical evidence regarding the effectiveness of the preparations and products presently available on the market, these preparations cannot be considered a satisfactory substitute for either the consumption of communal fluoridated water or the application of a concentrated topical fluoride to the teeth in the dental office.

—Richard E. Jennings, D.D.S., M.S.D.,
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Houston.

Good Writing Is Boon To Harassed Reader

THE INDIVIDUAL who tries to read even a small percentage of the millions of words which cross the desks of most professional people today understands the great problem of screening unwanted material.

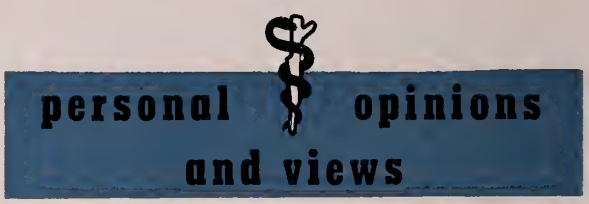
Communicators in all fields are diligently working on ways to woo readers, to improve content, and to help readers speedily digest and retain material.

The editors of the *Texas State Journal of Medicine* know that their readers are among the most harassed, that they must be kept informed in their field, and that they must be selective. A constant effort is being made to maintain valuable content, compactly edited, so that a reader's time is not wasted.

This month in New York City, both the *Journal* and *TMA Action*, the Association's news bulletin, were given awards by the International Council of Industrial Editors for keeping on the road toward these goals.

Commented one of the judges about the *Journal*, "Normally in journals of this type, the writing is stilted, dull, and complicated. . . . HOWEVER, I see no objection, particularly to your articles. You are either doing an excellent job in some cases, or you have your contributors well educated to keep paragraphs fairly short and sentences short."

We prefer to believe that we have our contributors well educated, and we look to our readers for further helpful criticisms. A scientific look at reading may keep important medical contributions from being obscured by poor writing or display.



Physicians, Pharmacists Must Solve Problems

IN GENERAL, the overall relationship between pharmacy and medicine is good. Only a small minority have any real problems, but this endangers our relationship and spoils the public image of both professions. We should recognize that these problems are not of the same magnitude in all states nor in all areas of a given state.

Physician ownership of drug stores is probably the main stumbling block of pharmacists in their relationship to medicine. There are 93 pharmacies in Texas that are

Dr. Robert Mayo Tenery originally presented this discussion before the AMA Congress on Medicine and Pharmacy in Chicago on March 12.

doctor-owned (or partially owned); this is less than 10 per cent of the pharmacies in our state, and most are located in three or four specific areas. Many of the physicians that participate in this practice do so on the basis that it is a sound business investment, and most of them make no attempt to direct their prescriptions to the store in which they have an interest—*but some do.*

More clinics and small hospitals are built every year, and their owners find that a pharmacy or a drug room is desirable because of convenience to both doctors and patients, completeness in the public eye, the requirements for accreditation of hospitals, and the need to increase the income of the facility. A pharmacy may be selected in preference to a drug room because of lack of personnel to care for the latter, because a drug room can be a losing proposition since prescriptions cannot be filled, and because more profitable items can be carried in a pharmacy.

The AMA House of Delegates has taken the position that physician-ownership of pharmacies is not unethical unless there is exploitation of patients; however, this is very difficult to prove when charges are filed in a county medical society as it is the prerogative of a doctor to use those medications that he believes are indicated for his patients, and testimony presented to a county medical society must be such that it will stand up if a law suit follows.

Physician ownership in a repackaging company or in a stock company which he can control should no longer be a problem since the House of Delegates of the AMA approved the opinion of the Judicial Council that such ownership is unethical; however, we should be alert to the fact that there will always be promoters both in and out of our two professions.

Not all of the stumbling blocks originate from doctors. We do still hear complaints about over-the-counter prescribing by pharmacists; and drug substitution is still practiced, especially in small stores and cut-rate stores. When a physician prescribes a brand-name product, he does so because he has confidence in the integrity of that particular manufacturer. Authorities are not always interested in protecting these brand names.

The pharmaceutical associations do not have sufficient power to discipline their members, otherwise physicians who own drug stores would not be able to employ licensed pharmacists to work for them. I feel certain that this lack of discipline arises because an appreciable number of druggists do not belong to organized pharmacy and because disciplinary action has little effect upon the business of its members.

The publicity and investigations concerning these interprofessional problems have already sparked legislation in some states, and more rigid federal regulation may be forthcoming. Our Board of Councilors, the judicial body of the Texas Medical Association, is strongly opposed to this approach which would result in more restrictions and red tape and would continue to make our problems political football which spoils the public image of both professions. We should be able to solve our problems within our own organizations by ethical principles.

The Board of Councilors of the Texas Medical Association is composed of one physician from each of our 15 Councilor districts, and several years ago the Texas Pharmaceutical Association reorganized to conform with this same pattern. Thus, within the districts the councilors of the two organizations can work together to solve ethical problems involving physicians and pharmacists.

Once a year, and more often if necessary, the two boards meet together to discuss mutual problems. Out of this liaison was born an Interprofessional Code which was approved by the Houses of Delegates of both associations in 1962. This code recognizes the need for cooperation between our professions and forbids many practices that are harmful to our relations.

The two boards have encouraged joint meetings at the local level to discuss such problems as ownership, rebates, gifts, designated prescription pads, substitutions, over-the-counter prescribing, and poor or illegal prescribing habits. One problem that must be solved in local meetings is that which arises when a physician changes medication for a specific condition after each detail man sells him on the superior value of his company's product. This frequently means that the local druggists may stock a particular item, fill one prescription for the item, and be left with the remaining stock to deteriorate on his shelves.

We have recognized that many physicians need education about the problems and expenses of ethical manufacturers and pharmacists. Key officials of the Texas Pharma-

ceutical Association are being invited to write guest editorials for the *Texas State Journal of Medicine.* Outstanding representatives of the pharmaceutical industry have been asked to speak at state and county medical society meetings. Physicians have been asked to encourage their local druggists to join state pharmaceutical organizations. Our two associations have worked together on legislation that affects the two professions.

Three years ago the Texas Health Council was formed to include the top officers and executive secretaries of the medical, pharmaceutical, dental, hospital, and nursing home associations plus representatives of Blue Cross-Blue Shield and the Texas Life Convention. This group meets two or three times a year to discuss matters that relate to the health care of the people of our state. No binding policies are established, but the Council serves to seal a closer relationship between the members of the health team and acts as a sounding board wherein the opinions of each profession can be presented for discussion. The pharmacists are an integral part of this council.

Our professions must work toward the goal of 100 per cent membership in pharmaceutical associations in order to establish disciplinary measures which would be comparable to the self-imposed discipline of medicine; then pharmacists would be able to prevent members from working in physician-owned drug stores. Our organizations should seek the means of protecting the brand-names of those pharmaceutical manufacturers that have contributed so much time and money toward the advancement of medical science.

In June, 1963, the AMA House of Delegates reaffirmed that "Drugs, remedies, and appliances may be supplied by the physician provided it is in the best interests of the patient"; however, the Judicial Council issued the following interpretation of this rule:

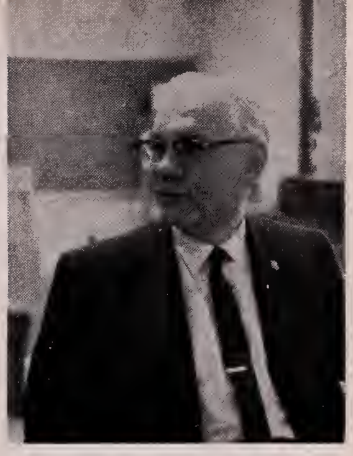
> Under this language the doctor is permitted to exercise his own best judgment when caring for his patients. It is known that there will be situations when it is necessary or desirable for a physician to dispense or supply what he has prescribed. The Principles permit this to be done. On the other hand, this broad language provides a means by which a component medical society can inquire into the facts of a particular practice. The profession thus can act to prevent abuse of discretion and protect patients from exploitation. In essence this language means that a physician in the exercise of sound discretion may dispense "in the best interest of his patient"; *it does not authorize him to dispense solely for his convenience or for the purpose of supplementing his income.*

I would suggest that this principle would be easier to enforce if all state medical association bylaws were amended to read: "A physician who participates in the ownership of a pharmacy must submit to his component county medical society acceptable evidence that such ownership is in the best interest of his patients."

Those physicians and clinics who could not meet this requirement should voluntarily lease or sell their drug facilities to pharmacists. If leased, the rent must not in any way be determined by the amount of income. If sold, the physicians would of course encounter a capital gains or loss tax, and the amount of the sale must not depend upon the future business of the facility. In small communities the doctor can dispense from his office the drugs needed for his own patients, and other items can be carried in grocery stores. We should be aware of the fact that a few physicians will avoid discipline by transferring ownership of pharmacies to members of their family as some have already done with repackaging and small stock companies — this practice, of course, is a detriment to our profession.

The majority of physicians and pharmacists are ethical and practice a friendly cooperative relationship. The two professions are dedicated to the belief that all Americans should continue to have the best health care of any nation in the world. Unfortunately, a few members of each profession are more concerned with self than service, and their actions tend to build a wall between us and to spoil the public image of both pharmacists and doctors. Honorable men need no code of conduct—they inherently follow the Golden Rule.

—R. MAYO TENERY, M.D.,
Waxahachie,
Past President,
Texas Medical Association.



MISSION TO WASHINGTON

A delegation from the allied health professions in Texas traveled to Washington in late May for a two-day visit with elected Congressional representatives. This group, consisting of members from the Texas Medical, Dental, Pharmaceutical, and Hospital Associations, profited from personal interviews with Senators John Tower and Ralph W. Yarborough and with Congressmen Clark W. Thompson and Bruce Alger, and then divided into smaller numbers to visit Congressmen from their respective districts. The delegation also hosted a reception and dinner honoring the Texas Senators, Congressmen, and their wives.

Our visit enabled us to become better acquainted with our elected representatives and to discuss with them our mutual problems. We believe this exchange of views has been vastly worthwhile.

We returned home, however, filled with a deep sense of alarm. The present outlook is frightening. Not only is the private practice of medicine in danger of being destroyed, but also the whole American system of private enterprise is in great peril.

Those members of Congress who favor federal government administered by bureaucratic decrees are determined to increase the public welfare on every front this year. It is likely that social security cash benefits will be increased 5 to 6 per cent across the board. Additional medical benefits will be given through the passage of King-Anderson type legislation or a much more preferred route through the Kerr-Mills amendment. On every front the government is advocating greater spending for such projects as its so-called "war on poverty," mass transportation, area redevelopment and countless other items. These costly programs are advocated at a time when we are told our economy is at an all-time high. Taxes have been lowered, but the limit of the public debt is to be increased to 324 billion dollars.

The time is at hand when we Americans must make a determined stand if we are to preserve our way of life. We must accept our personal responsibilities and see that our local, county, and state governments maintain their separate responsibilities. If we do not, the federal bureaucratic agencies will assuredly assume these duties for us.

The pressures on our elected representatives are tremendous. Unless we take a determined and positive stand on the many issues facing them and keep them well informed on our own views, we shall have only ourselves to blame should we lose our precious American heritage of private enterprise.

Mal E. Johnson

President
Texas Medical Association

Angiosarcoma of the Breast

CASE REPORTS

ANGIOSARCOMA is a highly malignant tumor of the endothelial lining of blood vessels. Its rare occurrence in the breast carries a uniformly fatal prognosis. A review of the literature in 1960[1] revealed only 22 cases reported and all of the patients except one, a short term follow-up, were dead within five years. The average reported survival time is 1.5 years[1] and the longest known survivor lived about five years before succumbing to the disease. Angiosarcoma represents 0.2 per cent of all malignant breast tumors,[10, 12] and is found, usually in females, between the ages of 20 and 40 years.

The clinical appearance of angiosarcoma of the breast is fairly constant. The patient usually is seen with a rather bulky, firm, breast lesion with indefinite margins. Fixation to the skin or deep structures is unusual, and nipple discharge or axillary adenopathy is rare. The duration of the lesion before medical assistance is sought is usually slightly less than a year. Its clinical course is characterized by episodes of sudden enlargement and pain, probably due to hemorrhage within the tumor. The most characteristic clinical finding is a bluish discoloration of the skin overlying the lesion. In differentiating this lesion from carcinoma, the most helpful features are probably the color change, tenderness, indefinite margins, and lack of stony hardness. Care must be taken not to mistake this tumor for an inflammatory lesion. Some authors correlate trauma with the onset of angiosarcoma, and it has been postulated that granulation tissue capillaries in a traumatized area could be the precursor.[9,13] The theory of malignant transformation of benign angiomas was proposed by McCarthy and Pack.[10]

No successful form of management has been described. The variations of conservative or radical surgery with or without pre- or postoperative irradiation have failed.[1,9,10] Angiosarcoma metastasizes via the blood stream and distant metastases have a predilection for the lungs, bone, brain, skin, and the abdominal viscera.[10] Local recurrences are common.[12] Neither the primary lesion nor distant metastases show any significant sensitivity to irradiation, although McCarthy and Pack report[10] that slight palliation may be obtained by such therapy in advanced cases.

Hemoptysis as a result of metastasis to the lungs is sometimes the first sign of recurrence of the disease and often appears before x-ray evidence of a metastatic pulmonary lesion is found. The metastatic skin lesions ulcerate early and bleed easily but the usual cause of death is massive hemorrhage into a chest cavity or the abdominal cavity.

Case Report

A 17-year-old colored female, who complained of a painful and enlarging breast mass for 13 months, was seen at the Hermann Hospital surgical clinic. The mass was small, nontender and was located in the area of the axillary tail of the left breast. When first observed by the patient, six months prior to her admission, the mass rapidly increased in size accompanied by a constant, dull, aching pain. About this same time, she noticed a dark discoloration of the skin overlying the mass and the prominence of superficial veins in that area. There was no history of nipple discharge or bleeding and no specific history of trauma to that breast. However, she was a basketball player and had sustained numerous bumps and bruises during the past few years.

Further questioning revealed that she was single, nulliparous, had normal menses, and no symptoms other than those described. There was no history of hemoptysis or other type of blood loss, no weight loss, and no lethargy or pain in other areas. She had had no previous significant illnesses, no surgical procedures, and no other masses. There was no family history of malignancy.

Physical examination on admission revealed her to be a 166-lb., 5′7″, young colored female who was ambulatory and in no distress. The oral temperature was 98.6 F., the blood pressure 130/90 in each arm, and the pulse rate 80. The only abnormal physical findings were in the left breast where a soft, slightly tender, nondiscrete, 5 by 5 cm. mass was palpable in the axillary tail. The skin over the area was a deep bluish-black in color and was not fixed to the lesion. There were several moderately dilated veins over the lateral part of the breast. A few small, nondiscrete, movable lymph

From the Department of Surgery at Hermann Hospital, Houston, comes this presentation of a rare entity. The patient was asymptomatic at her 2.5 year follow-up, although previously reported survival-times average 1.5 years.

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JOHN E. COGAN, M.D.

GEORGE W. WALDRON, M.D.*

nodes were palpated in the anterior part of the left axilla. Other regional nodes were not remarkable.

During the course of lung auscultation, a bruit was heard and proved to be most prominent directly over the mass. The initial impressions of several examiners included arteriovenous fistula, hematoma, hemangioma, enlarged axillary breast tail, and fibrocystic breast disease.

Laboratory examinations revealed a normal hemoglobin and white blood cell count. The urinalysis and chest x-ray were normal. An x-ray mammography showed an ill-defined soft tissue mass in the tail of the breast with irregular margins and no calcification. Because of the possibility of an arteriovenous fistula, a subclavian arteriogram was performed by insertion of a catheter into the proximal subclavian artery via the abdominal aorta. Using a Sanchez-Perez apparatus, multiple x-ray exposures were made to demonstrate both the filling and drainage of the regional vascular system. The vascular tumor was well demonstrated and seemed to encompass an area measuring 9 by 13 cm. There was moderate dilatation of the regional veins.

The patient was taken to the operating room where an incisional biopsy was done. The pathologist's examination of the frozen section revealed angiosarcoma. The biopsy wound was tightly closed and the area prepared for a more extensive procedure. A block dissection was carried out including the upper portion of the breast, overlying skin, pectoralis major and minor muscles, axillary contents, perivascular tissue along the subclavian and axillary vessels and a portion of the latissimus dorsi and serratus anterior muscles. The planes of dissection at no time approached the tumor. However, extreme dilatation of the usually small regional veins was encountered. The wound was irrigated with a 1 per cent solution of Chlorpactin XCB and closed over suction drainage catheters.

The postoperative course was uneventful and the patient was discharged on the tenth postoperative day.

On histologic examination, the margins of resection appeared adequate. The tumor invaded adjacent breast tissue and muscles. The most prominent miscroscopic features were multiple endothelial lined channels containing blood cells and showing considerable variation in thickness of the vessel walls. Other areas were composed mostly of large spindle or polygonal cells with eosinophilic cytoplasms and without vessel formation. A rare mitotic figure was seen. Seventeen lymph nodes were recovered from the specimen and metastatic tumor was found in four.

The patient was asymptomatic at her two and one-half year follow-up. There was no evidence of recurrence of the tumor on physical examination, and x-rays of the chest and bones failed to show any evidence of metastases. She is being followed at six month intervals in the Hermann Tumor Clinic and recently gave birth to her first child.

Discussion

The angiogram illustrates the large vascular component of these tumors. Although microscopically the endothelial cell prolifera-

*Deceased Feb. 15, 1964.

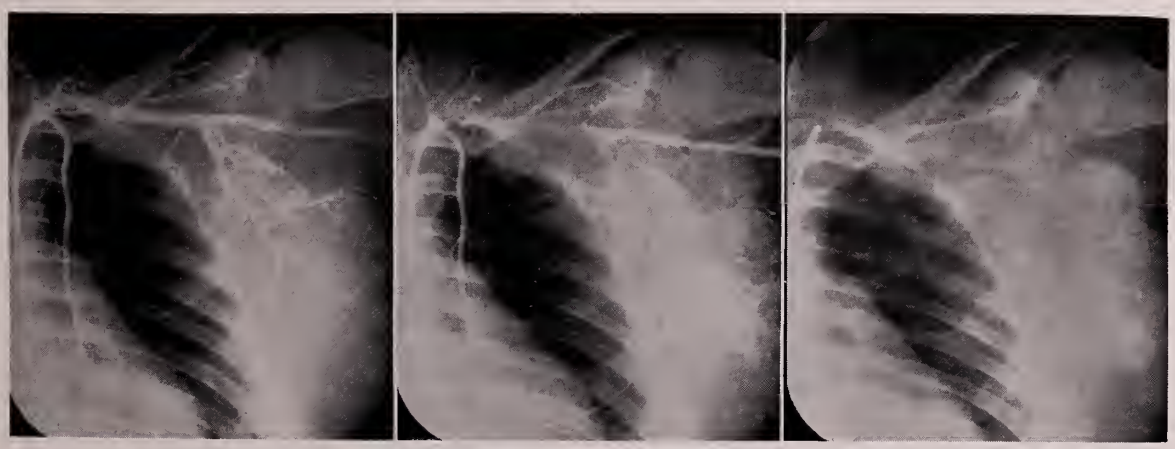

Fig. 1. Arteriograms showing, left, subclavian and axillary arteries with their branches, the breast shadow, and increased arterial supply in the area of the tumor; center, at mid phase the numerous small vascular channels of the tumor; and right, at late phase, the best demonstration of the boundaries of the tumor.

Fig. 2. A low power photomicrograph contrasting highly cellular areas of the tumor with the more vascular component.

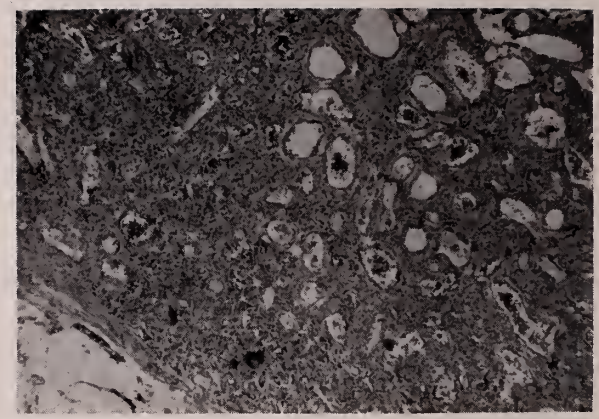

Fig. 3. Photomicrograph showing almost complete replacement of a lymph node by the vascular tumor. The node capsule is seen in the lower left of the photograph.

tion is the outstanding feature, angiography in this case was useful in differentiating the tumor from a benign arteriovenous fistula, and of some benefit in outlining the tumor margin. The operative procedure employed in this case was primarily a block dissection of the tumor-bearing area with the addition of a regional node dissection. Also an effort was made to ligate efferent vessels to the axillary and subclavian veins, prior to manipulation of the tumor mass, in order to avoid tumor emboli.

Stout, McClanahan and Hogg[11,14] have commented on the benign microscopic appearance of this tumor, and some cases in the review by Barber, and others,[1] were first recognized as malignant during a review of the microscopic sections at the time of appearance of metastatic lesions.

Histologically, angiosarcoma invades the adjacent structures, breast, and pectoral muscles, with little distortion of the normal architecture of these structures. One may find sheets of endothelial cells without a vascular lumen or a vessel lined by slightly swollen, but not atypical endothelial cells which in places are piled up and produce thick walls or papillary infoldings. Stout[14] emphasizes the importance of connective tissue staining to differentiate angiosarcoma from other tumors such as hemangiopericytoma, chorio-epithelioma and metastatic renal tumors.

Surgical excision and regional lymph node dissection is recommended for the treatment of angiosarcoma of the breast, with reservation of radiation therapy for palliation of symptomatic metastases. As with most malignant neoplasms, early detection and prompt therapy seem to offer the best chance for long-term survival. Gentle examination and surgical manipulation is advisable when such a vascular tumor is present.

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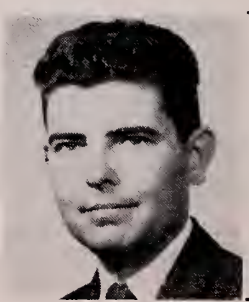

Dr. Scheid is a Houston surgeon.

Multiple Myeloma

With Pyroglobulinemia and Vacuolated Inclusion Cells

H. B. EISENSTADT, M.D.

A case of multiple myeloma is described in which numerous large reticulum cells filled with globular inclusions were found. Similar globules were seen outside the cells in the tissues at autopsy. These globules may be related to the pyroglobulins present in the patient's serum.

CASE REPORTS

MULTIPLE MYELOMA presents a fascinating clinical picture because of the great variation of its symptomatology and laboratory findings. It has been stated, "each case of myeloma is different."[7] The clinical spectrum includes isolated small solitary lesions,[8] massive involvement of an entire organ or a region, and generalized dissemination over the body with or without plasma cell leukemia. The great variety of bone lesions,[1] renal abnormalities, and hematologic and metabolic changes[6] observed in this disease has been repeatedly described. These entities may present themselves in manifold combinations. However, the large number of clinical manifestations cannot match the innumerable protein patterns that may be detected in urine and serum. Putnam found "that no two patients appeared to produce the same abnormal serum or urinary protein."[7] Osserman[5] stated that in each case, however, the physiochemical and immunochemical properties of the abnormal substances remain unchanged throughout the patient's life. His statement has been challenged by Parson[6] who stated that even the protein pattern of a single individual may undergo variations from time to time. Most abnormalities in the urine appear as Bence Jones protein, that is, they are precipitated by heating the urine to 50 C. and are redissolved by further heating above 60 C. However, Bence Jones protein is no uniform substance but a group of chemical bodies, and occasionally pathologic material is found in the urine with completely different physiochemical characteristics. The abnormal substances in the myeloma serum are generally described as paraproteins; they can be separated by many laboratory procedures. However, three groups can be identified by simple means within the reach of any physician:

1. Cryoglobulins precipitate at 4 C., or icebox temperature.
2. Pyroglobulins precipitate between 40 and 60 C., which is during inactivation of the serum required for most syphilis tests.
3. Macroglobulins precipitate from serum dropped into a test tube filled with water. Their exact identification requires ultracentrifugation.

The pyroglobulins are the rarest of these three paraproteins. They were discovered in 1899 by Ellinger[2] and received their name in 1953 by Martin and Mathieson.[3] Originally, they were thought to represent Bence Jones protein in the serum. However, they are entirely different substances. They precipitate also at 50 C. but cannot be redissolved by further heating. In addition, they show a number of other chemical, physical, and immunologic properties that differ from the Bence Jones bodies. Until 1959, only about 35 cases of myeloma with pyroglobulins were reported in the literature. Little is known about their origin, chemical composition, and relationship to pathologic cells and tissues. A case report of this disease in which pyroglobulins were associated with an unusual type of myeloma cells containing globular cytoplasmic inclusions is presented. This combination apparently has not previously been described in the literature.

Case Report

A 55-year-old white woman was seen in June, 1962, by an orthopedic surgeon* because of lumbar pain of several months' duration. Since she did not respond to simple medication, she was hospitalized and a roentgenographic examination of the spine was undertaken. It did not show any osseous abnormality in the region of her discomfort, but a collapse of the ninth dorsal vertebra was demonstrated. Laboratory examinations revealed a sedimentation rate of 61 mm. per hour (Wintrobe), and a total protein of 7 gm. per 100 ml. consisting of 2.9 gm. of albumin and 4.1 gm. of globulin. There was a mild normochromic anemia with a hemoglobin of 9.5 gm. per 100 ml., and a hematocrit of 30 per cent. The white cell count was 9,600 with 28 lymphocytes, 4 monocytes, 8 stabs, and 60 polymorphonuclear leukocytes. Various other laboratory tests, including urinalyses, were normal. Because of the pathologic findings the patient was referred to the author with a suspicion of myeloma.

Physical examination revealed a mildly anemic patient in fair nutritional state. However, a moderate weight loss could be noted as the patient had been obese before onset of her illness. Abnormal findings were limited to the spine which was painful on movement, jarring, and compression. For this reason the patient kept her trunk immovable and avoided unnecessary motions. A complete bone survey including the skull failed to show an additional abnormality. Careful study of various internal organs was performed in the search for a primary carcinoma that could be responsible for metastatic lesions; however, the study revealed nothing. Among additional laboratory tests, the following appeared significant: C-reactive protein was strongly positive. Electrophoresis showed albumin 35.8 per cent, α_1-globulin, 5.6 per cent; α_2-globulin, 3.8 per cent; β-globulin, 5.6 per cent; and γ-globulin 49 per cent. The electrophoresis failed to reveal a narrow single peak characteristic of myeloma but instead demonstrated a broad band in the gamma section.

During the following weeks several bone marrow specimens were taken, at first from the sternum and later from the iliac crest. They revealed the same unusual picture: innumerable mononuclear and multinuclear cells containing globular inclusion bodies (Fig. 1) were sprinkled among the normal marrow components. Some of these contained one large or two to five smaller globes whereas others had numerous fine round particles producing a honeycomb appearance. The pathologist† examining the marrow specimen was unable to arrive at a definite diagnosis. Therefore, the slides were sent to Dr. William Levin, Chief of Hematology of the University of Texas Medical Branch, Galveston, for study. He reported as follows:

"The myeloid elements appear rather toxic. There is some mild stimulation of the erythroid elements. The most striking abnormality is, however, the presence of reticulum cells, some of which are multinucleated, most of them containing globular inclusions. This suggests some form of reticuloendotheliosis. The diagnosis of myeloma is certainly not confirmed by these bone marrow specimens, but it is in no way completely excluded."

A serum sample was likewise submitted to Dr. Levin for evaluation of the dysproteinemia; he confirmed the abnormal electrophoretic pattern; the increased γ-globulin had all the characteristics of a

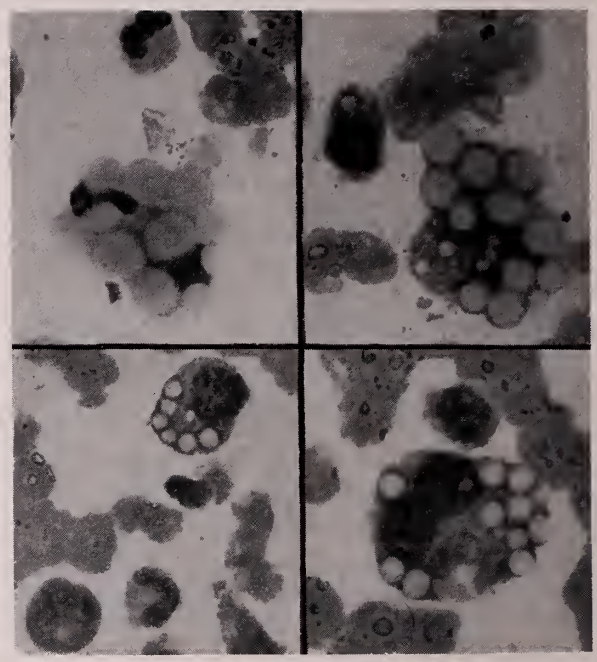

Fig. 1. Vacuolated inclusion cells.

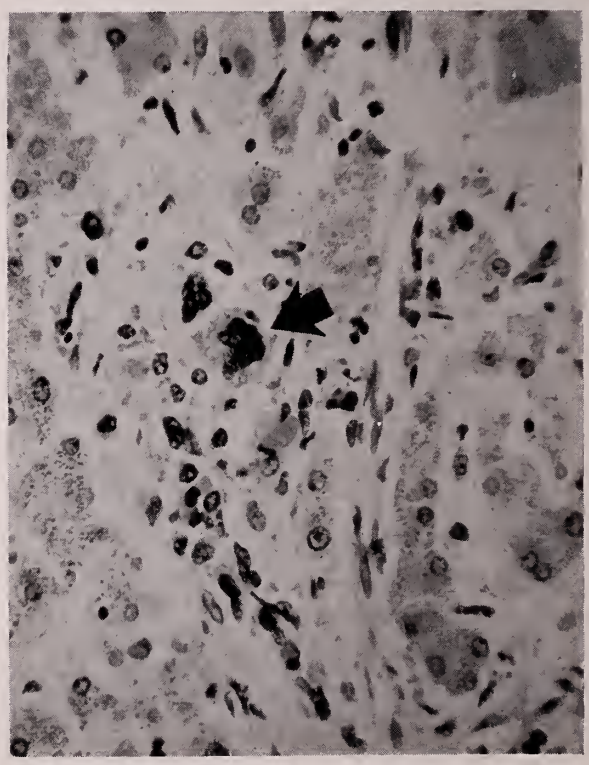

Fig. 2. Inclusion cells in liver section.

*Dr. Martin Haig, Port Arthur.

†Dr. Ellen Furey, Park Place Hospital, Port Arthur.

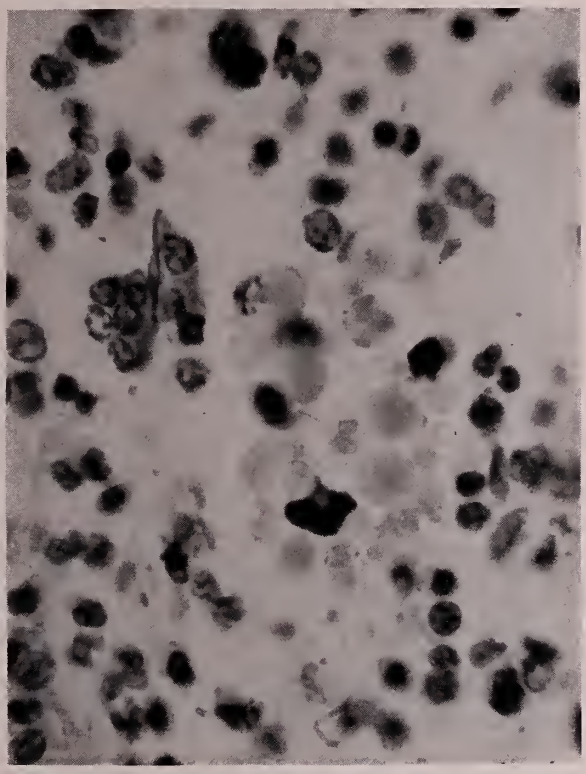

Fig. 3. Globular masses outside of cell.

pyroglobulin. The diagnosis of myeloma was now favored and appropriate therapeutic measures were undertaken. Unfortunately, neither urethan nor cyclophosphamide nor phenylalanine mustard had any effect on the course of the illness, nor did large doses of x-ray alleviate the excruciating bone pains and the general malaise and discomfort. The patient was degenerating rapidly and could not obtain the slightest relief until complete transection of the cord by a compression fracture produced anesthesia from the mid-dorsal region. Repetition of bone roentgenograms showed progressive osteolysis and collapse of additional vertebras but no bone involvement elsewhere. Only the portion of the ribs adjacent to the spine appeared to participate in the process of destruction. Toward the end an extreme anemia appeared in spite of almost daily blood transfusions. The patient died suddenly of aspiration pneumonia. At autopsy (head was excluded) the thoracic and lumbar vertebras and the spinal cord were completely replaced by a grayish fusiform mass enclosed by the periosteum. Specimens taken from other portions of the osseous system were normal. Liver and spleen showed little macroscopic changes but the tissue section revealed the same cells as found in the bone marrow specimens (Fig. 2). A single small lymph gland near the pancreas showed similar involvement. All other internal organs appeared normal.

Dr. Ellen Furey, Port Arthur, and Dr. William C. Levin, Galveston, assisted in the study of this patient.

Discussion

Various inclusions may occur in myeloma cells; Snapper and associates[8] depict them on the frontispiece of their monograph dealing with this disease. Usually, such cells are only few in number, if they are present at all. Rarely do they make up practically all of the abnormal bone marrow cells, as was seen in our patient. This unusual marrow picture (Fig. 1) suggested the diagnosis of reticuloendotheliosis. Not until various tissue sections became available for microscopic study at necropsy could the correct diagnosis be established with certainty. Curiously, at postmortem examination large globular masses were observed outside of the cells; they were too big to have escaped from degenerated cell bodies but apparently consisted of the same material as the intracellular occlusions (Fig. 3). The demonstration of pyroglobulins pointed toward multiple myeloma. However, these substances have occasionally been seen in lymphosarcoma, diffuse metastatic carcinoma and Waldenstrom's macroglobulinemia.[4, 9, 10] None of these entities was present in our patient; nevertheless, it is conceivable that reticuloendotheliosis and other rare blood dyscrasias could occasionally be associated with such abnormal heat precipitable proteins.[4, 9, 10] At the time of the patient's illness the differentiation between myeloma and reticuloendotheliosis was of little practical significance. However, such distinction may become important with the development of more specific drugs in the future.

Summary

A case of multiple myeloma with unusual bone marrow findings is reported. The classical myeloma marrow filled with masses or sheets of mature or immature cells was not observed in spite of repeated aspirations at different locations. Instead, numerous large reticulum cells filled with globular inclusions were found, and similar globules were seen outside the cells in the tissues at autopsy. One may speculate that these globules are identical with or related to the pyroglobulins present in the patient's serum.

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Epilepsy Not Related to Criminal Behavior

An epileptic is no more a potential murderer than the so-called normal individual, Dr. Samuel Livingston, Baltimore, says in an editorial in the April 13 *Journal of the American Medical Association.*

The impression that epilepsy is related to murder and crimes of passion which might have been gained from the Jack Ruby trial also was refuted in a letter signed by five expert witnesses published in the *Journal.*

"To my knowledge, there are no reports in the current medical literature which prove that there is a higher rate of criminal action among epileptics than among other individuals," Dr. Livingston said.

"Based on my 27 years of specializing in the treatment, study and contact with the course of living of some 15,000 epileptic patients on every social level, I can state positively that the instance of crimes committed by these patients was no greater than—even showed no difference from—that in a similar number of nonepileptics."

The letter signed by Dr. Francis M. Forster, Madison, Wis., Peter Kellaway, Ph.D., Houston, Dr. Roland P. Mackay, Chicago, Dr. Robert S. Schwab, Boston, and Dr. A. Earl Walker, Baltimore, said:

"We submit the following statements to the many physicians responsible for the health and peace of mind of nearly 1,000,000 patients with various types of epileptic seizures:

"Neither the clinical history nor the electroencephalogram of Mr. Ruby indicated any definite evidence of epilepsy.

"Epileptic seizures are never associated with complicated and planned criminal acts of violence.

"Epileptics are as safe to be with as any group of people except in extremely rare and usually predictable situations."

Epidermoid Cyst of Testis

STANLEY WEITZNER, M.D.

A case of epidermoid cyst of the testis is reported. Nine months after a simple excision of the cyst, the patient was asymptomatic.

CASE REPORTS

EPIDERMOID CYSTS OF the testis are uncommon. Dockerty and Priestley[2] in 1942 recorded the first case in this country. Since then, 12 other cases have been reported in the American literature.[1, 4-7, 9-11] In addition, Friedman and Moore[3] alluded to ten cases of epidermoid cysts of the testis in their study of 922 testicular neoplasms, but did not provide any specific clinicopathologic data.

The writer adds a well-documented case to the small number already recorded.

Case Report

A 21-year-old white man had a moderately tender mass at the posterior-superior aspect of the right testis. It was found during routine physical examination. Questioning revealed that this mass, which occasionally was mildly tender, had been present for three years, and had enlarged slightly during the past year. The past medical history was not otherwise remarkable.

The laboratory data including a skin test for tuberculosis (using purified protein derivative), chorionic gonadotropic studies, and chest roentgenograms were not remarkable.

Surgical exploration revealed a hard yellow tumor in the posterior-superior pole of the right testis. Incision of the tumor disclosed a cyst filled with tan-gray grumous material. The cyst wall was enucleated from the adjacent testis and the testis was conserved. The postoperative course was uneventful.

The specimen on gross examination consisted of a cystic mass measuring 2.3 cm. in greatest diameter. The outer surface was irregularly coated with light tan testicular tissue. The cyst contained soft, tan-gray material and had a smooth, shiny, gray lining. The wall of the cyst was 1 mm. in average thickness.

Microscopically, the cyst wall was lined in its entirety by stratified squamous epithelium covered by laminated keratin. The outer surface of the cyst was bordered by atrophic seminiferous tubules (Figs. 1 and 2). Hair within the cyst, and skin appendages and mesodermal and endodermal derivatives in the wall were absent. Histopathologically, the lesion was a characteristic epidermoid cyst.

Discussion

An epidermoid cyst is one lined by stratified squamous epithelium and filled with keratin. A dermoid cyst has the additional features of skin appendages within its wall, and hair and sebum as contents. A teratoma is a tumor composed of derivatives of the three primitive germ layers in varying combinations, and is designated benign (adult) or malignant according to the degree of maturation of any of its histologic elements.

The testicular cyst in this case was the epidermoid type. Conspicuous absence of epidermal appendages and mesodermal and endodermal elements eliminated the diagnosis of dermoid cyst or teratoma.

The genesis of this tumor is conjectural. Since the banal epidermoid cyst of the skin and the rare epidermoid cyst of the brain are derived from epidermal inclusions, it would be inviting to postulate a similar basis for the epidermoid cyst of the testis. Unfortunately, to the writer's knowledge, this has not been described. It would be preferable to consider this odd testicular tumor as an example of extreme unilateral maturation of a teratoma in which the development of the stratified squamous element completely excludes all other components. This process is not unique since both struma ovarii and pseudomucinous cystadenoma of the ovary are considered to represent unilateral developments in teratomas.[8]

Simple excision of the cyst was performed in this case as it has been in several[1, 9, 10] of

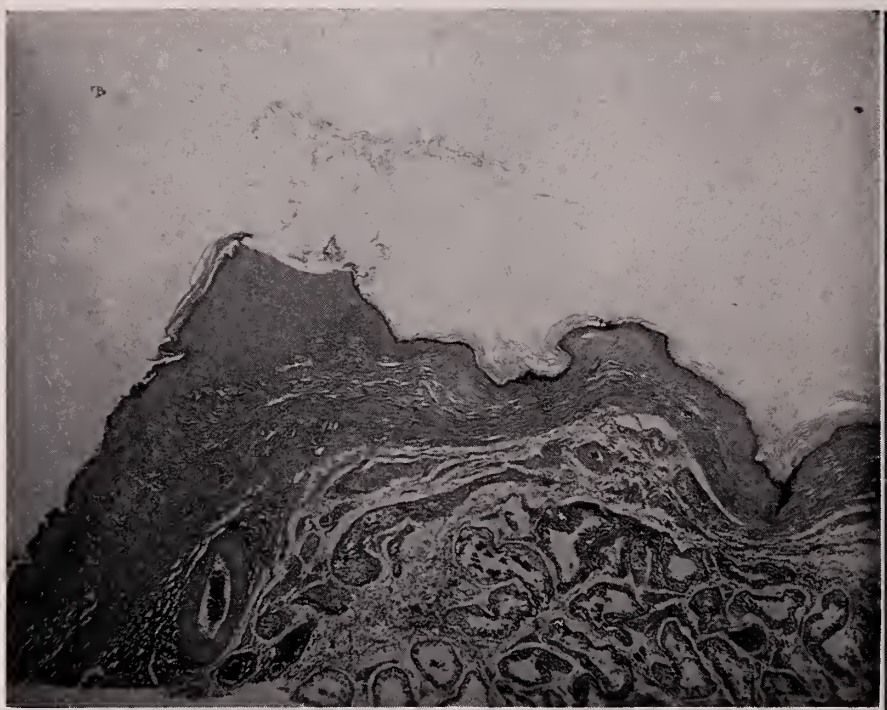


Fig. 1. Representative section of entire cyst showing the stratified squamous epithelial cyst lining covered by keratin and atrophic seminiferous tubules on the external surface. Hematoxylin and eosin stain, X 33.

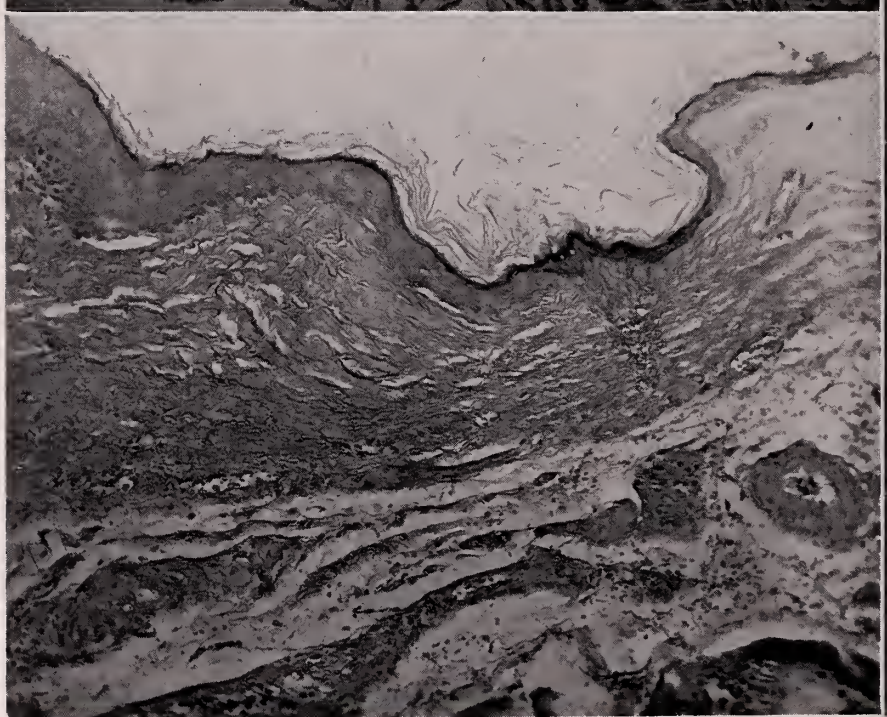


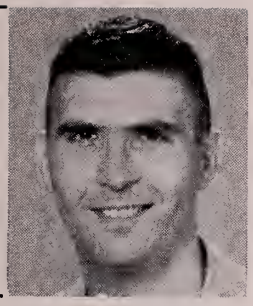
Fig. 2. Higher power of Fig. 1 showing wall of the cyst in greater detail. Hematoxylin and eosin, X 116.

the aforementioned reported cases. This is certainly the therapy of choice if the diagnosis is absolutely certain, and would be of value in patients in whom the opposite testis has been sacrificed previously as, for example, in cryptorchidism, post-trauma, or torsion. If any doubt of the diagnosis exists, orchiectomy is indicated rather than risking incision of a malignant tumor with possible resultant spread.

Summary

A case of epidermoid cyst of the testis, an unusual testicular neoplasm, is presented. The probability of this lesion representing unilateral development of a teratoma is considered. Simple excision of a testicular tumor may be performed only when one is absolutely certain of the nonmalignant nature of the tumor.

Dr. Stanley Weitzner was a captain serving with the Pathology Division, Fourth U.S. Army Medical Laboratory, Fort Sam Houston, when this case was treated. He now is with Southside Hospital, Bay Shore, N. Y.



Addendum—The patient is apparently asymptomatic after nine months of follow-up.

The Departments of Urology and Pathology of the U. S. Naval Hospital, Corpus Christi, granted permission for publication of the case.

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Cigarette Ads Changing

The report of Surgeon General Luther L. Terry's Advisory Committee on Smoking and Health has profoundly affected the tobacco industry, according to an article in the April 18 issue of *The Saturday Evening Post.*

"Even long-standing advertising policies are changing," the article reads.

R. J. Reynolds has announced its plan to withdraw all spot radio commercials from disc-jockey shows after 3 p.m. and on weekends when school children are apt to hear them. It has stopped all ads in campus publications and has ceased all promotional activity in colleges. It will no longer use models under 25 years of age, and the theme that virility goes with smoking has been dropped. He-men actors in the ads are being replaced with inanimate objects such as a simple pack of cigarettes.

Giant Parotid Tumor

HYLMAR E. KARBACH, JR., M.D., and WILLIAM J. HILLS, M.D.

CASE REPORTS

IN DECADES gone by giant tumors were more common than in recent years when it has become a rare thing to see a solid tissue tumor of large size. This case is the 23rd reported in the literature.

Case Report

An 80-year-old man with a large tumor anterior to his right ear was seen at the Nix Memorial Hospital Tumor Clinic in San Antonio on March 21, 1962. A former vaquero, he had first noticed the tumor in 1913 after he had been kicked in the head by a horse. The tumor grew to the size of a hen's egg and remained that size until about 1959 when it began to grow rapidly. The patient sought medical advice because he had trouble shaving and was experiencing some pain in the region of the right ear (Fig. 1).

Physical examination showed a nodular, firm, rubbery tumor about the size of the patient's head with superficial lateral ulcerations. The skin overlaying the tumor was thin, atrophic, and firmly adherent to the underlying tumor mass. Facial nerve defect was noted in the zygomaticotemporal branches. No enlarged lymph nodes were found in the neck. The patient was otherwise in good general health.

Routine laboratory tests and chest roentgen ray were normal.

Surgery was performed on April 12, 1962, and the gross pathologic finding was that the specimen was a multilobulated, firm unencapsulated mass of tissue weighing 2600 gm. and measuring 29 by 15 by 13.5 cm. Microscopic examination revealed a malignant mixed tumor. Following surgery the patient's appearance was good (Fig. 2).

The tumor recurred within six months and, in spite of radiation, advanced rapidly. The patient succumbed to the disease May 3, 1963.

Discussion

In reviewing the literature, 22 previous cases of tumors such as this were found, nine of which were larger. Many of these cases were reported in the 19th century.

The largest tumor (11.4 kg.) reported was not removed because the patient refused surgery, probably with just cause.

> Even by 1870, the removal of a tumour of any magnitude required a bold surgeon and a still bolder patient, for anaesthesia was still in its infancy and both haemostasis and asepsis totally inadequate by present day standards.[5]

Fig. 1. Preoperative appearance.

Fig. 2. Appearance on discharge from hospital.

An unusually large tumor developed over a period of 49 years, growing rapidly during the last three. Patient sought medical help because of pain and a shaving problem.

Fig. 3. Roentgenogram of tumor.

It seems fantastic that some of these early tumors were removed in three to six minutes.

It is important to mention that many of these tumors had been present for a number of years when the patient was first seen by members of the medical profession. Growth periods of 40 years,[1] 43 years,[2] 16 years,[5] 5 years,[4] and, in the present case, 49 years are representative. It should be noted that the growth over a long period of time of mixed tumors of the parotid glands in no way guarantees against malignant transformation.

The location of the tumor in the present case is worthy of mention. All of the tumors noted in earlier reports were below the ear and slightly more posterior.

Although mixed tumors as large as 2800 gm.[5] are sometimes made up entirely of soft tissue, it is not unusual to find calcification a prominent part of the tumor mass. A case of a mixed tumor of 2100 gm.[2] and the present case (note calcifications shown in Fig. 3) demonstrate this.

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Geographic Distribution Of Gastrointestinal Cancer

RESEARCH & INVESTIGATION

THE INCIDENCE OF gastrointestinal (G.I.) malignancy varies greatly in different countries and even within the boundaries of a country or a state. For example, in the United States, the death rate from G.I. cancer was reported to be 50.3 per 100,000 population in 1955,[9] while for Texas, the death rate of G.I. cancer is approximately 30 per cent per 100,000 population, or about 31 per cent of total malignancy deaths. These variations have been related to age, sex, race, ethnic origin, nativity, genetic or familial factors, socioeconomic status, dietary habits, urban-rural residence, geographic regions, and the nature of soils.[4, 6-15]

Texas is unique in many ways, particularly because of its large area of varied terrain, climate, and natural resources. Also, there are many relatively isolated ethnic groups, such as Germans, Czechoslovakians, Indians, and Latin Americans. The authors were hopeful that a study of gastrointestinal cancer mortality rates in this state by county, compared to known county variables, might suggest some leads regarding the etiology of G.I. cancer.

Materials and Methods

Our data is from the vital statistics of the United States Department of Health, Education and Welfare, and the 1960 population census. Information was also obtained from the "Texas Almanac, 1961-1962," and "The Atlas of Texas." The total number of reported deaths from all causes, and the number of G.I. cancer deaths for each of the 254 counties in Texas from 1953 to 1957 were tabulated. The mortality rate for G.I. cancer in each county was then calculated by dividing the observed number of G.I. cancer deaths by the total deaths from all causes. The rate is expressed as a per cent of the total deaths from all causes, and covers a five-year period (Table 1, col. 3).

One of the more obvious causes for a wide variation in the death rate is the variation of age groups in each county. Statistics are available for the actual number of G.I. cancer deaths in age groups (as 15-19, 20-24, 25-29) for the entire state, but not for individual counties. A census population of the corresponding age groups for the state, as well as each county, is available. Hence, by dividing the number of deaths from G.I. cancer in a particular age group by the census population of the corresponding age groups in the entire state a predicted cancer death rate (R) was obtained for that age group for the whole state:

$$\frac{\text{Total G.I. cancer deaths per age group}}{\text{Total population per age group}} = \text{R.}$$

Utilizing this factor, the population of that age group in each county (PC) multiplied by (R) gave a predicted death rate for gastrointestinal cancer in that age group in each county. Eleven age groups, from 20-24 years through 70 and over, were considered. The age groups below 20 years were not evaluated because the number of deaths from G.I. cancer was negligible. Finally, by the summation of the results for each age group, the predicted G.I. cancer deaths on the basis of age distribution for individual counties was obtained. This is expressed in percentage (Table 1, col. 4).

The observed and age expected mortality rates were compared and expressed as a mortality ratio in percentage (1:1 ratio= 100 per cent).

Results

The results are tabulated in Table 1. Fig. 1 shows the percentage distribution of G.I. cancer deaths by counties in relation to the total deaths from all causes. The mean is 4.4 per cent with a range of 0 to 28.5 per

Geographic distribution of deaths from gastrointestinal cancer was studied in relation to age, ethnic, and socioeconomic groups; dietary habits; urban-rural residence; geographic regions; and nature of soils. No obvious correlations were found; but further analysis may be justified.

HELEN ONG, M.D.
MARCEL PATTERSON, M.D.

cent, while the expected mortality (Fig. 2) on the basis of age distribution varies from .3 per cent to 14 per cent with a mean of 4.3 per cent.

In comparing the actual mortality from G.I. cancer with the predicted mortality rate according to age distribution by individual counties (Fig. 3), it is apparent that there are no significant differences except in seven counties. Here, the observed mortality ratio is over 200 per cent of the age predicted, and in two of these seven counties, the observed mortality ratio is over 300 per cent of the age predicted. With the exception of McMullen and Camp Counties (observed death rates of 5.8 per cent and 7.3 per cent respectively), the remaining five counties have an actual G.I. cancer mortality rate greater than 8 per cent. These counties are Blanco, Crane, La Salle, Loving, and Motley. The geographical patterns of these counties were mapped and compared with geographic maps depicting the various races, ethnic groups, socioeconomic status, soils, crops, and minerals.

The county with the largest observed mortality rate (28.5 per cent) is Loving, a sparsely settled county in far West Texas. The figure is misleading because this is the most sparsely populated county (226) in the state. During the five-year period observed, there were seven deaths, of which two were from G.I. cancer. Any death from G.I. cancer among this small population would appear as significant. The observed high incidence is probably a statistically spurious finding.

The deaths in La Salle and McMullen (8.8 and 5.8 per cent, respectively) seem less likely to be influenced by these small numbers. These two adjacent counties, located on the Rio Grande plain about 80 to 100 miles south of San Antonio, have a combined population of 7,088. Ethnically, there are four Negroes, no other nonwhites, and relatively few white

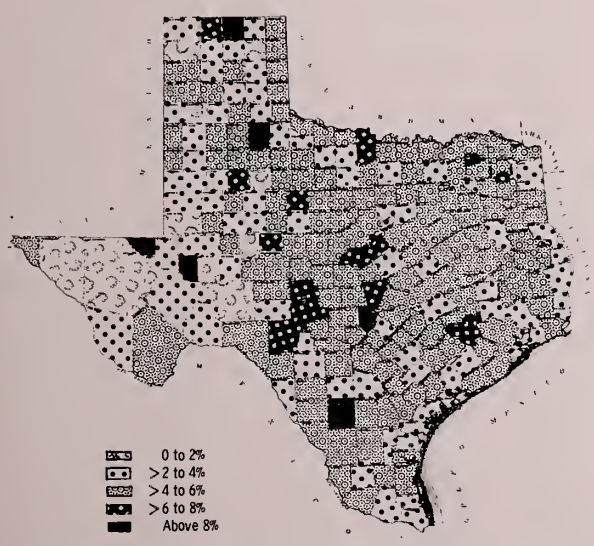

Fig. 1. The percentage of deaths in each county from gastrointestinal cancer is shown for the years 1953 through 1957.

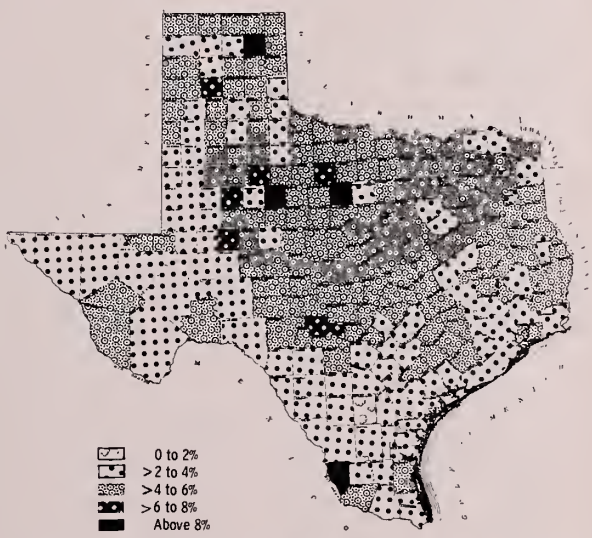

Fig. 2. The predicted percentage of deaths in each county from cancer of the gastrointestinal tract is shown. The prediction was based on known ages of county residents.

TABLE 1.—*Total Deaths, 1953-1957.*

County	Number of Deaths in County	Number of Deaths from G.I. Cancer	Actual Percentage, G.I. Cancer Deaths	Predicted Percentage, Deaths from G.I. Cancer Based on Age	Mortality Ratio Between Actual and Predicted Death Rates in Percentage
Anderson	1,626	71	4.3	4.1	104.9
Andrews	207	4	1.9	3.5	54.3
Angelina	1,534	72	4.6	4.4	104.5
Aransas	254	12	4.7	4.6	102.2
Archer	231	7	3.0	4.9	61.2
Armstrong	103	4	3.8	4.4	86.4
Atascosa	800	29	3.6	3.7	97.3
Austin	799	50	6.2	4.6	134.8
Bailey	256	14	5.4	4.1	131.7
Bandera	210	9	4.2	5.0	84.0
Bastrop	1,076	48	4.4	4.0	110.0
Baylor	288	10	3.4	4.9	69.4
Bee	839	41	4.8	3.3	145.5
Bell	2,767	126	4.5	4.5	100.0
Bexar	22,709	1,032	4.5	3.8	118.4
Blanco	199	21	10.5	5.0	210.0
Borden	17	1	5.8	8.0	72.5
Bosque	726	34	4.6	4.8	95.8
Bowie	2,555	137	5.3	4.3	123.3
Brazoria	1,888	78	4.1	4.0	102.5
Brazos	1,539	65	4.2	3.8	110.5
Brewster	259	11	4.2	3.6	116.7
Briscoe	146	6	4.1	4.0	102.5
Brooks	267	11	4.1	3.7	110.8
Brown	1,389	62	4.4	4.9	89.8
Burleson	673	19	2.8	4.2	66.7
Burnet	495	31	6.2	4.6	134.8
Caldwell	1,047	56	5.3	3.6	147.2
Calhoun	408	16	3.9	3.3	118.2
Callahan	443	22	4.9	5.2	94.2
Cameron	5,276	179	3.3	2.9	113.8
Camp	505	37	7.3	3.5	208.6
Carson	223	12	5.3	4.5	117.8
Cass	1,257	49	3.8	3.9	97.4
Castro	220	4	1.8	3.2	56.3
Chambers	328	10	3.0	4.1	73.2
Cherokee	1,654	65	3.9	4.6	84.8
Childress	514	24	4.6	3.9	117.9
Clay	422	27	6.3	4.8	131.3
Cochran	186	9	4.8	3.7	129.7
Coke	157	11	7.0	3.8	184.2
Coleman	743	31	4.1	4.8	85.4
Collin	2,069	84	4.0	4.4	90.9
Collingsworth	380	17	4.4	4.0	110.0
Colorado	923	39	4.2	4.1	102.4
Comal	769	34	4.4	4.5	97.8
Comanche	770	39	5.0	5.0	100.0
Concho	169	8	4.7	5.2	90.4
Cooke	976	50	5.1	4.7	108.5
Coryell	717	27	3.7	5.1	72.5
Cottle	248	6	2.4	3.5	68.6
Crane	104	9	8.6	3.8	226.3
Crockett	131	2	1.5	3.4	44.1
Crosby	359	19	5.2	4.3	120.9
Culberson	96	2	2.0	2.6	76.9
Dallam	233	9	3.8	5.1	74.5
Dallas	28,264	1,236	4.3	4.2	102.4
Dawson	667	22	3.2	3.8	84.2
Deaf Smith	259	15	5.7	5.9	96.6
Delta	422	27	6.3	4.3	146.5
Denton	1,775	92	5.1	4.5	113.3
De Witt	1,187	40	3.3	4.1	80.5
Dickens	227	10	4.4	5.3	83.0
Dimmit	366	18	4.9	3.2	153.1
Donley	269	9	3.3	4.6	71.7

TABLE 1.—*Total Deaths, 1953-1957.—Continued*

County	Number of Deaths in County	Number of Deaths from G.I. Cancer	Actual Percentage, G.I. Cancer Deaths	Predicted Percentage, Deaths from G.I. Cancer Based on Age	Mortality Ratio Between Actual and Predicted Death Rates in Percentage
Duval	522	24	4.5	3.5	128.6
Eastland	1,284	64	4.9	4.7	104.3
Ector	1,566	61	3.8	3.7	102.7
Edwards	79	5	6.3	5.2	121.2
Ellis	2,302	106	4.6	4.0	115.0
El Paso	8,068	370	4.5	3.5	128.6
Erath	974	51	5.2	5.2	100.0
Falls	1,318	52	3.9	4.1	95.1
Fannin	1,589	77	4.8	4.4	109.1
Fayette	1,327	62	4.6	4.4	104.5
Fisher	370	18	4.8	14.0	34.3
Floyd	506	27	5.3	3.7	143.2
Foard	187	6	3.2	4.2	76.2
Fort Bend	1,434	81	5.6	3.7	151.4
Franklin	271	12	4.4	5.4	81.5
Freestone	854	37	4.3	4.2	102.4
Frio	384	15	3.9	3.9	100.0
Gaines	346	9	2.6	3.3	78.8
Galveston	5,409	200	3.6	3.3	109.1
Garza	220	15	6.8	4.3	158.1
Gillespie	556	29	5.2	4.8	108.3
Glasscock	22	1	4.5	6.2	72.6
Goliad	289	16	5.5	3.9	141.0
Gonzales	1,055	39	3.6	4.0	90.0
Gray	935	35	3.5	4.3	86.0
Grayson	3,755	170	4.5	4.1	109.8
Gregg	2,650	121	4.5	3.9	115.4
Grimes	939	48	5.1	3.4	150.0
Guadalupe	1,253	68	5.4	4.0	135.0
Hale	1,191	50	4.1	4.1	100.0
Hall	403	16	3.9	4.1	95.1
Hamilton	562	35	6.2	5.0	124.0
Hansford	131	12	9.1	4.8	189.6
Hardeman	434	24	5.5	4.6	119.6
Hardin	846	35	4.1	4.6	89.1
Harris	36,157	1,641	4.5	3.8	118.4
Harrison	2,096	100	4.7	5.0	94.0
Hartley	103	2	1.9	3.0	63.3
Haskell	544	29	5.3	4.4	120.5
Hays	831	33	3.9	3.8	102.6
Hemphill	118	7	5.9	5.1	115.7
Henderson	1,089	51	4.6	4.7	97.9
Hidalgo	6,128	221	3.6	3.1	116.1
Hill	1,613	76	4.7	4.2	111.9
Hockley	620	25	4.0	3.9	102.6
Hood	319	9	2.8	4.6	60.9
Hopkins	1,135	53	4.6	4.4	104.5
Houston	957	43	4.4	4.6	95.7
Howard	1,126	34	3.0	4.1	73.2
Hudspeth	108	2	1.8	2.8	64.3
Hunt	2,134	91	4.2	4.2	100.0
Hutchinson	827	27	3.2	3.6	88.9
Irion	54	3	5.5	4.9	112.2
Jack	352	18	5.1	5.0	102.0
Jackson	561	23	4.0	3.6	111.1
Jasper	931	36	3.8	4.3	100.0
Jeff Davis	65	1	1.5	4.2	35.7
Jefferson	7,913	384	4.8	3.8	126.3
Jim Hogg	217	8	3.6	2.9	124.1
Jim Wells	1,040	39	3.7	3.4	108.8
Johnson	1,722	70	4.0	4.5	88.9
Jones	906	56	6.1	4.7	129.8
Karnes	754	42	5.5	3.2	171.9
Kaufman	1,617	76	4.7	4.5	104.4
Kendall	296	17	5.7	4.9	116.3

TABLE 1.—*Total Deaths, 1953-1957.—Continued*

County	Number of Deaths in County	Number of Deaths from G.I. Cancer	Actual Percentage, G.I. Cancer Deaths	Predicted Percentage, Deaths from G.I. Cancer Based on Age	Mortality Ratio Between Actual and Predicted Death Rates in Percentage
Kenedy	19	1	5.2	4.7	110.6
Kent	61	1	1.6	6.1	26.2
Kerr	796	37	4.6	6.6	69.7
Kimble	180	12	6.6	5.3	124.5
King	24	1	4.1	2.9	141.4
Kinney	105	5	4.7	3.9	120.5
Kleberg	876	36	4.1	3.1	132.3
Knox	354	19	5.3	4.7	112.8
Lamar	2,159	101	4.6	3.9	117.9
Lamb	761	27	3.5	4.0	87.5
Lampasas	488	22	4.5	4.6	97.8
La Salle	293	26	8.8	3.1	283.9
Lavaca	1,197	64	5.3	4.3	123.3
Lee	505	17	3.3	4.5	73.3
Leon	691	34	4.9	3.8	128.9
Liberty	1,309	47	3.5	3.8	92.1
Limestone	1,212	54	4.4	4.4	100.0
Lipscomb	142	6	4.2	4.7	89.4
Live Oak	340	14	4.1	3.6	113.9
Llano	286	12	4.1	5.2	78.8
Loving	7	2	28.5	4.2	678.6
Lubbock	3,669	152	4.1	4.3	95.3
Lynn	377	14	3.7	4.1	90.2
McCulloch	585	34	5.8	4.1	141.5
McLennan	5,961	262	4.3	4.3	100.0
McMullen	585	34	5.8	0.3	1933.3
Madison	410	19	4.9	4.2	109.5
Marion	516	19	3.6	3.4	105.9
Martin	203	12	5.9	3.3	178.8
Mason	221	11	4.9	4.9	100.0
Matagorda	1,037	44	4.2	3.7	113.5
Maverick	490	19	3.8	3.1	122.6
Medina	733	38	5.1	4.3	118.6
Menard	165	11	6.6	4.7	140.4
Midland	1,171	36	3.0	4.0	75.0
Milam	1,285	67	5.2	4.2	123.8
Mills	267	21	7.8	5.6	139.3
Mitchell	483	19	3.9	4.4	88.6
Montague	833	37	4.4	5.0	88.0
Montgomery	1,213	39	3.2	4.0	80.0
Moore	277	6	2.1	3.9	53.8
Morris	419	11	2.6	5.1	51.0
Motley	157	14	8.9	4.3	207.0
Nacogdoches	1,238	62	5.0	4.8	104.2
Navarro	2,187	97	4.4	3.9	112.8
Newton	400	15	3.7	5.0	74.0
Nolan	771	42	5.4	4.3	125.6
Nueces	6,000	243	4.0	3.5	114.3
Ochiltree	229	9	3.9	4.2	92.9
Oldham	62	3	4.8	4.3	111.6
Orange	1,464	57	3.8	3.9	97.4
Palo Pinto	1,110	51	4.5	4.0	112.5
Panola	679	38	5.5	4.9	112.2
Parker	1,040	56	5.3	4.9	108.2
Parmer	206	7	3.3	5.1	64.7
Pecos	340	11	3.2	3.5	91.4
Polk	753	34	4.5	3.9	115.4
Potter	3,407	126	3.6	3.9	92.3
Presidio	197	6	3.0	4.5	66.7
Rains	188	5	2.6	4.9	53.1
Randall	429	25	5.8	7.1	81.7
Reagan	117	4	3.4	3.3	103.0
Real	105	3	2.8	4.1	68.3
Red River	1,020	44	4.3	4.0	107.5
Reeves	608	12	1.9	2.5	76.0

TABLE 1.—*Total Deaths, 1953-1957.—Continued*

County	Number of Deaths in County	Number of Deaths from G.I. Cancer	Actual Percentage, G.I. Cancer Deaths	Predicted Percentage, Deaths from G.I. Cancer Based on Age	Mortality Ratio Between Actual and Predicted Death Rates in Percentage
Refugio	444	16	3.6	3.2	112.5
Roberts	26	----	----	8.2	0.0
Robertson	1,029	45	4.3	3.7	116.2
Rockwall	310	13	4.1	4.2	97.6
Runnels	707	32	4.5	4.7	95.7
Rusk	1,660	74	4.4	4.5	97.8
Sabine	294	11	3.7	5.6	66.1
San Augustine	424	22	5.1	3.7	137.8
San Jacinto	297	15	5.0	5.1	98.0
San Patricio	1,585	51	3.2	3.0	106.7
San Saba	371	21	5.6	5.2	107.7
Schleicher	107	6	5.6	4.6	121.7
Scurry	645	14	2.1	3.9	53.8
Shackelford	242	14	5.7	4.2	135.7
Shelby	1,122	37	3.2	4.3	74.4
Sherman	89	6	6.7	4.3	155.8
Smith	3,306	174	5.2	4.3	120.9
Somervell	141	5	3.8	5.8	60.3
Starr	450	24	5.3	4.2	126.2
Stephens	498	24	4.8	20.5	23.4
Sterling	51	1	1.9	4.1	46.3
Stonewall	138	8	5.7	4.4	129.5
Sutton	123	5	4.0	4.1	97.6
Swisher	315	9	2.8	4.7	59.6
Tarrant	17,287	778	4.5	4.1	109.8
Taylor	2,602	98	3.7	4.8	77.1
Terrell	68	2	2.9	5.2	55.8
Terry	503	15	2.9	3.7	78.4
Throckmorton	121	7	5.7	6.4	89.1
Titus	777	37	4.7	4.5	104.4
Tom Green	2,376	104	4.3	4.4	97.7
Travis	6,181	290	4.6	4.7	97.9
Trinity	481	26	5.4	4.2	128.6
Tyler	518	24	4.6	4.6	100.0
Upshur	938	54	5.7	4.4	129.5
Upton	168	2	1.1	3.6	30.6
Uvalde	717	28	3.9	3.8	102.6
Val Verde	862	40	4.6	2.8	164.3
Van Zandt	1,110	67	6.0	4.4	136.4
Victoria	1,533	73	4.7	3.5	134.3
Walker	776	23	2.9	4.6	63.0
Waller	533	34	6.3	3.9	161.5
Ward	425	12	2.8	3.3	84.8
Washington	1,108	50	4.5	4.5	100.0
Webb	2,236	92	4.1	3.5	117.1
Wharton	1,466	60	4.0	4.2	95.2
Wheeler	412	17	4.1	4.4	93.2
Wichita	3,716	166	4.4	4.5	97.8
Wilbarger	761	40	5.2	5.3	98.1
Willacy	734	22	2.9	2.7	107.4
Williamson	1,859	85	4.5	4.2	107.1
Wilson	675	32	4.7	3.8	123.7
Winkler	263	6	2.2	5.9	37.3
Wise	851	33	3.8	4.6	82.6
Wood	1,002	43	4.2	4.5	93.3
Yoakum	172	6	3.4	3.5	97.1
Young	782	26	3.3	4.8	68.8
Zapata	70	4	5.7	9.0	63.3
Zavala	430	18	4.1	3.2	128.1

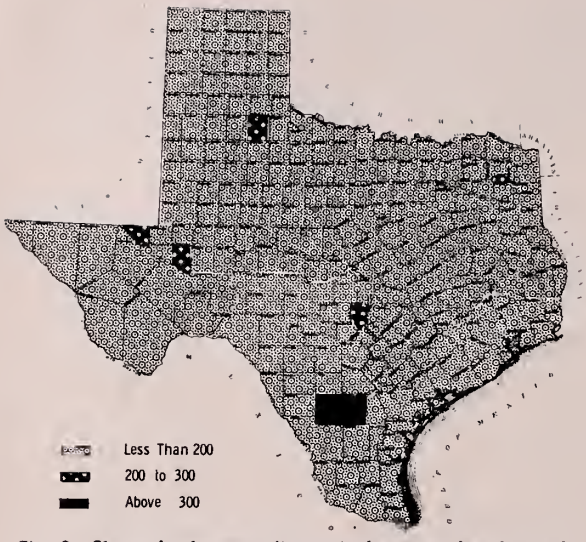

Fig. 3. Shown is the mortality ratio between the observed and the predicted gastrointestinal cancer death rates.

persons of Spanish surname. The soils are dark gray and sandy loam with some chocolate and black sands. Mesquite, cedar, and small oak timber are found mostly along streams. The oil production is small. It is excellent game country for deer and turkey, and for fishing in the Frio and Nueces Rivers. The main industry is ranching, although in La Salle there are some irrigated winter truck farms and in McMullen, a few gas wells. The observed death rate from G.I. cancer (8.8 per cent) in La Salle is much higher than would be predicted by age (3.1 per cent). The observed G.I. cancer death rate for McMullen is 5.8 per cent; the predicted is less than 1 per cent. The only obvious unique factor about McMullen County is the presence of twice as many single men as women.

The other four counties with higher cancer death rates are widely separated and have no mutually distinguishing features that the authors can detect or correlate.

Discussion

Several reports have indicated that G.I. cancer mortality varies with geographic latitude and is highest in those places farthest from the equator. Haenszel[9] has observed that stomach cancer mortality is definitely higher in the northeast and north central parts of the United States than in the south and that this relationship holds also for the intestine, rectum, and esophagus. Similar observations were noted in Britain[19] and the Netherlands.[20] Our analysis shows no significant differences between the northern and southern parts of the state.

Possible relationship between stomach cancer and soil[4] has also been studied in Wales[11] and the Netherlands.[20] There seems to be a significant correlation between high mortality and peat soil areas. It was suggested

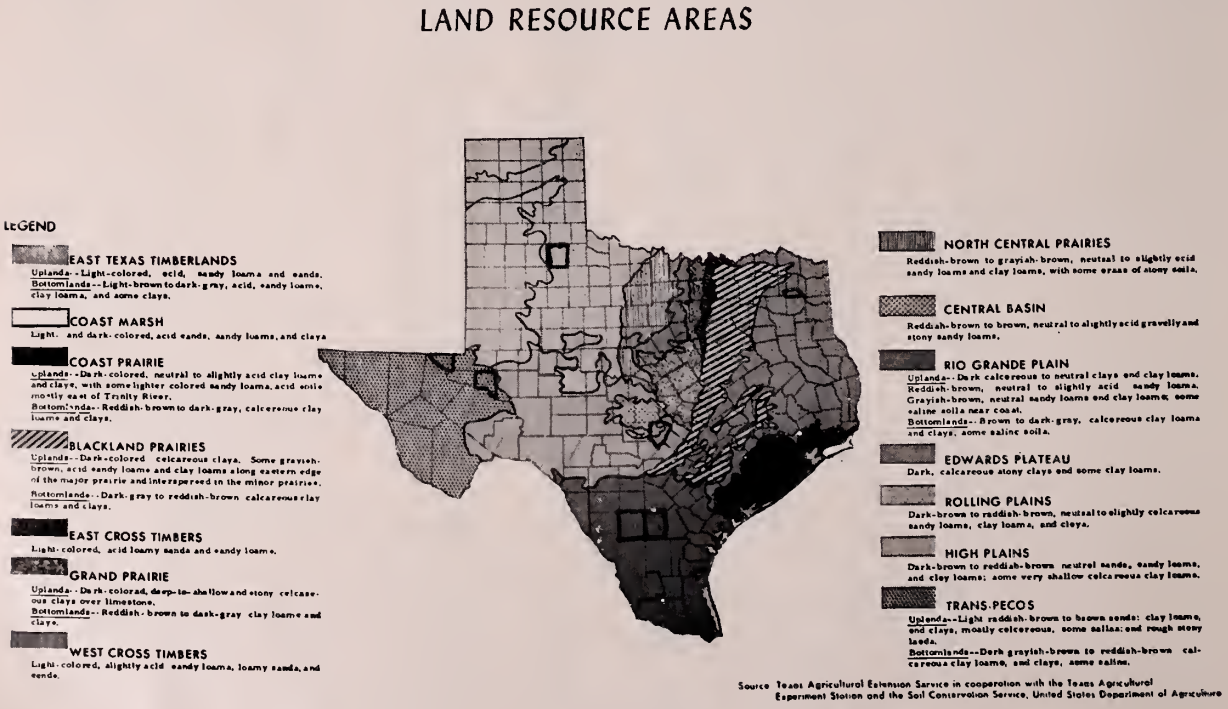
LAND RESOURCE AREAS
LEGEND
EAST TEXAS TIMBERLANDS
Uplands--Light-colored, acid, sandy loams and sands. Bottomlands--Light-brown to dark-gray, acid, sandy loams, clay loams, and some clays.
COAST MARSH
Light- and dark-colored, acid sands, sandy loams, and clays
COAST PRAIRIE
Uplands--Dark-colored, neutral to slightly acid clay loams and clays, with some lighter colored sandy loams, acid soils mostly east of Trinity River. Bottomlands--Reddish-brown to dark-gray, calcareous clay loams and clays.
BLACKLAND PRAIRIES
Uplands--Dark-colored, calcareous clays. Some grayish-brown, acid sandy loams and clay loams along eastern edge of the major prairie and interspersed in the minor prairies. Bottomlands--Dark-gray to reddish-brown calcareous clay loams and clays.
EAST CROSS TIMBERS
Light-colored, acid loamy sands and sandy loams.
GRAND PRAIRIE
Uplands--Dark-colored, deep-to-shallow and stony calcareous clays over limestone. Bottomlands--Reddish-brown to dark-gray clay loams and clays.
WEST CROSS TIMBERS
Light-colored, slightly acid sandy loams, loamy sands, and sands.
NORTH CENTRAL PRAIRIES
Reddish-brown to grayish-brown, neutral to slightly acid sandy loams and clay loams, with some areas of stony soils.
CENTRAL BASIN
Reddish-brown to brown, neutral to slightly acid gravelly and stony sandy loams.
RIO GRANDE PLAIN
Uplands--Dark calcareous to neutral clays and clay loams. Reddish-brown, neutral to slightly acid sandy loams. Grayish-brown, neutral sandy loams and clay loams; some saline soils near coast. Bottomlands--Brown to dark-gray, calcareous clay loams and clays; some saline soils.
EDWARDS PLATEAU
Dark, calcareous stony clays and some clay loams.
ROLLING PLAINS
Dark-brown to reddish-brown, neutral to slightly calcareous sandy loams, clay loams, and clays.
HIGH PLAINS
Dark-brown to reddish-brown neutral sands, sandy loams, and clay loams; some very shallow calcareous clay loams.
TRANS-PECOS
Uplands--Light reddish-brown to brown sands; clay loams, and clays, mostly calcareous, some saline; and rough stony lands. Bottomlands--Dark grayish-brown to reddish-brown calcareous clay loams, and clays, some saline.
Source Texas Agricultural Extension Service in cooperation with the Texas Agricultural Experiment Station and the Soil Conservation Service, United States Department of Agriculture

Fig. 4. The type of soil in each land area in Texas is outlined.

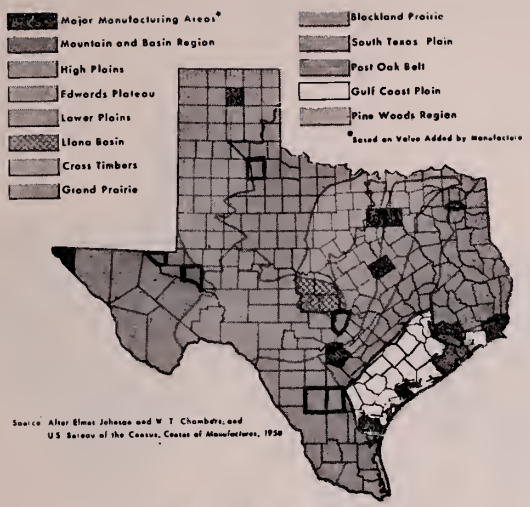

Fig. 5. Geographic regions of Texas.

that this may be related to the presence or absence of trace elements in the soils. These elements may produce cancer of the stomach, directly or indirectly, through the crops grown and ingested in these areas. Other dietary factors considered in the genesis of G.I. neoplasm are the eating of smoked fish in Iceland,[6] and the ability to produce hepatomas in rats with butter yellow.[10] No particular type of soil or known eating habits correlate with G.I. cancer deaths in Texas (Figs. 4, 5, 6).

The incidence of upper G.I. cancer in the United States is higher among Negroes than whites in metropolitan and nonmetropolitan counties.[9] However, the reverse is true for cancer of the intestine and rectum. Furthermore, Smith[18] noted that the Japanese have higher death rates than either whites or nonwhites from cancer of the esophagus, stomach, liver, and biliary passages. On the other hand, the Japanese mortality rate from cancer of the intestine, including the rectum, is lower than in whites. Also, foreign-born Japanese have a higher stomach cancer mortality rate than do those born in the U. S. This suggests an environmental factor, as does the observation that the incidence of cancer of the liver is higher among male Chinese of the United States than among those of Hawaii.[17] Lombard and Doering[13] found a definite difference in cancer between foreign and American-born, and correlated this variation to nativity in Massachusetts with the social class in England, thus providing further evidence for an environmental influence, especially the socioeconomic status.

Dorn and Cutler,[5] in their analysis, noted that white and nonwhite men and women in the lowest social class had an incidence of stomach cancer greater than 50 per cent in excess of that found in the highest social class. Cohart[2] noted a similar relationship for gastric cancer in New Haven, Conn., but could not demonstrate the relationship in cancer of the colon, rectum, esophagus, and pancreas.[3] Similar findings are reported

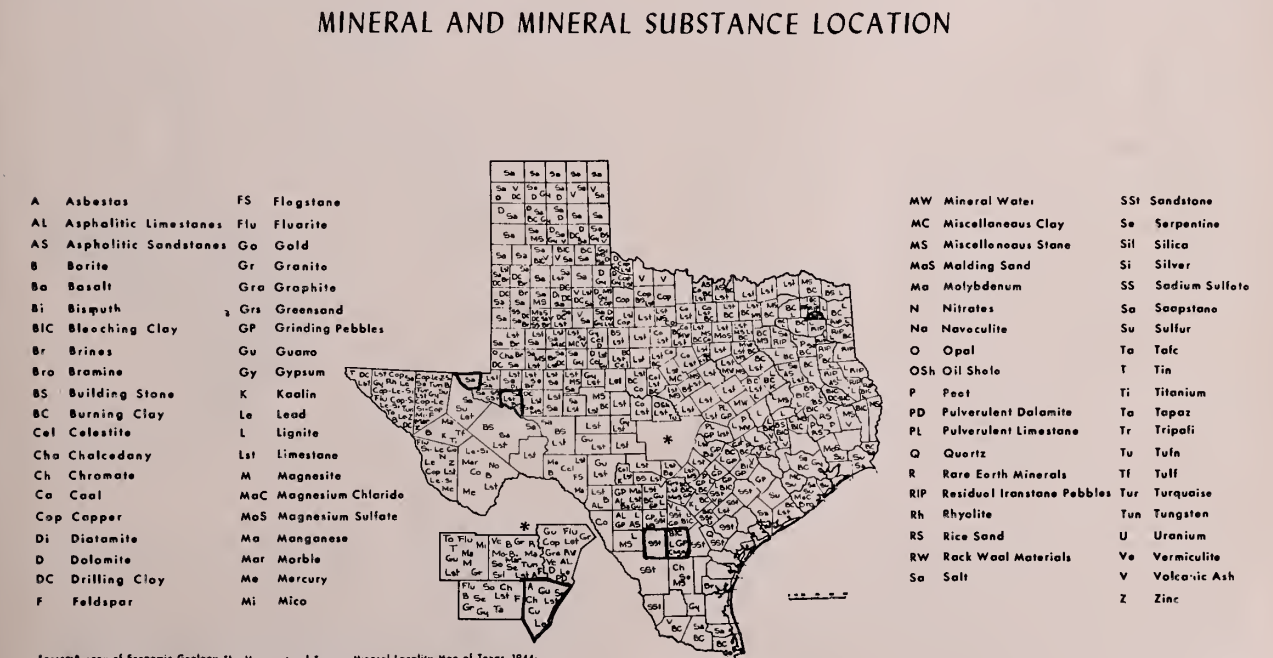

Fig. 6. Location of minerals and mineral substances.

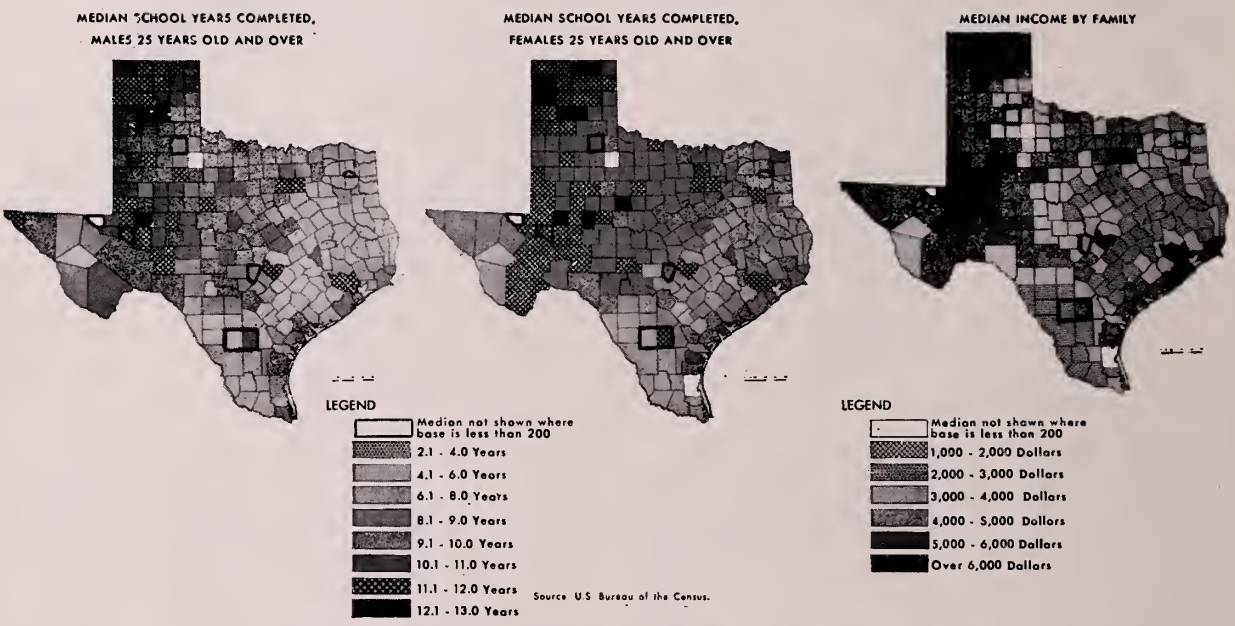
MEDIAN SCHOOL YEARS COMPLETED, MALES 25 YEARS OLD AND OVER
MEDIAN SCHOOL YEARS COMPLETED, FEMALES 25 YEARS OLD AND OVER
MEDIAN INCOME BY FAMILY
LEGEND
Median not shown where base is less than 200
2.1 - 4.0 Years
4.1 - 6.0 Years
6.1 - 8.0 Years
8.1 - 9.0 Years
9.1 - 10.0 Years
10.1 - 11.0 Years
11.1 - 12.0 Years
12.1 - 13.0 Years
Source U.S Bureau of the Census.
LEGEND
Median not shown where base is less than 200
1,000 - 2,000 Dollars
2,000 - 3,000 Dollars
3,000 - 4,000 Dollars
4,000 - 5,000 Dollars
5,000 - 6,000 Dollars
Over 6,000 Dollars

Fig. 7. Characteristics of education and income of population by counties.

from England and Wales by Logan[12] and from Denmark by Clemmesen and Nielsen.[1] However, the latter authors believed the variation was not great enough to lend any solid support to its reality. Contrary to the English findings, the Danes did not find an opposite tendency for intestinal cancers to occur with greater frequency in the upper classes. Graham and associates[7] also reported a similar socioeconomic relationship in Buffalo. No socioeconomic relationship was observed in Texas for G.I. cancers (Fig. 7).

Graham and Lilienfeld,[8] by analysis of twins and family members, found a suggestion of familial tendency for cancer of the stomach that was site specific. Macklin[14] noted that cancer of the stomach was significantly more frequent in relatives of patients with gastric cancer as well as in patients and their relatives with cancer of the large intestine. He suggests that both types of cancer have specific site factors for induction not influenced by the development of the other type. Wolf[21] made similar observations on cancer of the large intestine. He concluded that the organ specificity is a genetic component similar to that observed for cancer of the stomach and breast, since the incidence of other types of cancer in relatives of patients with cancer of the stomach is not increased as compared to a

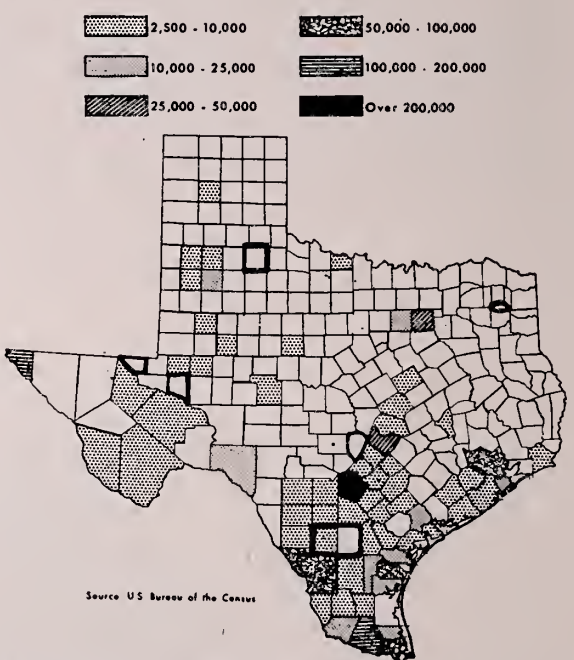

Fig. 8. Counties having more than 2,500 white persons with Spanish surnames (1960).

control group.

Variation in incidence of the gastric cancer in relation to ethnic groups has also been observed. In Canada, the provinces with the highest proportion of persons of French descent have higher stomach cancer mortality than the other provinces, with the exception

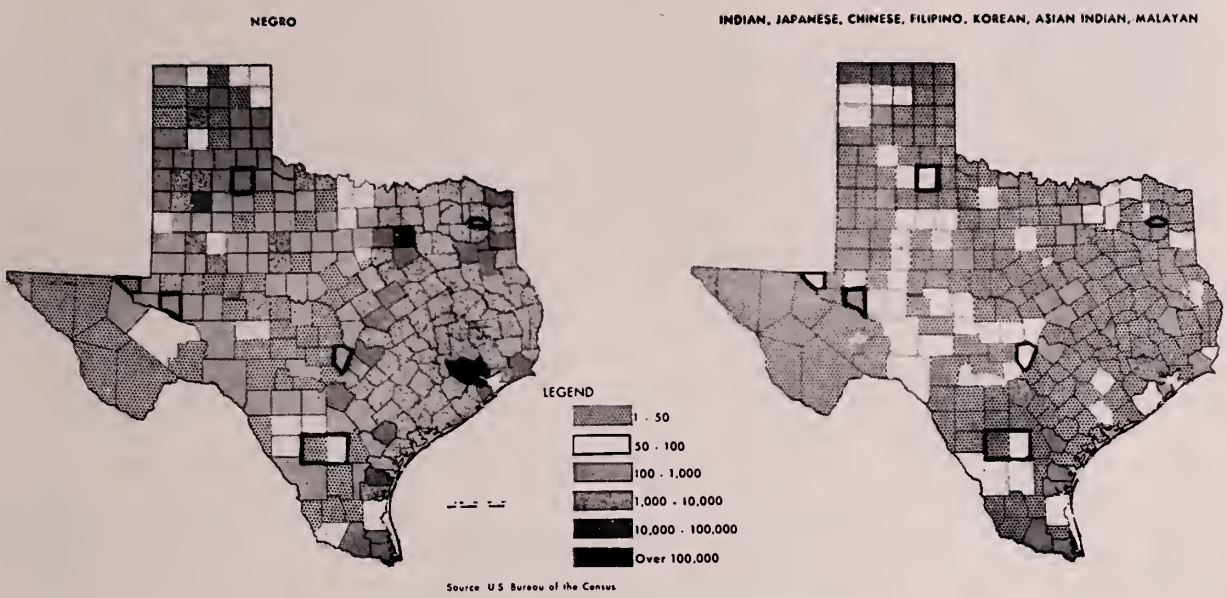

Fig. 9. Nonwhite population (1960).

of Nova Scotia.[16] This is in agreement with the observation of Lombard and Doering[13] that French Canadians in Boston had higher death rates from this cause. No ethnic group relationship of G.I. cancer deaths in Texas could be detected (Figs. 8, 9).

Our failure to demonstrate a correlation between the distribution of G.I. cancer deaths in the different counties in Texas and the factors that have been previously studied may be the result of our inability to compare the distribution by *specific* anatomic site. Unfortunately, the specific site of the G.I. cancer is not available in the vital statistics tables for individual counties.

Summary

In the search for possible etiologic factors, the geographic distribution of G.I. cancer deaths was studied in Texas. The incidence of G.I. cancer mortality by counties varies from 0 to 28.5 per cent. Although significant differences were found in certain counties between the observed and age-predicted mortality, comparison of this distribution pattern with many different factors showed no obvious correlation. Since evidence from other reported studies suggests anatomic site specificity, further analysis of G.I. cancer deaths by organ will be necessary before any definite conclusion, regarding lack of correlation with known factors in Texas, can be made.

The Computer Center, University of Texas Medical Branch, and Dr. A. W. B. Cunningham, associate professor of pathology, Galveston, provided analysis of these statistics, and the book "Atlas of Texas" by Professors S. A. Arbingast and L. G. Kennamer of the Bureau of Business Research, University of Texas, Austin, was the main source of geographic distribution maps.

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U. S. Cigarette Report Carried by Russian Press

U. S. Surgeon General Luther Terry's report on the hazards of cigarette smoking has caused unrest in the whole world, according to recent articles in the Soviet press.

Radio Liberty's research staff reports that the weekly Soviet magazine *Nedelya* carried an article dealing with the report on smoking under the headline "Health Service," with these remarks:

> The script against smoking contains 378 pages and 170,000 words. It was recently put together by a special commission of the United States and published.
>
> The activity of the Commission was surrounded by great secrecy. The chief of the team of assistants was dismissed before he had finished the job because he gave a short interview.
>
> Why was the secret so carefully kept?
>
> In the U. S. A. there are 180,000,000 people, of which 70,000,000 are smokers. Four million Americans make their living in connection with the planting of tobacco, the manufacturing and selling of tobacco products. Three billion dollars are taken in annually by the American treasury from tobacco products.
>
> Finally, the political motive: in seven Southern states, the income of the farmer is derived up to 50 per cent from planting tobacco and these states have 73 representatives in the electoral college which will choose the new President of the United States.

The Soviet news agency Tass, in carrying the subject further, recently distributed an article asserting that about 60,000 Americans will die yearly as a result of heart attacks connected with smoking. Mortality of cigarette smokers, it said, is three times that of nonsmokers or of pipe and cigar smokers.

Surgical Management of the Dumping Syndrome

George L. Jordan, Jr., M.D.

Four approaches are described for managing this postgastrectomy syndrome.

CLINICAL MEDICINE

THE DUMPING syndrome may be defined as a physiologic abnormality that may occur following surgical procedures which disturb normal function of the pylorus. It is most commonly observed following subtotal gastrectomy and is characterized by symptoms which appear during or immediately following a meal.[3] There are gastrointestinal and vasomotor components. The gastrointestinal symptoms include nausea, vomiting, and, occasionally, diarrhea. In some patients, crampy abdominal pain is experienced, but this symptom is relatively uncommon. The gastrointestinal symptoms are, therefore, not significantly different from those which may occur in other postgastrectomy states. Characteristic of the syndrome is the vasomotor component consisting of weakness, sweating, palpitation, and a feeling of warmth or coldness. Physical examination discloses an increase in pulse rate, and there may be a slight rise in blood pressure. Symptoms appear only in the postprandial period and are most often provoked by the ingestion of foods containing milk in a liquid form, or foods which have a high carbohydrate content. It is important to differentiate this syndrome from others that may occur following gastric surgery, for proper treatment depends upon an accurate diagnosis. The diagnosis will usually be made correctly if a detailed history is obtained and the patient is observed during a symptomatic period; however, confusion with other postgastrectomy syndromes may exist. There are a number of physiologic tests which may aid diagnosis. Postprandial changes in the electrocardiogram and a postprandial fall in blood volume occur consistently and are readily determined.[16, 18]

The pathogenesis of this syndrome has not been completely delineated. Distention of the jejunum due to rapid passage of hyperosmotic material into the bowel with stimulation of nervous reflexes, adrenocortical dysfunction, a fall in blood volume, a fall in serum potassium concentration, hyperglycemia, decreased bile excretion, jejunitis, allergies, and psychoneurosis have all been incriminated.[3, 6, 14, 15, 16, 18] Most of these theories are untenable. Stimulation of the gastrointestinal tract by rapid filling and the resultant fall in blood volume appear to play a role, but these changes are not sufficient to explain all the observed physiologic alterations. It is likely that some substance, possibly serotonin or a related hormone, is secreted by the intestine and is responsible for the vasomotor symptoms.[10] Regardless of which factor is of greater significance in producing symptoms, the initiating action is the passage of undigested food into the jejunum. It can readily be demonstrated that certain foodstuffs, such as milk and hypertonic sugar solutions, are more stimulating than others; and it can be readily demonstrated that the volume of material present in the jejunum at any given time is of significance.[7, 16] Consequently, from a surgical standpoint, procedures designed to allow a slow and gradual emptying of the gastric remnant could be anticipated to be associated with the lowest incidence of symptoms.

Through the years, a great number of technical procedures have been advocated for the surgical treatment of duodenal ulcer in an attempt to give the highest incidence

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of relief of ulcer symptoms and the lowest incidence of postgastrectomy sequelae. The most widely used method for reconstruction following gastric resection is anastomosis of the gastric remnant to the jejunum after closure of the duodenal stump—the Billroth II procedure. The anastomosis has been performed in several ways, placing the jejunum posterior to the colon or placing it anterior to the colon. Some surgeons anastomose the entire transected portion of the gastric remnant to the jejunum whereas others close a portion and perform a smaller anastomosis. The advantages of placing the stoma on the greater curvature over those of placing it on the lesser curvature is debated. The afferent loop has been brought to the lesser curvature, as well as to the greater curvature of the stomach. At times, attempts have been made to create a diaphragm to slow the emptying of food from the gastric remnant. Despite various claims of the advantages of specific techniques of reconstruction, a critical review of the problem does not indicate that any type of gastrojejunal anastomosis has a great advantage over others in reducing the incidence of recurrent ulceration or in reducing the incidence of postgastrectomy sequelae, so long as the afferent and efferent loops empty readily and the size of the stoma is greater than the size of the lumen of the jejunum to which the anastomosis is made. Regardless of the size of the gastrojejunal anastomosis or its location, emptying into the jejunum cannot proceed at a more rapid rate than the jejunal size permits.

Gastrointestinal continuity may also be reconstituted by gastroduodenostomy—the Billroth I procedure. There is fairly general agreement that this method of reconstruction has definite advantages in eliminating certain postgastrectomy sequelae, particularly afferent and efferent loop obstruction. Furthermore, it decreases the fecal loss of fat and nitrogen as compared to the Billroth II procedure. Some authors maintain that it is also associated with a lower incidence of dumping syndrome than the Billroth II procedure, but there is not general agreement on this point.[5,9,20] This writer's experience would indicate that the total incidence of the dumping syndrome is not less following the Billroth I procedure, but there is probably a lower incidence of severe symptoms. This procedure, therefore, is the treatment of choice following resection for gastric ulcer, as marginal ulceration rarely occurs. When it is used for the treatment of duodenal ulcer, however, the incidence of stomal ulceration is prohibitively high.[5] Some studies currently reported would suggest that the combination of vagotomy with gastrectomy may eliminate this high incidence of marginal ulceration, but additional experience is needed before final conclusions are drawn.[9]

In recent years, a number of variations in technique have been advocated in an attempt to reduce the incidence of postgastrectomy sequelae. One of the most interesting has been the attempt to preserve pyloric function. Owen Wangensteen, several years ago, advocated the excision of 75 per cent of the stomach, removing the midportion of the stomach with an anastomosis of the cardia to the antrum. As originally performed, this procedure transected the vagus nerves, and pyloroplasty was required to allow emptying of the gastric remnant. The procedure proved successful in the control of duodenal ulcer, but it was not attended with a lower incidence of postgastrectomy symptoms than other techniques in use. In an attempt to improve this procedure, some investigators performed a meticulous dissection of the vagus nerves, preserving the fibers supplying the pylorus, thereby obviating the need for pyloroplasty. Experiments in dogs suggested that this technique would prevent postgastrectomy symptoms.[6] A recent report by Ferguson, however, describing the experience with a large number of patients treated in this manner indicates that the incidence of postgastrectomy symptoms has been as great as following other techniques of gastrectomy.[4] On the basis of current concepts of this syndrome the rationale of the procedure appears excellent, and it is disappointing that the procedure has not solved the problem.

A second technique that has been of great interest in recent years is the construction of a small gastroenteric stoma, one which is significantly smaller than the lumen of the bowel into which the gastric contents will be emptied. Such procedures have been performed as a primary method of reconstruction following gastrectomy. The small stoma does slow emptying from the gastric remnant and does in fact decrease the incidence of postgastrectomy sequelae.[1] Unfortunately, the very small stoma may cause gastric retention, and in some patients obstruction

has been sufficiently severe that reoperation and the construction of a larger anastomosis has been necessary.

A third method of reconstruction that has been of interest in recent years has been the creation of a jejunal pouch at the gastroenteric stoma. A few years ago this concept was investigated, creating a jejunal pouch with a relatively large gastroenteric stoma. It was hoped that this pouch would serve to increase functional gastric capacity and would serve as an area where better mixing of bile and pancreatic juices could occur prior to passage of food into the distal jejunum. This procedure was performed in only a few patients, however, because it became readily apparent that the procedure did not significantly decrease the incidence of the dumping syndrome.

A fourth technique that has had some popularity in England and the Scandinavian countries has been the use of an interposed isoperistaltic segment of bowel between the gastric remnant and the jejunum or duodenum.[8] An extremely low incidence of dumping syndrome has been reported following this technique, but it is much more complicated than the classic procedures.

There are those who perform vagotomy and gastroenterostomy or vagotomy and pyloroplasty rather than any type of resection to minimize postgastrectomy sequelae, but these procedures are less successful in the control of the ulcer problem, and they do not completely eliminate the dumping syndrome.

The writer's experience indicates that the dumping syndrome occurs in approximately 36 per cent of patients following the Billroth II subtotal gastrectomy, but the syndrome is not usually disabling and is easily controlled by nonoperative means. In a study of 400 patients subjected to gastrectomy, only 2 per cent had severe symptoms.[11] Consequently, the use of any special technique which increases the magnitude of the surgical procedure or is attended by an increased incidence of complications, is of doubtful value as a routine procedure in the treatment of benign ulcer disease. Special surgical techniques should be reserved for patients who have definite symptoms of dumping and who fail to respond to conservative management. Most symptoms are easily controlled by the patient without special instructions from a physician, by slight alterations in their diet or simple maneuvers to slow gastric emptying.[2,7,11] This is well illustrated by a patient who underwent operation a few years ago. When first seen in the follow-up clinic, he complained of the typical symptoms of the dumping syndrome. On a return visit six months postoperatively, however, he denied postprandial symptoms and stated that he had had an excellent result from his operation. He repeatedly stated that he was able to enjoy any food without difficulty. Only after specific questioning was it possible to elicit an accurate history. This patient had learned to lie down for 30 minutes immediately after each meal. This he did regularly, and thereby had complete control of symptoms. He stated, however, that if he failed to lie down immediately following ingestion of a meal, he had symptoms to the same degree as previously. This man, then, had rapidly learned to control his symptoms by a simple maneuver that slowed emptying of his gastric remnant.

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A second group of patients have symptoms not controlled by minor dietary alterations, and these patients may require the aid of a physician to gain symptom relief. A number of simple therapeutic measures may be instituted by the physician for patients who fail to control their own symptoms satisfactorily. These include the prescription of a dry, multiple small feeding diet in which the patient limits the intake of food at any one meal and ingests the food in a consistency which will not pass rapidly through the gastric stoma. Liquids are taken between meals. Stimulating foods, such as milk and foods having a high carbohydrate content, are eliminated. This diet alone will control symptoms in many patients. Assuming the recumbent position in the postprandial period will relieve symptoms in other patients, as in the patient noted earlier, because this position does decrease the speed of emptying of the gastric remnant.[2] In patients having more severe symptoms, antispasmodics may be used to slow the motility

in the bowel, thus decreasing the speed of gastric emptying, as well as decreasing the outpouring of fluid into the lumen of the bowel.[11,16]

If the blood volume is low, transfusions may aid in the relief of symptoms.[12]

A number of other agents have been advocated for use in this syndrome, particularly the use of potassium salts or the local oral anesthetic agents.[3,14] These have not proved of value in the hands of most observers. The most recent medications believed to be of help are the antiserotonin agents, and some investigators have stated that the use of these agents has virtually eliminated the need for surgical treatment of this syndome.[10]

Certainly, the majority of patients will obtain symptom relief by conservative management and, as noted, surgical treatment should be reserved for the occasional patient who continues to have disabling symptoms despite attempts at dietary and medicinal management. When surgical management is necessary for control of severe symptoms, a number of procedures may be considered.

The most popular surgical maneuver has been the conversion from a Billroth II to a Billroth I procedure when this is possible. As noted previously, this technique has a good rationale, and many investigators have reported good results with it.[20] A second procedure which may be used is the creation of a very small stoma. As stated previously, we do not believe that this is justified as a primary procedure, but it may prove of value in the occasional patient with severe dumping symptoms.[1]

In recent years, our interest has centered on the use of a reversed intestinal loop in the treatment of the syndrome, and it now appears to be the most promising of the surgical procedures available. The properties of a reversed intestinal loop have been studied by several investigators during this century. It has been demonstrated that such a segment retains its peristaltic gradient, and thus acts to impede the passage of bowel contents during the contraction phase. Short loops, therefore, temporarily and intermittently delay passage of intestinal contents, while long segments produce a true, symptomatic obstruction, because passage completely through a long segment will not occur before contraction begins.

These data suggested that a short, reversed loop, interposed between the gastric remnant and the jejunum, would function to some degree as a valve, allowing a slow, gradual emptying of the stomach pouch. Original investigations in the dog were performed to study the effect of this procedure upon fat and nitrogen loss in the stool following subtotal gastrectomy. Roentgenographic studies after the ingestion of barium demonstrated that the interposed reversed loop did delay emptying of the gastric remnant and resulted in a prolonged, gradual emptying similar to that seen in animals with an intact stomach. The initial objective was thus achieved, but unfortunately, metabolic studies failed to demonstrate a difference in fecal fat and nitrogen loss as compared to control animals. On the other hand, postprandial studies of the electrocardiographic tracings and blood volume changes proved that the slower emptying prevented the physiologic changes usually seen in the dumping syndrome, and this difference was marked when compared to control animals having gastrectomy without a reversed segment.[19] As a result of these studies, an investigation of this procedure in humans seemed justified, and now we have utilized the procedure in the treatment of two patients with severe dumping syndromes which were totally unresponsive to nonoperative management.

Case Reports

Case 1.—The first patient, previously reported in detail,[13] was a 55-year-old white male who had a massive hemorrhage from a duodenal ulcer in 1940. This was successfully treated by nonoperative management, but symptoms of abdominal pain recurred intermittently, and the patient experienced several episodes of gastrointestinal bleeding during the subsequent 14 years. A subtotal gastrectomy was performed on Jan. 4, 1954, and with his first postoperative meal he developed nausea, vomiting, pallor, palpitation, weakness, sweating, and dizziness. These symptoms recurred with almost every meal, and during the ensuing two years the patient lost 63 pounds in weight, thereafter maintaining his weight at 121 pounds. Attempts to obtain relief by postprandial recumbency, utilization of a dry diet, antacids, tranquilizers, and antiemetics had all failed to give relief. Symptoms after lunch were so incapacitating that the patient often could not work. He was hospitalized in September of 1961 for further evaluation of his problem. Roentgenographic studies demonstrated very rapid emptying of the gastric remnant in the upright position, and pre- and postprandial determinations of plasma volume, electro-

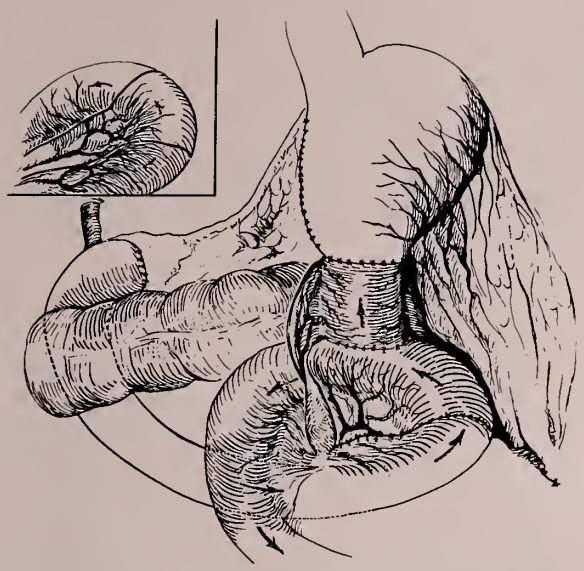

Fig. 1. Diagram illustrates the surgical technique employed. A 6 cm. segment of jejunum was isolated ond interposed between the gastric remnant and the jejunum. The suture line in the jejunum represents the point of reconstruction of jejunal continuity after isolation of the interposed segment.*

cardiographic recordings, digital blood flow determinations, and serum potassium concentrations demonstrated the typical changes seen in association with the dumping syndrome. The glucose tolerance test disclosed rapid glucose absorption.

On Sept. 28, 1961, a 6 cm. antiperistaltic segment of jejunum was placed between the gastric remnant and the remainder of the small bowel (Fig. 1). On the sixth postoperative day, the patient ingested a liquid diet without symptoms and stated that it was the first time since 1954 that he had eaten comfortably. He was readmitted to the hospital on Dec. 26, 1961, three months postoperatively, for repeat studies. He had gained 21 pounds in weight and stated that he had been asymptomatic except for some belching with very large meals. Roentgenographic studies now demonstrated a slow, gradual emptying of the gastric pouch, and physiologic studies demonstrated a return to normal values in all tests except one—the blood volume study. This latter test, though not completely normal, showed obvious improvement as compared to that recorded preoperatively. The patient was last seen in January of 1963, 15 months postoperatively. He had continued to eat a normal diet without difficulty, and he had maintained his weight gain. At this time all physiologic tests, including the blood volume test, were within the normal range (Figs. 2, 3).

Case 2.—The second patient developed symptoms of the dumping syndrome following a hemigastrectomy and vagotomy for duodenal ulcer. His history was similar to that of the first patient treated, but in addition, he experienced diarrhea. This latter symptom was thought to represent a postvagotomy

*From Willms, R. K., et al.: Effect of a Reversed Jejunal Segment upon Gastric Emptying, Nutrition, and Plasma Volume Following Subtotal Gastrectomy. Surg. Forum 12:317, 1961.

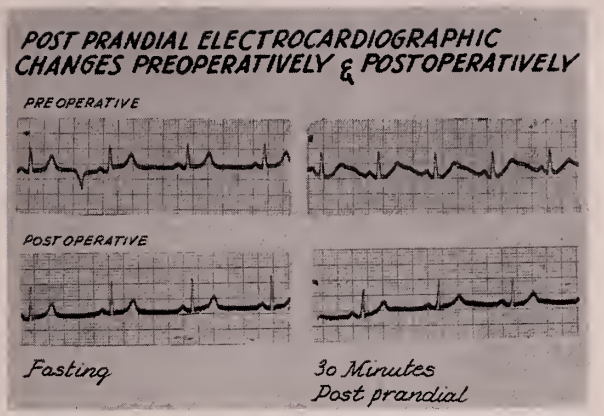

Fig. 2. Electrocardiographic tracings show that in the preoperative period noticeable chonges in the electrocardiogram occurred. In the postoperative period the electrocordiogram remained normal in the postprondial period.†

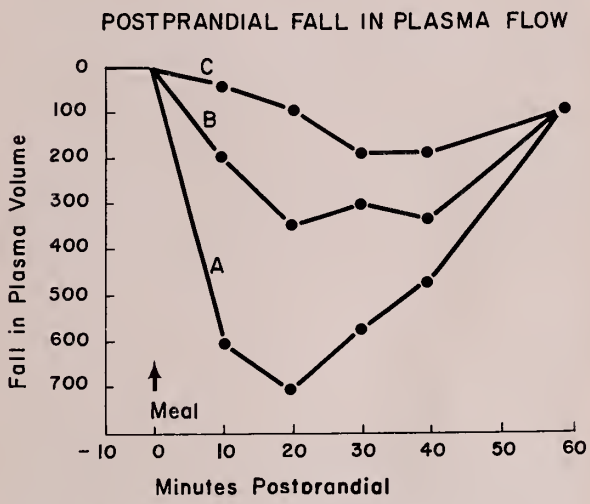

Fig. 3. A comparison of postprandial chonges in blood volume during three periods is made. Curve A illustrates the extreme postprandial fall in blood volume preoperatively. Curve B represents the fall in blood volume three months postoperatively, showing a return towards, but not to, normal. Curve C was recorded 15 months postoperatively and is within the range of normal.

effect rather than a part of the dumping syndrome. This patient was also treated surgically by interposition of a 6 cm. reversed jejunal segment between the gastric pouch and the small bowel. He was relieved of his upper gastrointestinal and vasomotor symptoms following surgery, and physiologic studies have shown a similar improvement as noted in the first patient. He has not, however, experienced any significant improvement in his diarrhea.

The results obtained in these patients have been excellent for both had severe, intractable

†From Jordan, G. L., Jr., et al.: Treatment of the Postgastrectomy Dumping Syndrome with a Reversed Jejunal Segment Interposed Between the Gastric Remnant and the Jejunum. Amer. J. Surg. 106:451 (Sept.) 1963.

symptoms with typical physiologic changes in the preoperative period, and they can now eat a normal diet comfortably.

Different types of interposition have been used by others, also with good results. Poth, using a slightly different technique was the first to utilize the concept of an antiperistaltic segment of bowel for the dumping syndrome, and his published reports to date indicate results similar to the writer's.[17] Hedenstedt has interposed isoperistaltic loops. He reports that these also have a valve-like action and are effective in preventing precipitate gastric emptying.[8] Additional experience will be required to evaluate this procedure fully, to compare various techniques, and to compare the results of isoperistaltic and antiperistaltic loops, but the results in these two patients are so gratifying that this is considered the procedure of choice at the present time. The use of the interposed reversed segment is not recommended as a routine procedure following gastrectomy in the treatment of ulcer disease, but should be reserved for patients who have intractable symptoms over a prolonged period.

Summary

The postgastrectomy dumping syndrome occurs during or immediately following a meal. It can be accurately diagnosed and differentiated from other postgastrectomy symptoms by appropriate objective measurements. Although this syndrome is common following gastric resection, in most instances it is mild and easily controlled by conservative measures. In only the occasional patient will surgical intervention be justified in an attempt to improve symptoms. The procedures which have been most popular in the past include conversion to a gastroduodenostomy and the creation of a very small stoma. Recently utilized has been a reversed jejunal segment interposed between the gastric remnant and the jejunum in two patients with severe dumping symptoms. In both patients gastric emptying was slowed, and relief of symptoms occurred. The symptomatic relief was correlated with a return of physiologic abnormalities to or towards normal.

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Slim Outlook

From the *Long Island Press:* A Department of Health, Education, and Welfare employee, deciding on a poll while vacationing in New England, asked a sweet old lady of 71: "What do you think of Medicare?" She replied: "I don't know for myself, but I have a friend that tried it and lost 21 pounds."

Tuberculin Test: Some Inherent Limitations

RICHARD D. BATES, M.D., and JOHN S. CHAPMAN, M.D.

Dallas County experience with tuberculin reactions and the problem there of nonspecific cross reaction among the mycobacteria is reported.

RESEARCH & INVESTIGATION

TUBERCULIN has been known to medicine since it was first used as a therapeutic agent by Robert Koch in the latter part of the 19th century. It was Clemens Peter Pirquet von Cesenatico who was primarily responsible for converting tuberculin to a diagnostic tool as a skin-testing agent. When the tuberculin skin test was first introduced in the early years of this century, the incidence of positive reactors was so high that the test was effective only as a survey of the incidence of tuberculosis infection in population groups. As the incidence of tuberculosis infection has declined, this skin test has become effective as a case finding tool and as a diagnostic aid in the individual patient. Even before ten years ago, the frequency of tuberculosis was so high that no opportunity existed to study the tuberculin test itself. Now during the past five years with a significant decrease in the incidence of the positive tuberculin reactors, the reaction itself has come under more critical analysis and study. One of the facets of this renewed look at the tuberculin test has been in the epidemiologic work of Edwards and others,[7] and the supporting experimental work of Palmer and others.[13] They demonstrated that many of the reactions to the intermediate dosage and most of the reactions to stronger test dosage of purified protein derivative (PPD) were not caused by tuberculosis infection but were cross reactions caused by naturally occurring infections by other mycobacteria, the so-called anonymous mycobacteria.

Presented in this article is the result of experience in Dallas County, Tex., with tuberculin reactions, especially as it relates to this problem of nonspecific cross reaction among the mycobacteria. In Dallas County the positive tuberculin reaction rate for high school students fell from 22 per cent in the years 1939-1944 to 8.1 per cent in 1957 in the low income areas. During a corresponding period the tuberculin rate of first grade students in similar areas fell from 12.35 per cent to 3.3 per cent. This first grade rate is still lower among Anglo-Americans and Negroes where incidence is 2.0 per cent and in the higher income groups where incidence is 0.5 per cent. The incidence of positive tuberculin reactions in this area was such that in 1957 a more accurate appraisal of its significance could be undertaken.

One of the first of these Dallas County experiences, a study by Dewlett and others,[6] has been reported previously in the *American Review of Respiratory Diseases.* The contacts of patients with proved open tuberculosis disease were tested with old tuberculin 1:1000, and 47 per cent showed a reaction greater than 5 mm. The contacts of proved anonymous bacteria pulmonary disease were tested with group-specific antigens prepared by Chapman.[11] Of the contacts of group I or photochromogen disease, 36 per cent had significantly positive skin tests. The pattern of distribution of group I reactors by size of induration showed two distinct peaks in the distributional curve. The first peak at approximately 3 mm. reaction and the second at 8-10 mm. reaction. The question this raises is whether these small reactions represent recently acquired infections, or old partially lost reactions, or possibly cross reactions with some other mycobacterial products.

The next step was to find an adequate control for this study. Dewlett[6] had studied a Dallas County orphanage with 506 children ages 2 to 20 years. These were all originally skin tested with OT 1:1000, photochromogen (PC) 1:1000, scotochromogen

1:1000, and nonchromogen 1:1000. Of the 506 children tested, 126 showed positive reactions to one or more of the antigens. The positive reactors were then retested with OT 1:100 and histoplasmin antigen 1:100. Only 3.2 per cent of these originally tested reacted to the OT 1:1000 compared to 22 per cent reactors to photochromogen, 7.2 per cent to scotochromogen, and 3.2 per cent to the nonchromogen antigens. Of those retested with OT 1:100 47.2 per cent showed reaction of 4 mm. or more. Of those retested with histoplasmin 1:100, 30.7 per cent showed similar reactions, generally over 10 mm. The distribution of the size of reactions to the OT 1:1000 and to PC 1:1000 closely followed the usual bell-shaped distribution curve for both antigens. In correlating the reactions to both OT and PC 1:1000 four distinct groups were found. Those who reacted to neither antigen consisted of 380 of the 506 children tested. There were 95 who reacted to the PC 1:1000 but not to OT 1:1000. There were only seven reactions to OT 1:1000 who did not react to PC 1:1000. These three groups respectively apparently represent those children with no previous infection with either tuberculosis or photochromogen mycobacteria, those with previous photochromogen infection, and those with tuberculosis infection at some previous date. The last group consisted of nine children with significant and approximately equal reaction to both OT 1:1000 and PC 1:1000. The exact status of this last group cannot at present be satisfactorily explained beyond their representing a group who have had infection with photochromogen, tuberculosis, or some other mycobacterium. Another interesting facet in this problem was the result of x-ray examinations of the chest of all reactors to any antigen. From this group, 29 showed a calcified primary lesion. Of these, seven reacted to OT 1:1000, 28 to PC 1:1000, and 13 to histoplasmin 1:100. Of these only one reacted solely to OT, four reacted solely to PC, none reacted solely to histoplasmin; the remainder reacted to various combinations of the five different antigens used.

Dr. Bates is an Irving pediatrician.

The results of these studies have shown a 22 per cent control reaction to PC 1:1000 compared to 36 per cent by contacts of photochromogen infections, a 3.2 per cent control reaction to OT 1:1000 compared to 47 per cent reaction by contacts of tuberculosis infections, an inordinate degree of cross reaction between OT and PC reactors with only 1.3 per cent reacting only to OT and 18.7 per cent reacting to PC, and a high percentage of calcified primaries were associated with only the photochromogen or group I reaction.

Another study to be published soon was carried out locally by Potts.[14] He studied the skin reaction of 90 patients in Freeman Clinic, aged three months to two and one-half years, found to be reactors on routine testing. These were all sole reactors (Mantoux 1:1000 over 6 mm.) out of a group of siblings with no definite contacts with a known tubercular individual. Of these, 61 patients (65 per cent) reverted to a negative Mantoux on retesting at a later date. These all showed a good reaction to the photochromogen antigen. There were no reverters on retesting where there was contact with a known case of tuberculosis.

Another recent combined study by the Dallas Tuberculosis Association, the Dallas Tuberculosis Case Finding Project, and the Dallas County Health Department, was directed toward a better understanding of the small tuberculin reaction. This consisted of the administration of a Heaf type tuberculin test to 745 children in grades one through six with further study of the 1 and 2 plus Heaf reactors. Of those studied, 70 were positive Heaf reactors: five showed 3 plus or better reaction, eight showed 2 plus reaction, and 57 showed 1 plus reaction. This represented 9.4 per cent positive reactors of 1 plus or greater. Of these, seven with 2 plus and 53 with 1 plus reactions were retested with OT 1:1000 and PC 1:1000. It was found that 37 (or 61.7 per cent) of these were negative to OT, with 15 (25 per cent) showing induration 5 to 9 mm. and eight (13.3 per cent) over 9 mm. On the other hand 100 per

cent showed in excess of 7 mm. induration for PC 1:1000 with 88 per cent (53) exceeding 9 mm. In comparing the relative size of the OT and PC reactions, they found here, as in the case of the control group tested by Dewlett, that the PC reactions far exceed those of the OT. Of those with OT induration, less than 6 mm. (45), 86.6 per cent (39) showed over 9 mm. reaction to PC antigen. Of the OT reactors with up to 9 mm. induration (52), 90 per cent (47) showed over 9 mm. induration to the PC antigen. Or more significantly, of those with OT induration of 10 mm. or more (eight), 100 per cent (eight) showed PC induration equal to or in excess of that of the OT induration. It is believed not too great an assumption that these small Heaf reactors are primarily caused by cross reaction following photochromogen disease.

The photochromogen or group I anonymous mycobacterium is the predominant organism in Texas. A similar relationship undoubtedly exists between the routine tuberculin test and the other anonymous mycobacterium in other areas, as shown by Palmer and others with respect to group III reactors.[7,10,12] It is believed that the method as employed by the Children's Medical Center Clinic of retesting with OT 1:100 or intermediate PPD only without advancing to greater concentration has allowed some of these observations to be made. It is believed also that the use of this method of retesting will allow for the separation of the reverter and will reduce the number of cross reactions originally found with positive tuberculin tests. It is also thought that these studies have led to the identification and separation of some other entities that had been included with tuberculous disease prior to this time, namely, (1) the individual with the unstable or reverting tuberculin test and (2) the individual with the small tuberculin test.

These studies support the conclusion that a healthy child with a negative chest x-ray and no known contact but a positive tuberculin test need not be treated with antituberculosis drugs. Similarly, in the case of a healthy child with a reaction only to the second strength PPD or OT 1:100 without clinical or roentgenographic support, the positive tuberculin test may be disregarded so far as a definite diagnosis of tuberculosis is concerned. However, both of these types of patients must be reexamined and retested at frequent regular intervals for other clinical or roentgenographic evidence of disease.

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Kansas Bans Cigarettes on Campus

Sale of cigarettes on all state-operated college and university campuses in Kansas has been banned upon the recommendation to the state's Board of Regents made by Dr. W. Clarke Wescoe, chancellor of the University of Kansas and Dr. Arden Miller, dean and director of the KU Medical Center.

"COTTON FORKER'S INJURY"

Fracture of the Upper Dorsal Spinous Processes

Bill D. Murphy, M.D.

CASE REPORTS

AN UNUSUAL type of injury has been seen in the past two years in Latin American farm workers. In the fall and winter of 1961, four cases of the injury were seen, and in the fall of 1962, six cases were seen. It might be called "cotton forker's injury."

These injuries have occurred in fields where cotton is pulled by stripping machines. The patients were workers who forked cotton toward the back of trailers drawn by tractors having attachments which pull the bolls of cotton and throw them into the trailers. Each patient experienced severe pain in the upper back or lower neck as he exerted to throw the cotton toward the back of the trailer. The pain was usually fairly severe, leaving the individual unable to work after the injury.

Examination of these patients revealed tenderness over the spinous processes of the seventh cervical vertebra (C-7) and the first and second thoracic vertebras (T-1 and T-2). Sometimes there was evidence of crepitation upon pressure on these bones. Some radiation of pain and tenderness to the adjacent muscles was noted, but usually not much. This is in contrast to the frequent injury seen under the same circumstances—the straining of the rhomboid and trapezius muscles which is characterized by tenderness over the involved muscles. Using a special technique which allowed the spinal processes to be seen, lateral view x-rays of the lower cervical and upper dorsal spine, revealed the fracture of the spinous process of C-7, T-1, and T-2 at various levels of the process. Two fractures of C-7, six fractures of T-1, and two fractures of T-2 spinous processes have been seen. Two cases were combination fractures of C-7 and T-1, and one case a combination of fractures of spinous process of T-1 and T-2.

It is surmised that this injury is caused by extreme exertion of the trapezius and rhomboid muscles which have their origin on the spines of the lower cervical and upper thoracic vertebra. Probably the trapezius of one side pulls the spine in one direction or else fixes the spine, and the rhomboids on the other side contract in the opposite direction thereby causing the fracture. None of these patients gave a history of direct blow to the area involved.

Treatment consists of rest and analgesics to relieve the pain, which is usually severe for one or two weeks. However, in one case, the patient continued to work for several days after injury and was not in excessive pain.

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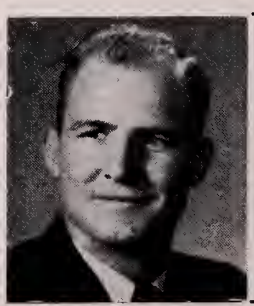
COMMENT

This fracture is described by Watson-Jones, a British orthopedic surgeon, in his book "Fractures and Joint Injuries" and is given the name of "clay-shoveler's fracture." Apparently it is seen rather frequently in Europe among workers who are spading or shoveling heavy clay. Watson-Jones makes a point of the fact that this type of fracture is seldom seen in most countries and that few surgeons have ever seen more than two or three such cases. However, in certain countries of Europe, particularly Germany, large series have been reported.

Dr. Murphy's paper should be of particular interest to those physicians practicing in the cotton growing areas of our state. I think few of us realize that this fracture is so common in Texas.

—FREDERICK C. LOWRY, M.D., Austin.



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Jack A. Barnett, M.D., and C. T. Ashworth, M.D.

Clinicopathologic Conference

Progressive dyspnea brought a 39-year-old woman to Parkland Hospital. She had had rheumatic heart disease and rheumatoid arthritis for many years. Her death occurred suddenly. At autopsy, evidence of still a third major disease was found. What was the cause of death?

Clinical Summary

DR. EDMONDSON: A 39-year-old white woman was admitted to Parkland Hospital for the first time in February, 1959, complaining of progressive dyspnea of four years duration.

At the age of 18 months she had had painful, red, hot, swollen joints which were relieved somewhat by aspirin. The acute episode lasted several weeks, but active disease continued in smoldering form with occasional acute exacerbations until she was 12. The joints most involved were the wrists, metacarpophalangeals, proximal interphalangeals, ankles, and back. At about age 8, her wrist joints began to flex and became progressively more deformed over the years. Red, painless knots occurred transiently about the elbows, usually lasting weeks or months. A tonsillectomy had been done when she was 18 months old.

When she was six, she had scarlet fever and when seven, St. Vitus' dance. At 12, for the first time, she was found to have a "slight heart disorder." At 30, she rather suddenly developed exertional dyspnea, followed by abdominal swelling and pedal edema, but without orthopnea. She was placed on digitalis and salt restriction and gained symptomatic relief. In 1958, at 34, she stopped working because of increasing dyspnea. Two weeks before admission in 1959, the dyspnea increased coincidently with onset of fever and cough with yellow and rusty sputum. She was treated with penicillin, followed by the onset of chest and abdominal rash. She came to Parkland when her dyspnea was unrelieved despite increased digitalis.

Family history showed that her mother, now 56, had a heart attack at 49 and was on digitalis. Her father, uncle, and two sisters had apparent rheumatoid arthritis.

Physical examination revealed an irregular pulse of 60 and blood pressure of 150/70. Moderate dorsal kyphosis and flexion deformity of both wrists were present. The proximal interphalangeal joints were swollen and there was muscular atrophy of the arms, distally. There was trace edema of both ankles and brownish discoloration of the skin. Neck vein distention and right posterior-basilar crepitant rales were described. The point of maximum impulse was in the fifth intercostal space in the anterior axillary line. P2 > A2. A grade 2 diastolic decrescendo murmur was heard in the second right intercostal space which radiated down the left sternal border. A grade 2 holosystolic, coarse, low-pitched murmur was heard at the apex. A grade 2 diastolic apical

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This is the third in the 1964-1965 series of clinicopathologic conferences to be provided by the University of Texas Southwestern Medical School at Dallas. Dr. C. T. Ashworth, chairman of the Department of Pathology, is guest editor and also presents the pathologic findings.

Clinical summary is presented by Dr. E. B. Edmondson of the Department of Medicine; radiologic findings by Dr. Milton L. Wagner, of the Department of Radiology; and clinical discussion by Drs. Morris Ziff, Eugene P. Frenkel, John Baum, and Jack A. Barnett, of the Department of Medicine.

murmur and grade 1 midsystolic aortic murmur were heard. The liver was felt 2 cm. below the right costal margin. Some observers felt the spleen down 2 cm. Electrocardiograph showed atrial fibrillation with bigeminy. Chest x-ray showed cardiomegaly with enlarged left atrium and pulmonary congestion. Hemoglobin was 13.3, erythrocyte sedimentation rate 33 to 22, white blood count 8,200 with normal differential.

The congestive heart failure and presumed digitalis intoxication were treated with potassium chloride and low salt diet. The history of skin rash after penicillin was pursued with 500 units intradermal penicillin skin test, which produced an immediate erythematous area of 4-5 cm. surrounded by pseudopods. She improved in the hospital and was discharged on 0.1 mg. digitoxin and 250 mg. erythromycin per day.

She was readmitted in March, 1959, for left and right heart catheterization with findings of elevated pulmonary artery and wedge pressures with a gradient across the mitral valve. These findings were felt to indicate mitral stenosis, but there was no hemodynamically significant aortic lesion. X-rays showed calcification of the mitral valve. In April, 1959, mitral commissurotomy was done. At surgery an apparent old thrombus was evacuated from the left atrium. The mitral valve was calcified and estimated to have 0.8 sq. cm. orifice. This was enlarged to 3.0 sq. cm. by finger fracture. In spite of a rapidly corrected cardiac arrest during surgery, she was symptomatically improved and x-rays of the chest revealed decreased prominence of the pulmonary vascular volume. Her murmurs were apparently changed with decreased intensity of mitral diastolic murmur. She reverted to atrial fibrillation in February, 1960, and was again converted.

She had repeat left heart catheterization in October, 1961, with documentation of aortic insufficiency and mitral insufficiency. There was felt to be no gradient across the aortic valve. Diminished perfusion of the left coronary artery was described.

In early 1963 she noticed reduction in exercise tolerance and developed intermittent right-sided chest or upper abdominal tenderness and pain. The lungs were clear, and the liver, kidneys, and spleen were not felt. Chest x-ray showed heavy markings in both bases. Hemoglobin was 8.4 and total serum iron was 30 μg. Upper gastrointestinal and gallbladder x-ray studies were within normal limits. The barium enema showed deformity of the cecum.

The patient was admitted for further evaluation in April, 1963. Physical examination was unchanged except that the blood pressure was 100/70. Hemogram revealed normochromic, normocytic anemia, leukopenia, and thrombocytopenia without reticulocytosis. Stools were negative for blood on six occasions. Total serum iron was 38 with total iron-binding capacity of 265 μg. Venereal disease research laboratory test for syphilis was weakly reactive. Total serum proteins were 7.4 gm. with 3.5/3.9 albumin-globulin ratio. Thymol turbidity was 18.3. Direct Coombs and bone marrow aspirate were interpreted as normal with normally budding megakaryocytes being present in reduced numbers, and slight delay of erythrocyte maturation. Stainable iron was present. Skin tests were negative. During the hospital course, the patient ran an intermittent low-grade fever to 101 F. The spleen tip was palpable and several subungual splinter hemorrhages were noted on one occasion. Erythromycin was discontinued and eight blood cultures were obtained with negative results. Repeat barium enema proved no definite cecal abnormality. The patient was discharged after ten days with an impression of hypersplenism secondary to rheumatoid arthritis accounting for the blood abnormalities. The antinuclear test reported after discharge was 4+ positive, undiluted.

She was readmitted in May with complaints of painful, tensely swollen ankles, increased productive cough, fever and continuing right chest pain. Venous pressure was normal. Hepatosplenomegaly without adenopathy was present. She had occasional low grade fever spikes. The legs were elevated and edema cleared with rest and diuretics with relief of leg pain. A lupus erythematosus prep was strongly positive. Five blood cultures were negative while she was off erythromycin.

Increased right chest pain and fever prompted readmission in September, 1963. Decreased breath sounds in the right base with rales and a questionable friction rub were associated with x-ray evidence of haziness in the right costophrenic angle. Blood cultures were negative x3, and sputum was unremarkable. Pain and fever subsided five days after treatment with 1 gm. erythromycin per day.

Her tenth hospitalization in November, 1963, was on the surgery service for treatment of cellulitis of the left ankle. An ulcer of the ankle had not healed with continued therapy on an outpatient basis, and she had developed several superficial ulcers on the right foot. Treatment included erythromycin and streptomycin followed by tetracycline and vigorous local care. She was discharged on Dec. 11 on digitoxin 0.25 mg. daily and soaks for the ulcer.

Her final admission followed gradually increasing dyspnea for two weeks. One week prior to this admission her cough increased and became productive of yellowish sputum. Associated with this was bilateral pleuritic anterior chest pain. Two days previous to admission she began coughing up occasional bright red blood. She had fever without chills, low back pain, and vague midepigastric discomfort. Examination showed blood pressure 120/85, pulse 150, respiration 36, temperature 101.8 F. General appearance was that of a malnourished, chronically ill white female in moderate respiratory distress. The neck veins were distended. Chest revealed increased vocal fremitus without decreased breath sounds, rales, or a rub in the right lateral base. Examination of the heart revealed the point of maximum impulse in the sixth intercostal space medial to the anterior axillary line. Sounds were difficult to evaluate at rate of 150. A grade 4 holosystolic murmur was heard at the apex. Liver and spleen were both felt 4 to 5 cm. below the costal margins and were tender. A 2+ pitting pedal edema was present and two ulcers were present over the lateral and medial malleolus of the left ankle.

Chest x-ray was interpreted to show slightly more infiltrate in the right base than previously described. Sputum smear revealed numerous gram-positive diplococci and leucocytes. Hemoglobin was 14.6, hematocrit 47, white blood count 12,250 with 84 neutrophils and 16 lymphocytes. Electrocardiograph showed an atrial flutter with 2:1 conduction. Venous pressure was 17.5 cm., and circulation time was 45 sec.

The patient was placed on bed rest and given 0.25 mg. digoxin intravenously with the pulse subsequently falling to 120. Tetracycline 500 mg. was instituted every six hours along with intermittent positive pressure breathing. The respiratory distress was significantly improved. At 4 a.m. on Dec. 30, the temperature spiked to 103 F. At 7 a.m. the patient was unexpectedly found without respirations or pulse and was pronounced dead.

TABLE 1. *Additional Laboratory Findings.*

Cardiac Catheterization	Systolic	Diastolic	Mean	
March 1959				
Pulmonary artery	60	29		
Right ventricle	78	8		
Right atrium			8	
Wedge			23	
Aortic	117	79		C.O.=3.18
Left ventricle	122	0		C.T.=2.25
Oct. 1961				
Left ventricle	136	16	53	
Aortic (after dye injection)	159	69	100	

Miscellaneous
May 1963
Reiter protein complement test weakly reactive; fluorescent treponema antibody nonreactive.
April 1963
Protein electrophoresis: total protein, 7.2 gm.; albumin, 33%; alpha 1 globulin, 5%; alpha 2, 9%; beta, 12%; gamma 40%. Direct Coombs ±: cold agglutinin + 1:8; Reiter's anticomplementary; BSP no retention.

Radiologic Findings

DR. WAGNER: The first films were available in January, 1963. They show cardiomegaly with multichamber prominence. Both atria are enlarged and the apex is elevated. I suspected biventricular, but mainly right-sided involvement. The main pulmonary artery and intrapulmonary vasculature are increased in size and mainly venous in nature. The aorta is normal. There are Kerley's lines at the bases. No confluent infiltrations or effusions are seen. Amorphous calcifications are noted within the cardiac shadow and appear to be in the mitral valve. There is evidence of previous resection of the left fifth rib posteriorly; otherwise, the bony framework is normal.

Films of the abdomen were obtained prior to the barium enema studies in April and reveal a normal sized liver shadow, but there is some splenic prominence. The bowel pattern appears normal and there is no evidence of mass lesions. There was some question of abnormality within the cecum on the initial barium enema study, but follow-up examination failed to reveal any irritability or specific lesions within the cecum or terminal ileum.

In May of 1963 there was little change in

the appearance of the lung fields, except possibly some increase in the vascular volume. From this point on, it would appear that the findings are mainly those of a patient in and out of failure. In August of 1963, for the first time, a hazy infiltrate in the left base was observed. There has always been some irregularity along the left heart border which could be secondary to pericardial adhesions from the previous surgical procedure. Minimal blunting of both costophrenic angles may be noted. In September, 1963, the findings of congestive failure were again observed. The films of November, 1963, reveal progressive cardiomegaly with vascular congestion. Minimal bibasilar infiltrations were seen. The final chest films were made in December and show little change except a density in the left suprahilar region which has the appearance of an infiltrate.

Films of the wrists and angles are of interest. There are bilateral flexion contractures of the wrists at the radiocarpal joint. Complete fusion of most of the carpal bones is seen and these, in turn, are fused with the bases of the metacarpals. The bones appear very well mineralized in this patient. There are contractures of several of the fingers but little in the way of joint narrowing. Where there is narrowing, it appears mainly in the proximal interphalangeal regions. The same findings are true in the feet. Some of the tarsal bones are fused and there is superimposed osteoarthritis in several intertarsal joints. Diffuse vascular calcification is observed about the ankles and feet. The last films of the ankles were made in September, 1963, and reveal a defect within the soft tissues posteriorly just above the ankle joint which represents a large ulceration of the skin.

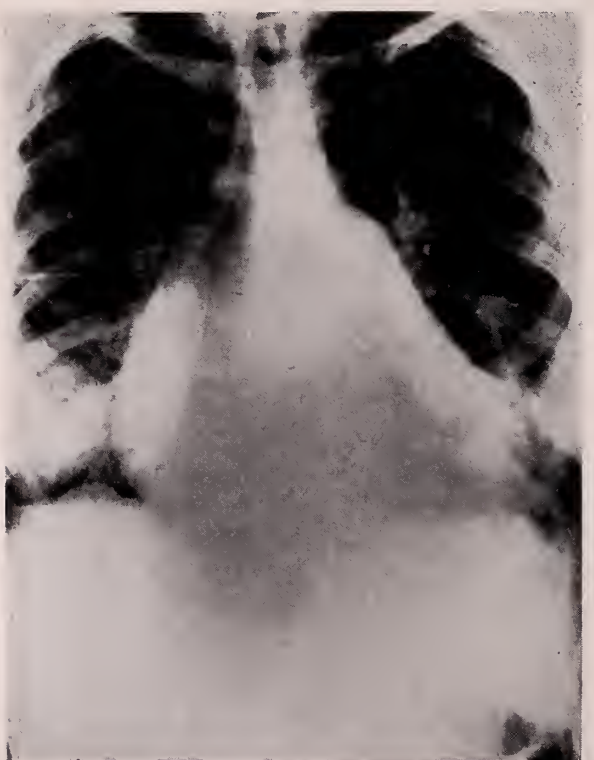


Fig. 1. Antero-posterior x-ray of patient's chest.

Clinical Discussion

DR. ZIFF: The patient had rheumatic heart disease and rheumatoid arthritis for many years. It is unlikely that she would have rheumatoid heart disease. This is a rare condition which would be unlikely to persist over a period of 27 years as the heart disease did in this patient, but would be of short duration. As for the association of rheumatic heart disease with rheumatoid arthritis, autopsy data indicate that rheumatic heart disease is twice as common in patients with rheumatoid arthritis as in the general population. But, of course, autopsy statistics are fraught with all kinds of statistical errors.

The patient had juvenile rheumatoid arthritis beginning at 18 months. The cervical spine films show fusion between the C_2 and C_3 and C_3 and C_4 apophyseal joints. This is a characteristic finding in the grown-up who began with juvenile rheumatoid arthritis. Practically all juvenile patients develop this change by the time they have passed

Fig. 2. Hands show evidence of rheumatoid arthritis.

puberty. Progression may involve the entire spine down to the sacroiliac joint. This finding is of some use diagnostically. When polyarthritis has occurred in childhood, and one is not sure whether it was rheumatic fever or rheumatoid arthritis, the presence of fusion of these spinal joints is helpful in pinpointing the diagnosis of juvenile rheumatoid arthritis.

This patient also brings up the interesting question of the relationship between juvenile rheumatoid arthritis and the adult form of the disease. Recently in England, the impression has begun to develop that juvenile rheumatoid arthritis is not related to the adult form, at least in many patients, but rather to ankylosing spondylitis. This is based on the spinal joint involvement, such as we see in this patient, often with sacroiliac and more extensive involvement; on the low incidence and titer of rheumatoid factor; and the increased incidence of uveitis in the juvenile group. Uveitis is common in adult ankylosing spondylitis. Also, certain limited family studies have indicated that relatives of patients with juvenile rheumatoid arthritis may have an increased incidence of ankylosing spondylitis. We don't have this impression here, and the patient we are discussing really refutes this idea. Though she had spinal involvement, she had rheumatoid factor in high titer, the latter reaching 1:224 on one occasion by our records. The patient also showed some evidence for the diagnosis of systemic lupus erythematosus, as we shall see, which patients with ankylosing spondylitis never do, and her relatives have true rheumatoid arthritis and rheumatoid factor in the serum.

This patient, as we have just seen, came from a rheumatoid family. The father had classic rheumatoid arthritis. His sensitized sheep cell agglutination titer was elevated. An uncle apparently had the same disease. A sister had juvenile rheumatoid arthritis, like the patient beginning at age two, and she also showed a positive test for the rheumatoid factor. Another sister had joint symptoms for 15 years. The "rheumatoid family" is not an infrequent phenomenon. It raises important questions about genetic and environmental factors which might lead to a familial occurrence since she had a positive lupus erythematosus test. From 9 to 20 per cent of patients with rheumatoid arthritis have positive L.E. tests. Frequently, such individuals also show positive serologic tests for syphilis. This was true in this case. Such patients generally have severe rheumatoid arthritis, but usually do not have other collagen diseases. They may have vascular lesions which on muscle biopsy will resemble that of polyarteritis nodosa and manifest themselves as peripheral neuropathy, or with ischemic skin lesions. This patient, however, was somewhat different. She had pancytopenia. This could have represented a Felty's syndrome in association with hypersplenism, but the fact that she did not have pancytopenia on her last admission would be against this diagnosis, and raises the questions of whether she did not in fact have systemic lupus erythematosus. In her last ten months of life, she had repeated episodes of pneumonitis associated, on one occasion, with pleuritic pain. There was blunting of a costophrenic angle on x-ray at one time. Pleurisy with repeated pneumonitis are suggestive of lupus erythematosus, although the fact that she seemed to respond to antibiotics with these attacks raises the possibility of an infectious etiology—especially since congestive heart failure was present.

Will the findings in the kidney help us in our dilemma? The patient had negative urinary findings. Generally, the kidney is negative in rheumatoid arthritis unless amyloidosis is present. Berman and Dubois performed renal biopsies on six rheumatoid patients who had positive L.E. tests, and found that five had positive changes on biopsy even though their urines were negative and blood urea nitrogen normal. Histologically, they described focal hypercellularity, adhesions, necrosis, and thickening of the basement membrane. It is hard to evaluate this data since a full paper has not yet been published.

In systemic lupus erythematosus itself, we have recently had renal biopsies on four patients who had negative urinary findings. Dr. Ashworth has, however, described mild lesions in the biopsy specimens. These included hypercellularity, mild focal basement membrane thickening, and interstitial infiltration. This raises the question of the clinical significance of mild lesions of this type. A group at New York University has recently indicated an unwillingness to accept these mild lesions in the absence of diffuse basement membrane thickening, wire loops, glomerular swelling, and necrosis as a basis for the diagnosis of lupus erythematosus. Using these stringent criteria, they have ar-

gued that patients with systemic lupus erythematosus who do not have abnormal findings in the urine, will not have positive findings in the kidney. This is still an open question in my opinion.

In essence, then, this patient brings up many interesting questions: the relationship of juvenile rheumatoid arthritis to the adult form of the disease, the relationship of lupus erythematosus to rheumatoid arthritis, significance of familial occurrence of rheumatoid arthritis, and the relation of the renal lesions of lupus erythematosus to the clinical findings. In fact, she stirs up the entire problem of the underlying nature of rheumatoid arthritis and systemic lupus erythematosus.

DR. FRENKEL: The question of "hypersplenism" has been raised. Having the entire clinical course before us today permits us to say she did not have "hypersplenism." If we focus on the admission in early 1963 as an isolated event, we note a normochromic, normocytic anemia, a leukopenia, and thrombocytopenia. The leukopenia was characterized by a relative persistence of polymorphonuclear leukocytes. The marrow report is in the protocol.

The term "hypersplenism" was popularized by Dr. Doan of Ohio State some three decades ago to describe a circumstance of a peripheral cytopenia or pancytopenia with a marrow that was hypercellular for the cell line noted to be decreased in the circulation. In addition the marrow generally demonstrates a shift toward immaturity for the specific cell line so involved. Generally splenomegaly is present. The basis for the morphologic changes is supposed to be splenic either in terms of unusual sequestration, supposed splenic humoral suppression of the marrow or splenic production of an antibody. In the jargon, "maturation arrest" has been applied to the marrow picture in "hypersplenism," but this may be rapid release and premature peripheral destruction as well as an inhibition of maturation.

As is evident, we did not have the marrow of "hypersplenism" here and the resultant clinical course is very much against it as well. The question of Felty's syndrome was also raised. We consider Felty's to be a form of "hypersplenism" in which a selective neutropenia is the hallmark. The syndrome is certainly not well understood and is, almost by clinical definition, related to the presence of long standing rheumatoid arthritis. Since selective neutropenia was not a feature in today's case, we can easily rule out Felty's syndrome.

In considering the leukopenia alone here, the relative persistence of neutrophils would raise the question of lupus erythematosus, since the leukopenia seen in this disorder is characterized by a selective retention of neutrophils. In terms of her bone marrow findings, we merely have a morphologic picture compatible with that found in rheumatoid arthritis with no distinctive features.

In summary, I think this patient did have the hematologic findings seen in patients with rheumatoid arthritis.

We saw the patient in 1963, and I had at that time explained the episode of pancytopenia on the basis of underlying infection. Clinically, I favored subacute bacterial endocarditis at the time.

DR. BAUM: The serology in this patient is interesting. In a survey we made of the antinuclear factor in patients with rheumatoid arthritis and with systemic lupus, it was found that in the majority of the patients with rheumatoid arthritis the antinuclear factor is macroglobulin while in those patients with systemic lupus it is practically always a 7S which would put it more in the lupus class rather than in the rheumatoid, at least as far as the antinuclear factor goes. One other thing, we tested other members of the family, five sisters, the mother, and the father for thyroglobulin antibodies and found none. This is somewhat different from other families with collagen diseases where we often find thyroglobulin antibodies.

The only sister in which we found rheumatoid factor was the one that I examined who actually showed clinical evidence of old juvenile rheumatoid arthritis. The other sister who had complaints had no physical findings and no factor.

DR. BARNETT: The presence of heart murmurs, low grade fever, splenomegaly and subungual hemorrhages made bacterial endocarditis a disturbing possibility in this patient. It should be emphasized that this constellation of clinical features is not pathognomonic of endocarditis in any case, and this particular patient had a chronic underlying disease which could very well account for each of the features. Weighing heavily against the diagnosis of endocarditis are the several negative blood cultures. Although the

patient had been on erythromycin, it seems quite unlikely that the small prophylactic dose could have concealed a bacteremia. Although bacterial endocarditis with "negative blood cultures" is often reported, proven cases are indeed few in number and most of these can be explained by the collection of blood cultures in the face of or soon after suboptimal antibiotic therapy. Although the erythromycin used in this patient could theoretically fall into the latter category, it seems unlikely that the blood levels expected from such a small dose would be suppressive against the organisms expected in endocarditis; also, most of the blood cultures were taken several days after withdrawal of the drug and past the point of any residual effect. I feel we would have been remiss to have empirically treated her for bacterial endocarditis without positive blood cultures.

Pathologic Findings

DR. ASHWORTH: This is a complex case and the pathologic findings at autopsy are extensive. It will be necessary to limit the discussion to the highlights only.

First, there was found ample evidence of the previously discussed rheumatoid arthritis. This was indicated by fixed ulnar deviation and swelling of the wrists, fixed and swollen ankles, fixed and flexed elbows and knees, and deformity of the toes and fingers with swelling of the middle phalangeal joints. Examination of the sternoclavicular joint histologically showed the typical pannus (of granulation tissue) extending onto the articular surface, replacing the lateral portions of the articular cartilage (Fig. 3). Mild lymphocytic infiltration and fibrosis were present in the pannus and in the joint capsule. There are no recorded differences in histologic findings between the juvenile type, ankylosing spondylitis, and rheumatoid arthritis, and therefore our pathologic findings do not help in this problem as discussed by Dr. Ziff.

The most impressive findings were in the heart, where clear evidence of severe chronic rheumatic heart disease was found. The heart was enlarged due to right and left ventricular hypertrophy, the former predominating. It weighed 480 gm. Chronic adhesive pericarditis obliterated the pericardial sac. The left auricular appendage was absent because of previous surgery. The most severe lesion was in the mitral valve which was the seat of obvious stenosis (Fig. 4). The orifice was 2 cm. in width, but the two cusps were extremely rigid due to fibrosis and calcification. The site of commissurotomy was still evident where the fused commissure had been fractured, but rigidity of the cusps had eventually rendered the commissurotomy relatively ineffectual. Yellowish-gray, firm to hard excrescences were located on the atrial surfaces of the thickened mitral leaflets. Viewing them grossly suggested possible bacterial endocarditis, but the excrescences were much firmer than thrombotic vegetations and gram stains of sections of these valves revealed no bacteria. They may have represented a previously active bacterial or other form of thrombotic endocarditis which then healed. The left atrium was greatly hypertrophied and di-

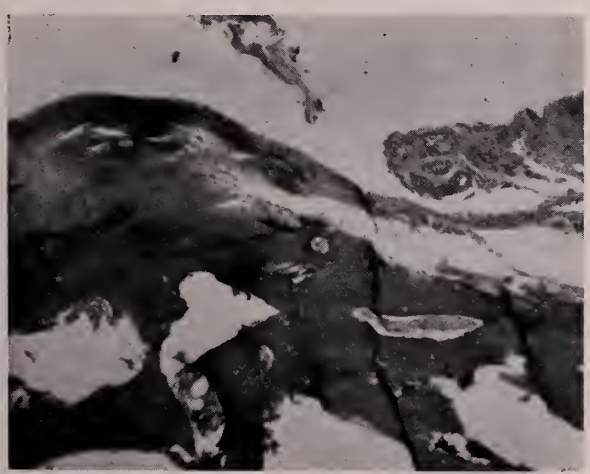


Fig. 3. Photomicrograph of sternoclavicular joint revealing replacement of articular cartilage by pannus.

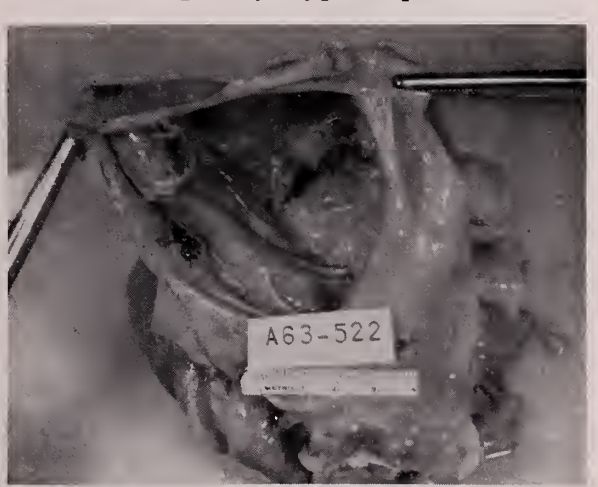


Fig. 4. Gross view of heart through opened left atrium, show calcified, stenotic mitral valve and evidence of previous commissurotomy. The lesion is typical of chronic rheumatic mitral valvulitis.

lated. Yellowish-gray endocardial plaques were present in the left atrium. Histologic sections of the mitral (and other) valves showed severe sclerosis, calcification, and vascularization, but no active inflammation was found. There were no large areas of fibrinoid necrosis nor nodules as would be seen in the entity of valvular heart disease due to rheumatoid arthritis; nor were there found small foci of fibrinoid degeneration or irregularly distributed valvular verrucous vegetations as occur in valvulitis due to lupus erythematosus (atypical verrucous endocarditis, or Libman—Sacks lesion). A few fine thrombotic deposits were present in areas of the lines of closure of aortic and mitral valves which had the appearance of degenerative endocardiosis.

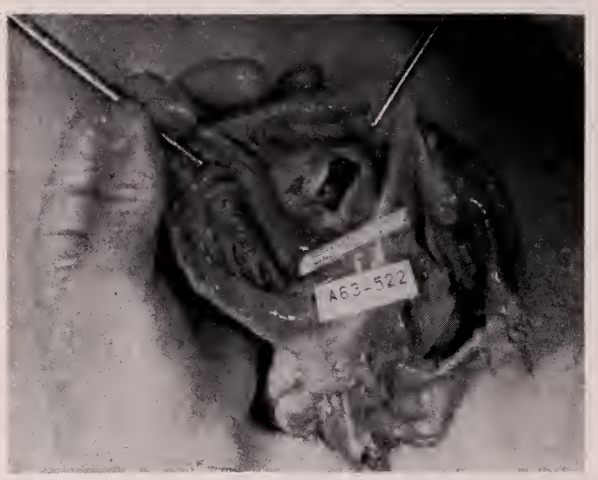


Fig. 5. Gross view of tricuspid valve through opened right atrium, showing moderate tricuspid stenosis.

Other heart valves were also affected. The aortic valve cusps were thickened and shortened, and there was partial loss of the lunular portions of the cusps. These alterations indicated that a mild degree of aortic valvular insufficiency might have been present. The tricuspid orifice was moderately stenotic (Fig. 5). This was due to fusion of the leaflets at all three commissures. The constellation of gross and microscopic changes in the heart are surely characteristic of chronic rheumatic heart disease, and we could find no support for the idea that either rheumatoid arthritis or lupus participated in the production of cardiac lesions.

Gross examination did not reveal any clear evidence of systemic lupus. The only suggestions of this were subacute and chronic fibrous perihepatitis and perisplenitis, but these changes of course are not very specific. Histologic study, however, revealed that the lesions of systemic lupus erythematosus were indeed present. In the spleen were found fibrinoid trabeculitis and concentric periarteriolar fibrous and fibrinoid thickening (Fig. 6). The connective tissue of liver, splenic, and adrenal capsules also revealed fibrinoid degeneration. This was indicated by the formation of small fragmented clumps of collagenous bundles taking a bright eosinophilic stain, and an increased amount of pale basophilic mucoid-appearing ground substance (Fig. 7).

The kidneys also were the seat of glomerular alterations indicative of a moderate degree of lupus nephritis. About half the glomeruli appeared normal, but the remainder were enlarged, hypercellular, contained lobules in which capillary lumens were ob-

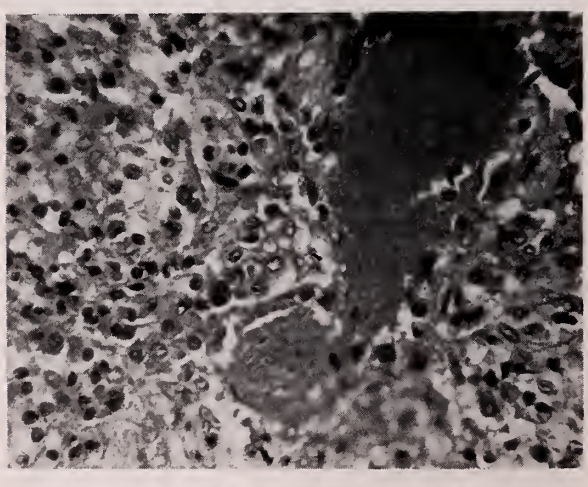


Fig. 6. Photomicrograph showing fibrinoid degeneration of trabecula in spleen.

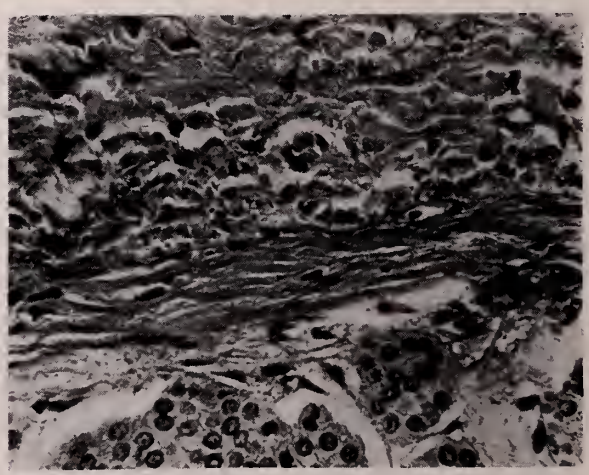


Fig. 7. Photomicrograph of adrenal capsule showing fibrinoid degeneration of connective tissue.

literated by endothelial cell swelling, and there were several areas of wire-loop appearance with fibrinoid-thickened capillary walls (Fig. 8). Previous chronic glomerular injury was also reflected by patchy areas of hyalinized obliteration (Fig. 9).

The spleen weighed 250 gm. and showed only slight reticuloendothelial hyperplasia. The changes in the spleen were mainly those of connective tissue degeneration associated with lupus. Marked hypersplenism could hardly be accounted for, it seems to me, on the basis of these findings.

The above findings indicate that the patient suffered from the unusual combination of three incompletely understood entities falling in the category of collagen-vascular disease, namely chronic rheumatoid arthritis, rheumatic fever, and lupus erythematosus. It is most unusual, in our experience, to see all of these together. The combination of rheumatoid arthritis with rheumatic fever or with lupus is perhaps not so rare, but all these in the same patient is, I believe, something of a rarity. Even so, their concurrence in this patient suggests an underlying common factor for the three disorders. If we follow the currently favored concepts of the collagen-vascular diseases, this common factor would be assumed to be a predisposition toward disease production by the auto-immune mechanism. Dr. Ziff's remarks about the familial occurrence of some of these diseases might be applied to the concept of such a special diathesis.

Since the pathologist is called upon to cite the cause of death, one might think that with the demonstration of three major diseases, two of which are easily acceptable as fatal diseases, his task would be easy in this case. Nevertheless, the increasing concern among physicians today for precise chemical and molecular explanations for biologic phenomena prevent the pathologist from becoming complacent. Our findings at autopsy disclosed evidence in the lungs and liver of severe congestive heart failure, and I believe that the joint consideration of the clinical course and our pathologic findings indicates that death was due to congestive heart failure.

There follows a list of the major pathologic diagnoses revealed at autopsy:

1. Chronic rheumatic valvular disease with congestive heart failure.
   a. Pronounced mitral stenosis, postcommissurotomy.
   b. Mild aortic valve insufficiency.
   c. Moderate tricuspid stenosis.
   d. Right and left ventricular hypertrophy.
   e. Focal myocardial fibrosis.
   f. Old fibrotic, calcified thrombotic vegetations on mitral leaflets.
2. Chronic passive congestion of lungs.
3. Acute and chronic passive congestion of liver.
4. Chronic rheumatoid arthritis, multiple joints involved.
5. Acute systemic lupus erythematosus.
   a. Fibrinoid degeneration of multiple fibrous connective tissue sites.
   b. Concentric periarteriolar fibrosis and fibrinoid degeneration of central arteries and fibrinoid trabeculitis of spleen.
   c. Mild lupus nephritis.
   d. Plasmacytosis of lymph nodes.
   e. Subacute perihepatitis and perisplenic serositis.

REFERENCE

1. Berman, L. B., and Dubois, E. L.: Renal Biopsy in Rheumatoid Arthritis with L. E. Cells, abstracted, Arthritis Rheum. 5:283 (June) 1962.

Fig. 8. Photomicrograph of portion of renal glomerulus showing moderate basement membrane thickening, endothelial swelling, and wire-loop appearance.

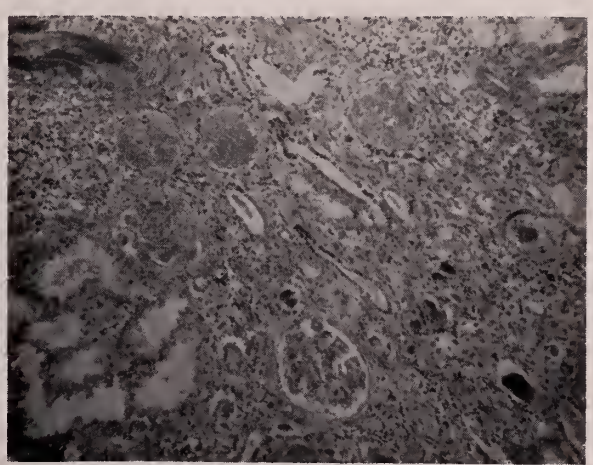


Fig. 9. Photomicrograph of kidney showing patchy parenchymal cortical atrophy with lymphocytic infiltration and glomerular hyalinization.

NEWS

Two Regional Medical Meetings Scheduled for Month of July

MEETINGS have been announced for two regional groups of physicians during the month of July. They are the Fifth and Sixth District meeting in Corpus Christi July 10-11 and the Postgraduate Medical Assembly of South Texas in Houston July 16-18.

Five special speakers will make up the program of the Fifth and Sixth District medical meeting to be held at the Driscoll Hotel in Corpus Christi.

They are Dr. Max E. Johnson, San Antonio, President of Texas Medical Association; Dr. George Ehni, Houston neurosurgeon; Dr. Bromley Smith Freeman, Houston plastic and maxillofacial surgeon; Dr. Louis J. Girard, professor and chairman, Department of Ophthalmology, Baylor University College of Medicine, Houston; and Dr. Joseph W. Goldzieher, director and chairman in research, Department of Endocrinology, Southwest Foundation of Research and Education, San Antonio.

The Postgraduate Medical Assembly of South Texas will hold its 30th annual meeting at the Shamrock Hilton Hotel in Houston.

Luncheon speakers are to include Dr. Edward R. Annis, president of the American Medical Association; Dr. Charles A. Berry, chief of Center Medical Operations Office, Manned Spacecraft Center, NASA, and William W. Slider, Th.D., Louisville, Ky.

The following scientific speakers will make up the three-day program which is acceptable for 18 accredited hours by the American Academy of General Practice:

Dr. Jose I. Barraquer, Bogotá, Colombia; Dr. Richard J. Bellucci, New York; Dr. Frank D. Costenbader, Washington, D.C.; Dr. Frederick P. Dewar, Toronto, Ontario; Dr. Edwin H. Ellison, Milwaukee; Dr. Richard T. Farrior, Tampa, Fla.; Dr. Harry Fields, Philadelphia; Dr. Melvin M. Figley, Seattle;

Dr. Lytt I. Gardner, Syracuse, N.Y.; Dr. J. Alex Haller, Jr., Baltimore; Dr. Zeph J. R. Hollenbeck, Columbus, Ohio; Dr. J. Willis Hurst, Atlanta; Dr. W. R. Lang, Philadelphia; Dr. Jack Lapides, Ann Arbor, Mich.; Dr. George E. Moore, Buffalo; Dr. John McLean Morris, New Haven, Conn.; Dr. Charles A. Neumeister, Minneapolis; Dr. John T. Sessions, Chapel Hill, N.C.; Dr. F. Mason Sones, Jr., Cleveland; and Dr. James F. Toole, Winston Salem, N.C.

Additional information may be obtained by writing Postgraduate Medical Assembly of South Texas, 209 Jesse H. Jones Library Building, Houston.

AAPS Contest Winners Announced in Houston

Winners of the 1964 Texas state American Association of Physicians and Surgeons essay contest have been announced by the chairman, Dr. Herbert C. Allen, Jr., of Houston.

First place winner is Sundra Dobbs of Route 5, Pittsburg; second place went to Kay Steger, Route 4, Pittsburg, and third place winner is Nancy Head of Big Spring. Essays are entitled "The Advantages of the American Free Enterprise System over Communism." All winners are high school students.

In her essay, Miss Dobbs noted that, "Too many people put too much emphasis on

'free' and too little emphasis on 'enterprise.' There are many misleading conceptions of free enterprise that are leading to the decline of our nation. Free enterprise does not mean that the world owes you a living and that you can get something for nothing. Yet this is how many people have accepted free enterprise. It means that the government grants an individual opportunity . . . and nothing more."

Communists, Miss Dobbs said, believe that "only the state deserves glory, and man exists only for its welfare . . . . Individual freedom is impractical because it interferes with state planning."

Postgraduate Courses

Cardiology, La Jolla, Calif., Sept. 15, 1964, to June 15, 1965.—A nine-month tutorial program will be offered by the Institute for CardioPulmonary Diseases of Scripps Clinic and Research Foundation.

This intensive program covering the field of cardiovascular diseases is designed for the physician in private practice who wants a year of organized instruction with freedom from direct patient responsibility.

Information may be obtained from Dr. E. Grey Dimond, Institute for Cardio-Pulmonary Diseases, Scripps Clinic and Research Foundation, La Jolla, Calif.

General Practice, Ruidoso, N. M., July 20-23.—The 1964 Ruidoso Summer Clinic will be sponsored by the New Mexico Chapter of the American Academy of General Practice.

Information may be obtained from Bob Reid, Mott, Reid and McFall, 310 N. Stanton St., El Paso.

Arthritis, Denver, June 15-16.—A review of problems commonly encountered in the management of the many forms of arthritis will be presented along with a review of therapy concepts.

The Rocky Mountain Chapter of Arthritis and Rheumatism Foundation, the Chronic Disease Section of the Colorado State Department of Public Health, and the University of Colorado School of Medicine will sponsor the program jointly. It will be held at the Humphreys Postgraduate Center of the University of Colorado School of Medicine.

Two panels, the medical management of rheumatoid arthritis and gout and surgical management of the arthritic patient, and clinical case presentations will be on the program.

A certificate of credit for 13 hours will be given to members of the American Academy of General Practice.

Registration and tuition fee for Texas physicians is $30.

Information may be obtained from the Office of Postgraduate Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver.

Pathology Seminar Slides Available

Slide sets of the 1964 Houston clinical pathology seminar on "Respiratory System, Gastrointestinal System, Genitourinary System and Bone" are still available at $10 per set and may be obtained from Dr. William T. Hill, St. Luke's Episcopal Hospital, Texas Medical Center, Houston.

"When you looked at my X-rays, Doctor, did you discover the beautiful inner ME?"

Nueces Medical Students Get Emergency Funds

When suddenly confronted with financial crises, students from Nueces County who are attending Texas medical schools may draw upon emergency funds which have been deposited with the deans.

A total of $5,000 which became available to Nueces County Medical Society as a result of surplus donations during the 1962 Victory Over Polio drive in Nueces County has been designated for these funds. Disbursements are outright gifts, not loans, and are made at the discretion of the deans.

In addition to immunizing more than 165,000 persons, the drive sponsored by the medical society netted a surplus of nearly $15,000 more than was needed for the oral vaccine and other supplies.

Also receiving portions of the surplus were Goodwill Industries, Boys City, Community Settlement House, Bethune Day Nursery, Coastal Bend Habilitation Association, and local libraries.

Courts Hold Surgeon "Captain of Ship"

TEXAS COURTS have recognized that during the operating process the surgeon is in charge of the operating room and is thus the "captain of the ship" with respect to what is done and by whom. Several other state courts also have recognized this doctrine of responsibility, and a recent Pennsylvania case is an example of what could very well happen in Texas.

Under the Pennsylvania "captain of the ship" doctrine, an anesthesiologist would not be liable but a surgeon would be liable for any negligence in or adjacent to the operating room by a head technician of a blood bank where the negligence resulted in the transfusion of a patient with incompatible blood. A majority of the U. S. Court of Appeals for the Third Circuit so ruled.

The patient, Israel Abrams, was hospitalized for gallbladder surgery to be performed the following day. He was placed in Room 807. The same day another patient of the same name was admitted and placed in Room 342. The anesthesiologist directed the blood bank to set aside two bottles of blood for the Abrams on whom surgery was to be performed the following day. The blood bank's head technician, after checking the hospital census, directed the I-V team to take a specimen of the blood of the Abrams in Room 342. That blood was type A. Apparently, the head technician discovered that afternoon that there were two Israel Abramses in the hospital and took a blood sample from the Abrams in Room 807. That Abrams' blood was type O, Rh-positive.

During the operation the next morning on the Abrams of Room 807, his cystic artery was severed, making a transfusion imperative. The anesthesiologist then discovered that the bottle of blood which had been placed in the operating room was labeled "Israel Abrams" but that the operating room chart showed the bottle was marked "342 A Rh-positive." He summoned the head technician to the operating room and talked to him, at or through the door, which was only a few feet away from the surgeon. The technician said that the room number was a

clerical error and that it would be proper to give the blood to the patient. One pint of the blood was given to him. The blood was not of the proper type, and the patient suffered a transfusion reaction. The patient died several days later. There was substantial evidence that the administration of the incompatible blood was the cause of death.

The trial court instructed the jury that neither the surgeon nor the anesthesiologist could be held liable for the negligence of any employee of the hospital. This instruction was erroneous as to the possible liability of the surgeon for any negligence on the part of the head technician of the blood bank and, although no proper exception was taken to the instruction, the error was so plain as to require a new trial, the court ruled.

During the course of an operation, the surgeon is, under the law of Pennsylvania, in the same complete charge of those who are present and assisting him as is the captain of a ship over all on board. Since the surgeon has this complete charge over all those present and assisting him, he is responsible for any negligence on their part. When the head technician came to the operating room door, he came under the surgeon's control, the court said. It was the head technician's assurance that the blood was proper that caused the transfusion, an integral and essential part of the operative procedure, to be proceeded with. If the head technician was negligent in giving that assurance, the surgeon would be liable for that negligence, the court ruled.

There was no evidence of any individual negligence on the anesthesiologist's part. The anesthesiologist could not be held liable for any negligence on the part of the head technician because he was not the "captain of the ship," the court ruled. To say that because the anesthesiologist was in charge of the transfusion, he was responsible for any negligence on the part of the head technician would extend the ship's captain doctrine too far beyond the present bounds of the law of Pennsylvania. It would greatly distort that law to say that it was plain error to fail to instruct a jury that there can be two captains of the ship at one and the same time, the court said.*

It is interesting to note the division of responsibility the case illustrates between the surgeon and the anesthesiologist. The conclusion of such a case study is that some actual act of negligence on the part of the anesthesiologist must be shown in order to make him responsible for negligence in the operating room.
—PHILIP R. OVERTON, LL.B., Austin.

*Law Department, American Medical Association, The Citation 9:18 (April 30) 1964.

Personals

New officers of the Texas Society of Anesthesiologists are *Dr. E. L. Slataper,* Houston, president; *Dr. I. E. Lamberth,* Tyler, vice president; and *Dr. Fred P. Thomas,* Houston, secretary.

New officers of the South Texas District, American College of Surgeons, are *Dr. Lawrence Shefts,* San Antonio, president; *Dr. Sanford Glanz,* Corpus Christi, president-elect; and *Dr. August Herff, Jr.,* San Antonio, secretary-treasurer.

The Texas chapter of the Flying Physicians Association recently elected *Dr. James R. Old* of Beaumont, president; *Dr. Walter A. Brooks* of Quanah, vice president, and *Dr. Loren W. Haus* of Houston, secretary-treasurer.

Dr. Howard C. Hopps has moved to Washington, D. C., where he is chief of geographic pathology division of the Armed Forces Institute of Pathology.

Dr. William K. Wright of Houston is the president-elect of the American Otorhinologic Society for Plastic Surgery, Inc.

Among the speakers on the program of the 1964 Conference of the National Society for the Prevention of Blindness, Inc., was *Dr. Otto Lippmann* of Austin. The meeting was held May 6-8 in Miami Beach, Fla.

Recently inducted into Rotary District 587's Roll of Fame is *Dr. Howard O. Smith* of Marlin.

Dr. Louis Gibson of Corsicana and *Dr. Per H. Langsjoen* of Temple were recent speakers at meetings of the Hill County Medical Society.

Mr. Roy Haeusler of the Chrysler Corporation recently spoke to members of the Tarrant County Medical Society on the subject of "The Automobile Manufacturer's Approach to Automobile Safety."

A junior at the University of Texas Southwestern Medical School, *Thomas B. Carleton* has received one of ten SAMA-Sears Preceptor-Scholarships awarded this year by the Student American Medical Association and provided by the Sears Roebuck Foundation.

Texas Industrial Medical Association officers are *Dr. Roy E. Joyner,* Texas City, president; *Dr. Robert R. Dugan,* Houston, vice president; and *Dr. James K. Stewart,* Beaumont, secretary-treasurer.

Texas Dermatological Society has announced the following officers: *Dr. J. Franklin Campbell,* Fort Worth, president; *Dr. A. C. Ressmann,* San Antonio, vice president; *Dr. M. Allen Forbes, Jr.,* Austin, secretary-treasurer; and *Dr. Coleman Jacobson, Dallas,* program chairman.

Dr. E. Ivan Bruce, Jr., of Galveston is president of the Texas Neuropsychiatric Association. Other officers are *Dr. David Keedy,* San Antonio, vice president, and *Dr. J. K. Peden,* Dallas, secretary-treasurer.

Officers of the Texas Otolaryngological Association are *Dr. Louis Adin,* Dallas, president; *Dr. Ben T. Withers,* Houston, vice president; *Dr. Norman Wright,* Amarillo, secretary; *Dr. Jesse D. Gomillion,* Midland; and *Dr. J. Patrick Moran,* president-elect, Corpus Christi.

Texas Society of Pathologists, Inc., has elected the following officers: *Dr. Mervin Grossman,* Dallas, president; *Dr. Wilson Brown,* Houston, vice president; *Dr. Vernie A. Stembridge,* Dallas, secretary-treasurer; and *Dr. John Rainey,* Austin, president-elect.

Dr. John L. Jackson, III, of Wichita Falls is the president of the 13th District Society. Other officers are *Ervin E. Addy, Jr.,* Cisco, vice president; and *W. H. Neil,* Fort Worth, secretary-treasurer.

New officers of the Texas Association of Obstetricians and Gynecologists are *Dr. John Wall,* Houston, president; *Dr. Jack Pritchard,* Dallas, vice president; *Dr. James T. Downs, III,* Dallas, secretary-treasurer; and *Dr. Hugh Savage,* Fort Worth, president-elect.

Dr. E. E. Anthony, Jr., of Fort Worth is the new vice president of the Association of American Physicians and Surgeons, Inc.

The Board of Regents of the University of Texas has announced the appointment of *Dr. John B. Truslow,* present executive dean and director of the Medical Branch in Galveston, to the system-wide position of consultant to the chancellor on programs in the health sciences. The appointment takes effect Sept. 1, 1964. At that time, Dr. Truslow will be succeeded by *Dr. Truman Blocker,* chairman of the Department of Surgery, who will become executive director of the Medical Branch.

Dr. Truslow has been associated with the Medical Branch since 1956. During his tenure the academic program and physical plant have been greatly improved. In commenting on Dr. Truslow's work, Judge W. W. Heath, chairman of the Board of Regents, and Harry Ransom, Ph.D., chancellor of the University of Texas System, said, "Dr. Truslow is a tremen-

dously imaginative educator. We welcome his talents in the new program to study plans for development in the health sciences, including pre-medical education."

Dr. Blocker has been associated with the University of Texas since 1936. As a surgeon he has gained an international reputation. His work and that of his colleagues were recently recognized by the establishment of the Shriners Burns Institute in connection with the Medical School.

Dr. Blocker has also had wide experience in federal programs for medicine, holding the rank of brigadier general in the Air Force.

Officers of the Texas Society of Athletic Team Physicians are *Dr. Lewis J. Levy,* Fort Worth, president; *Dr. Dwight R. Knapp,* Kerrville, vice president; *Dr. Dan Spivey,* Lufkin, secretary-treasurer; and *Dr. Louis Breck,* El Paso, president-elect.

Officers of the Texas Orthopaedic Association are *Dr. Marvin P. Knight,* Dallas, president; *Dr. Phillip Day,* San Antonio, vice president; *Dr. Margaret Watkins,* Dallas, secretary-treasurer; and *Dr. Jack Maxfield,* Wichita Falls, president-elect.

Dr. C. F. Pelphrey, Austin, is vice president of the South Central Association of Blood Banks.

Texas Air-Medics Association officers are *Dr. W. B. Adams, Sr.,* Wichita Falls, president; *Dr. R. C. Suttle,* Dallas, vice president; *Dr. C. F. Miller,* Waco, secretary-treasurer; *Dr. B. B. Elster,* Port Arthur, director for three-year term.

Texas Ophthalmological Association officers are *Dr. Everett R. Veirs,* Temple, president; *Dr. Harold E. Hunt,* Paris, vice president; *Dr. J. L. Mims, Jr.,* San Antonio, secretary; *Dr. Harl Mansur,* Wichita Falls, treasurer; and *Drs. Louis Daily,* Houston, *Tom McCrory,* Dallas, *Richard Leigh, Jr.,* Houston, *Joe Bussey,* Fort Worth, *Sam Key, Jr.,* Austin, *Walter G. Morrow, Jr.,* El Paso, and *Jerry Johnson,* Beaumont, councillors.

Officers of the Texas Society of Plastic Surgeons are *Dr. Steve Lewis,* Galveston, president; *Dr. Willard C. Sellman, Jr.,* Dallas, vice president; and *Dr. John Gannon,* Houston, secretary-treasurer.

Officers of the Texas Association of Public Health Physicians are *Dr. Albert G. Randall* of Amarillo, president; *Dr. W. S. Brumage* of Austin, president-elect; *Dr. John B. Bryson* of Belton, vice president; *Dr. Francine Jensen,* Houston, secretary-treasurer; and *Dr. W. R. Metzger,* Corpus Christi, new trustee.

Dr. Boen Swinny of San Antonio is president of the Amercian College of Allergists.

Dr. Robert P. Green of Texas City is the new president of the East Texas Chamber of Commerce. He has also been named by *East Texas Magazine* as its Man of the Month for May.

Dr. James Wynn has received the Alice Jaycee award as the most outstanding young man in Alice during 1963.

Dr. Pat Ireland Nixon's collection of books on Texas history has been given to Trinity University to be placed in the school's rare books room. The gift was made in Dr. Nixon's name by his sons, *Dr. Pat Nixon, Jr.,* and *Dr. Robert Nixon.* All are San Antonio physicians. The eldest Dr. Nixon was recently honored for his half-century of medical service in San Antonio at a dinner attended by more than 200 Bexar County physicians and their wives.

Dr. Charles F. Pelphrey of Austin has been elected vice president of the South Central Association of Blood Banks at its annual meeting in Oklahoma City.

Dr. Elizabeth Donaldson of Del Rio has retired from practice after 33 years.

Dr. Bob Alexander of Eastland has been named "Outstanding Young Man of the Year" by the Eastland Jaycees.

Dr. Lloyd M. Southwick of Edinburg has been named one of the three new directors of the American Academy of General Practice.

Dr. James D. Murphy of Fort Worth is cochairman of the National Advisory Committee on Medic Alert, nonprofit organization which promotes use of bracelets and necklaces to identify the wearers' hidden medical problems.

Dr. H. P. Sammons was recently honored at a party given by people of his community in appreciation of his long years of service as a general practitioner. Dr. Sammons is from Hubbard.

Dr. Thomas D. Cronin of Houston was elected a trustee of the American Association of Plastic Surgeons May 15 at the annual meeting in Chicago. He served as president of the organization this year.

Officers of the Texas Division, International College of Surgeons are *Dr. William Basom,* El Paso, president; *Dr. Harvey Renger,* Hallettsville, president-elect; *Dr. Albert P. D'Errico,* Dallas, vice president; *Dr. Robert G. McCorkle,* Houston, secretary; and *Dr. Jack Crow,* Abilene, treasurer.

New Books In TMA Library

Avery, M. E.: **Lung and Its Disorders in the Newborn Infant**, Philadelphia, W. B. Saunders Co., 1964.

Berne, E.: **Structure and Dynamics of Organizations and Groups**, Philadelphia, J. B. Lippincott Co., 1964.

Ciba Foundation Study Group No. 16, Wolstenholme, G. E. W., Ed.: **Immunologically Competent Cell**, Boston, Little, Brown and Co., 1964.

Ciba Foundation Symposium, De-Reuck, A. V. S., Ed.: **Disorders of Language**, Boston, Little, Brown and Co., 1964.

Hansell, P., and Ollerenshaw, R., Ed.: **Longmore's Medical Photography**, 7th rev. ed., Philadelphia, J. B. Lippincott Co., 1962.

Michael, P.: **Tumors of Infancy and Childhood**, Philadelphia, J. B. Lippincott Co., 1964.

Smith, A.: **Birth of a Drug**, Washington, D. C., Pharmaceutical Manufacturers Association, 1963.

Top, F. H.: **Communicable and Infectious Diseases**, 5th ed., St. Louis, C. V. Mosby Co., 1964.

University of Texas, M. D. Anderson Hospital & Tumor Institute: **First Twenty Years of the University of Texas M. D. Anderson Hospital and Tumor Institute**, Houston, 1964.

University of Texas, M. D. Anderson Hospital and Tumor Institute: **Research Report 1963**, Houston, 1963.

Book Notes

Books reviewed in this column are available from the TMA library.

Physical Diagnosis: The History and Examination of the Patient. Drs. John A. Prior and Jack S. Silberstein. 455 pp. St. Louis, C. V. Mosby Co., 1963. $8.50.

PHYSICAL DIAGNOSIS is not a static subject. As the result of new medical and surgical techniques in diagnosis, certain alterations in body functions assume increasing significance. For example, the growth of vascular surgery has made possible the repair of abdominal aortic aneurysms. As a consequence, such advances necessitate a more thorough consideration of examination techniques as applied to the abdomen. Tracings of heart sounds have resulted in a clearer understanding of the dynamics of production of these sounds. The chapter on the cardiovascular system has incorporated a number of heart sound tracings in order that the student may correlate the physical findings with the tracings. Other additions to the current edition include evaluation of hearing from a clinical point of view, measurement of the visual fields, localization of blind spots, and bedside tests for venous competency in the extremities.

Synopsis of Ear, Nose, and Throat Diseases. Drs. Robert E. Ryan, William C. Thornell, and Hans von Leden. 425 pp. St. Louis, C. V. Mosby Co., 1963. $7.50.

NEW CONCEPTS in diagnosis and treatment of ear, nose, and throat conditions have made this third edition necessary. The synopsis continues to serve as a handy guide for the physician, intern, and medical student as well as nurses and speech pathologists.

Thymectomy for Myasthenia Gravis. Henry R. Viets, M.D., and Robert S. Schwab, M.D. 143 pp. Springfield, Ill., Charles C Thomas, 1960. $7.

A MONOGRAPH on a special form of treatment for such an uncommon disease as myasthenia gravis would not be justified except on the basis of a comprehensive review of the subject, covering the historical background, the current theories regarding neuromuscular transmissions of the nerve impulse across the myoneural junction, and the presumed functions of the thymus, as these factors affect our concepts of present-day treatment.

A Study of Psychophysical Methods for Relief of Childbirth Pain. C. Lee Buxton, M.D. 116 pp. Philadelphia, W. B. Saunders Co., 1962. $4.75.

DR. BUXTON, inspired by the curiosity and interest of women of childbearing age, obstetricians, and others involved in the process of parturition, has taken the time to visit and observe the different techniques of mental and physical training for relief of pain in childbirth in more than 30 clinics in the United States and Europe. A determined effort is made to present the reports without effort to convince or condemn.

Atomic Energy Encyclopedia in the Life Sciences. Charles Wesley Shilling, M.D., D.Sc. 474 pp. Philadelphia, W. B. Saunders Co., 1964. $10.50.

DR. SHILLING and his distinguished board of consultants have combined the features of both dictionary and encyclopedia in this new one-volume sourcebook. It offers valuable reference material to scientists working in the field of atomic energy, to biologists, to medical researchers, to specialists in agriculture, to teachers and students, and to civil defense and public health officers. It is particularly illuminating to physicians working in radiology, genetics, and many other areas of medical practice.

Topics range from the latest radiation instrumentation to methods of radioactive waste disposal.

Diseases of the Heart and Blood Vessels: Nomenclature and Criteria for Diagnosis. Criteria Committee of the New York Heart Association. 440 pp. Boston, Little, Brown, and Co., 1964. $5.50.

THE SIXTH EDITION has been written to integrate a clinically usable set of terms and rules. These are the terms and rules necessary to further progress, and essential to the achievement of the ultimate goal of all intellectual medical effort—the prevention and management of disease in man.

In the preparation of this new edition, many changes from the previous edition have been made. These changes simply reflect the numerous additions to the understanding of cardiovascular disease over the last decade.

Diagnostic Laboratory Hematology. George E. Cartwright. 3rd ed. 339 pp. New York, Grune & Stratton, Inc., 1963. $8.75.

A CONCISE, PRECISE, well organized and well written practical laboratory manual of hematology procedures, this book is well suited to physicians, laboratory technologists, students, and others engaged in laboratory diagnostic procedures. The book, by the author's intention, is selective as to choice of methods. It gives only those procedures which in the author's experience have proven of most practical value in the average hospital laboratory. The book briefly covers details of technical procedures, interpretation of results, sources of error, methods of calculations and calibrations, and statistical evaluation of results. These permit the laboratory worker to maintain quality control work.

—D. E. Fletcher, M.D.,
Wichita Falls.

Diseases of the Chest. H. C. Hinshaw, M.D., and L. H. Garland, M.D. 767 pp. Philadelphia, W. B. Saunders Co., 1963. $20.

THE SECOND EDITION of this book should be of particular interest to internists and generalists. The book is written for the clinician rather than the research worker or chest specialist and is oriented toward practical rather than theoretic consideration.

Several chapters are worthy of special merit. The difficult subject of emphysema is well handled in one of the best chapters in the book. Even the definition of emphysema is subject to wide variation and the difference in viewpoint between American and British workers is brought into clear focus. The various forms in which this disease may appear are also well defined and explained. The pathogenesis of tuberculosis likewise is written with unusual clarity. The chapter on segmental anatomy of the tracheobronchial tree and lungs contains unusually helpful illustrations, among the best of which are those showing the approximate location of roentgenographic shadows produced by consolidated pulmonary segments.

One of the most valuable features of this book is the list of references which appear at the end of each chapter. The references have been intelligently selected, rather than exhaustively compiled, and are oriented toward the practical rather than the theoretic.

The chapter on pulmonary function testing is not an easy one to read. Admittedly this is a difficult subject but one might justly have hoped to find it covered with greater clarity. The three chapters covering diagnostic procedures consume 59 pages, most of which could be omitted without decreasing the value of the book.

The authors have drawn on their extensive experience gained from a busy practice and have passed this experience on to the reader in a very skillful presentation.

—Edwin B. Wilson, Jr., M.D.,
Fort Worth.

The Knee in Athletics. Karl K. Klein and William L. Hall. 49 pp. Washington, D. C., American Association for Health, Physical Education, and Recreation. 1963.

THE BOOK will help coaches and trainers cope with these problems by outlining programs to strengthen the knee and restore it quickly to normal action after injury. It includes sections on preventive conditioning, traumatic injury, physical rehabilitation, and related research.

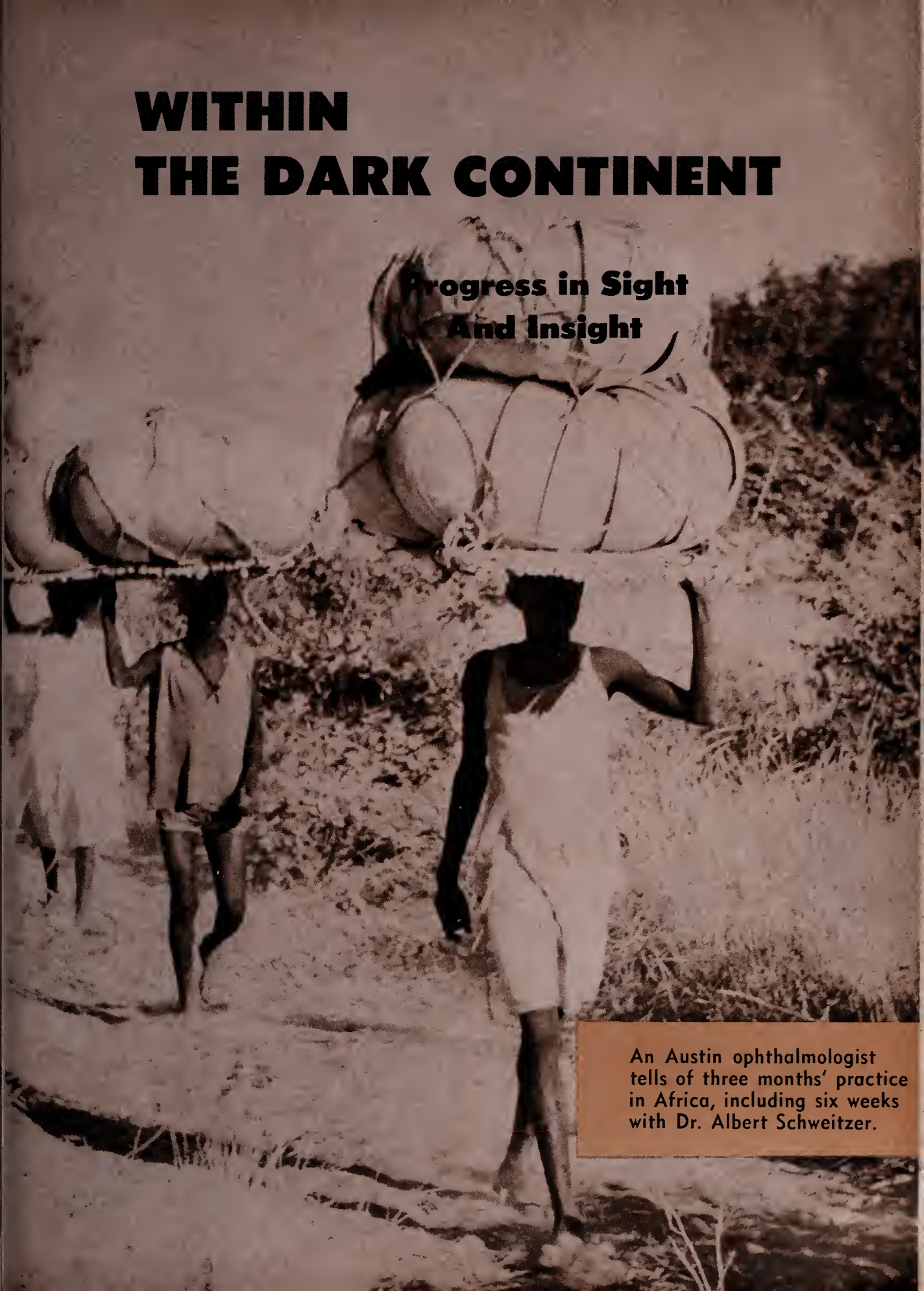
New Films in TMA Library

Hospital Sepsis—A Communicable Disease—1959. Sound, color, 28 minutes.

DESIGNED to educate all levels of hospital personnel concerning the many avenues by which infection can be spread throughout a hospital, this film utilizes staphylococcus as an example of one of the most important phases of the problem.

WITHIN THE DARK CONTINENT

Progress in Sight And Insight



An Austin ophthalmologist tells of three months' practice in Africa, including six weeks with Dr. Albert Schweitzer.

TO WESTERN EYES, the sights on African roads are often curious. This is a primitive land where, for example, giant gourds* may be used instead of washtubs, milk pails, or cardboard cartons. It is a country chained to the past by ignorance and superstition, yet in certain areas Christian missionaries have superimposed a new way of life upon an old culture. Their ministry of healing has been gladly received by multitudes within the Dark Continent, and toward their hospitals, down the jungle paths and up the treacherous rivers, have come the lame, the halt, and the blind.

Dr. Robert L. Rock never dreamed that so early in his career he would see so many patients, but as a Christian Medical Society short-term missionary, he found himself, in the fall of 1962, one of four or five ophthalmologists in a land of 50,000,000 people. For three months of that year the young Texan donated his time and services to two African hospitals, Dr. Albert Schweitzer's hospital in Lambaréné and the mission eye hospital in Kano. These two medical centers, he found, contrast the old and new ideals in missionary activity.

In a jungle almost too thick to walk through, its lush green dotted with orange tops of trees called "flame of the jungle," the famous Schweitzer hospital lies hidden from the world. The beautiful wide Ogowe River divides the mass of foliage and provides the only travellable avenue to Lambaréné. Slender dugouts called pirogues carry the sick through waters infested with crocodiles and hippopotamuses to the hospital on the bank of the river. Ashore, instead of the landscaped gardens and concrete sidewalks which enhance modern hospitals, wash hangs on clotheslines between brown, tin-roofed buildings. There is no whir of air conditioners; instead, monkeys chatter and birds call. Cats, antelope, and goats wander about the grounds waiting for Nobel prize winner Schweitzer to pet and feed them.

Far from being out of place in this backward country, the Schweitzer hospital is part of it, Dr. Rock soon discovered. There is no running water at Lambaréné, no indoor toilets, and no electricity except for an operating lamp in surgery. Once when Dr. Schweitzer was in Europe, members of the staff began wiring the hospital for electricity to surprise him, but the old doctor had it removed when he returned. It is his philosophy that patients should be kept in their own environment.

The hospital is well equipped, however, and the laboratory can do basic tests, x-rays, and electrocardiograms. Outpatient clinic is held six days a week. Despite the apparent crudeness, the Schweitzer hospital facilities offer the best medical care in the entire country of Gabon.

While no accurate census of inpatients is kept, a count was made in August, 1962, at which time the occupancy rate was reported at 1.65 patients per bed. These extra patients were placed on straw floor mats, Dr. Rock learned. The patient's family, he found, also goes to the hospital and sets up housekeeping outside the patient's room, and one of

*Photograph on preceding page shows calabash gourd shells being borne to market.

Primitive Lambarene still offers the best medical care in the entire country of Gabon. Schweitzer's hospital has no running water, no indoor toilets, a minimum of electric lighting.

Sudan Interior Mission Eye Hospital in Kano, North Nigeria, is the largest eye hospital in Africa. Patients arrive via train and donkey; sometimes travel 800 miles.

SUDAN INTERIOR MISSION
EYE HOSPITAL

the largest expenditures in the hospital budget is food for these families.

Practically every patient Dr. Rock saw was a victim of malaria, a disease almost 100 per cent prevalent in this part of Africa. Other common diseases are skin ulcers, syphilis, hernias, genitourinary problems, cataracts, leprosy, and parasitic infections. He rarely saw heart diseases and cancer since life expectancy in Gabon is only about 40 years.

The medical work at the clinic is carried out by a European staff of five physicians and several nurses who usually stay about two years.

Dr. Schweitzer himself, now 89 years old, no longer practices medicine. He spends most of his time reading and writing political, philosophical, and theological books. Far from being a meek saint, "Le Grand Docteur" is a firm taskmaster for himself and others. The outline of his shock of white hair and bushy mustache may be seen bent over his writing table late each night. During the day, he gives attention to every detail at the clinic, and nothing is constructed, planned, or carried out without his exacting supervision.

To Dr. Rock, this almost legendary man is puzzling. He is training no successor, and he trains no Africans above the level of a nurse's aide because he believes that further training is above their mentality. More than a physician, teacher, and evangelist, Schweitzer is a humanitarian, Dr. Rock has decided, and probably one of his greatest contributions to humanity has been his inspiration to others to donate their services to missionary activities.

A thousand miles away in a sub-Sahara desert of North Nigeria where the climate could easily be mistaken for dusty West Texas, Dr. Rock found a new concept in missionary hospitals. The staff of the Sudan Interior Mission Eye Hospital, located in Kano, encourages all possible progress.

At this hospital, the largest eye hospital in Africa, an attempt is made to train Africans in everything. They are employed as laboratory technicians, nurses, and optometrists, and the hospital is eager to get an African doctor to train as an ophthalmologist.

The 150-bed hospital is a gleaming white building, modern both in appearance and equipment. A book store and an optical shop which dispenses approximately 6,000 pairs of glasses yearly are located next to the hospital.

Dr. Kietzman examines one of 300 to 600 patients he sees during a morning in outpatient clinic at the Kano hospital. He is the only full-time physician at the hospital.

Dr. Rock joins Dr. Schweitzer in the older doctor's frugally furnished office. More than a physician, teacher, and evangelist, "Le Grande Docteur" is a humanitarian. He is now 89.

The laboratory is equipped to do blood cell counts, urinalyses, skin snips for onchocerciasis, and other basic tests, and a nearby hospital has rather complete x-ray and pathologic facilities.

Although this is a charity hospital, an attempt is made to charge each patient, Dr. Rock learned. "This helps the mission and gives a sense of dignity to the patients. They feel they're paying the full amount," he was told. One of his poor patients received a 40-cent bill for bilateral surgery and cataract glasses. Usually bilateral cataract surgery is $6-$8 and glasses are $3. The cheapest clinic visit is 2 cents, and the only dif-

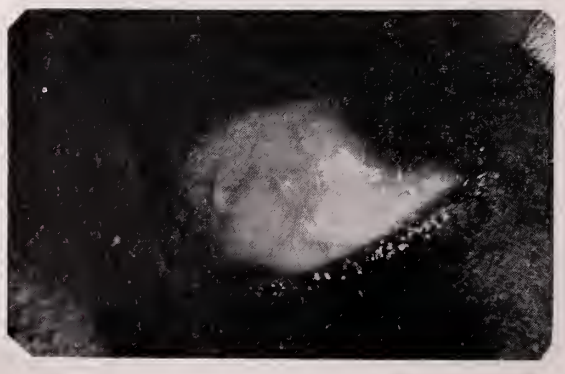
Witch doctor has treated this patient, a victim of hyphema and glaucoma. Brow lacerations were made to release evil spirits.

Smallpox still strikes in Africa. This patient has suffered total destruction of his eyes from staphyloma following smallpox keratitis.

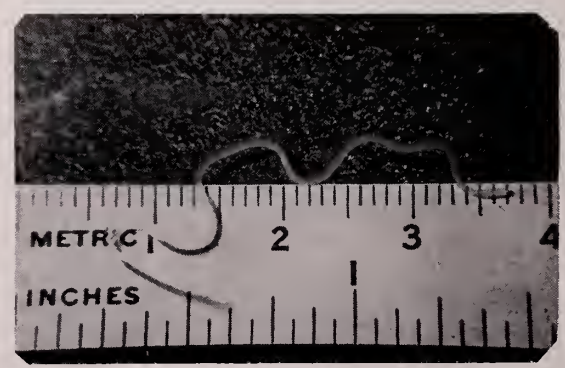
Typical bilateral mature cataracts in a 30-year-old man. Patient waited until he was blind before he consulted a medical doctor.

Long-neglected is this orbital granuloma with abscess formation in an African child. Exenteration was done, and the child lived.

Subconjunctival Onchocerca adult worm is a fairly common finding in the eyes of Africans.



Onchocerca worm following removal from the eye. Excision is a simple procedure.



ference in a visit at a higher fee is a shorter waiting line.

Patients come 800 to 1,000 miles to the eye hospital by train and donkey, Dr. Rock observed. "They have the usual eye diseases we see in the United States, cataracts, glaucoma, corneal scars, and infectious diseases, but always in advanced stages; we almost never saw the early stages of the diseases."

A great many patients have bilateral mature cataracts, and most cataract victims are from 20 to 40 years old. In addition to the more common diseases, Dr. Rock saw cases of old and fresh smallpox and onchocerciasis, and almost every patient he saw was a victim or had been a victim of trachoma.

Only one full-time physician is at the mission eye hospital to handle all these diseases and direct the hospital. Dr. Rock came to know Dr. Ben Kietzman as "a quiet, unassuming physician." In his early 30's, he is a combination mechanic, plumber, electrician, and physician who spends three years in Africa and one year in the States. His only help comes from short-term missionaries.

In addition to seeing 300 to 600 patients each morning in the outpatient clinic, Dr. Kietzman and Dr. Rock usually performed 35 to 45 operations every afternoon.

To Dr. Rock's surprise, even though gloves were not worn in surgery and patients were often placed on straw mats on the floor, the rate of infections compared favorably with hospitals in this country. "It seems they do very well with their own organisms," he says.

Dr. Rock found that some of the nurses were trained to help with surgery. A 22-year-old male nurse performed lid surgery to correct scarring from trachoma, and a white missionary nurse from Ohio performed intraocular surgery, mostly cataract extractions.

Optical iridectomies are frequent operations at the eye hospital since corneal transplants cannot be given to all patients who need them, Dr. Rock said. Most Africans will not donate their eyes or any part of their bodies because of a superstition that the person who possesses the part will have control of the donor after death.

Donations were sometimes flown in from the United States, and the mission eye hospital made national headlines when a jet plane flew in two eyes to be used in corneal transplants. It was hoped this publicity would encourage more Africans to donate.

Local anesthesia is used for most eye surgery, and frequently both eyes are operated on at the same time. Dr. Rock's youngest patient to have a local anesthetic was 5 years old; he had an optical iridectomy performed in both eyes. Most Africans believe they are killed with general anesthesia and later brought back to life.

Prayers are said for each patient before his surgery. Dr. Rock's Moslem patients even welcomed these prayers "because you people pray to God for us and don't cheat us out of our money."

Reared in a Christian nation, Dr. Rock was amazed that honesty baffles most African people. A serious problem hospital personnel confront is thieves waiting at train stations for blind people on their way to the hospital. These thieves promise to get the prospective patients into the hospital, then disappear after being paid.

Corruption is not considered wrong if it goes undetected, it seems; small wonder the hospital hires only Christian Africans. Even these educated Christian Africans, however, asked Dr. Rock a disturbing question: "Why did you leave a money-making practice to come over here? What are you going to gain?"

It seems to Dr. Rock that although they are suspicious of white people, Africans still consider medicine synonymous with white doctors. All white people are considered doctors, and practically every missionary home in Africa is a first aid station. One woman, probably the equivalent of a licensed practical nurse, treated every day 200 to 300 patients with all kinds of diseases, including, hernias, tumors, and ulcers.

Dr. Rock's moments of visiting with Dr. Schweitzer at his writing table and listening to him read psalms after dinner are past. He no longer sees 300 to 600 patients in the morning, nor does he perform 15 to 20 surgeries in the afternoon. But back in Austin the young ophthalmologist is still busy with missionary activities to aid work in Africa. Through the group of physicians and dentists who form the Christian Medical Society, he continues his effort to bring better eye care to the millions in this primitive region. The story he recounts of his three months there offers a new insight to audiences who hear Dr. Rock speak and who see his fascinating color slides of Kano and Lambaréné.

Recent Deaths

DR. L. C. WAYLAND

Dr. Levi Clarence Wayland, pioneer Plainview physician, died in Santa Barbara, Calif., on Jan. 27. The 85-year-old physician and his wife had gone to California only recently to be with their son, Dr. Newton Wayland. The elder physician had undergone surgery about two weeks before his death.

Born Jan. 2, 1879, in Randolph County, Mo., he was the youngest of the six children of Joseph H. and Catherine (Gates) Wayland. He with two of his brothers came to the Texas plains to make their homes. One of his brothers was also a physician, Dr. J. H. Wayland, who practiced many years in Plainview. Dr. L. C. Wayland, however, probably practiced longer than any physician so far in the northwest Texas area. He was active more than 60 years.

Dr. Wayland went to the public schools in Salisbury, Mo., and after receiving his bachelor of arts degree from Salisbury Academy there, he came to Texas. He opened a drug store in Lubbock where there were then only four stores. But he left the state soon afterwards to enter the Kentucky School of Medicine, now the University of Louisville. After receiving his degree and interning at Louisville City Hospital, he came back to Texas to practice in Plainview. In 1906 he did postgraduate work at Tulane University in New Orleans.

He served as county health officer for two different periods, 1907 to 1918, and from 1937 until 1959.

He was an honorary member of the Hale-Floyd-Briscoe Counties Medical Society and of Texas Medical Association. He was a Methodist and a Mason.

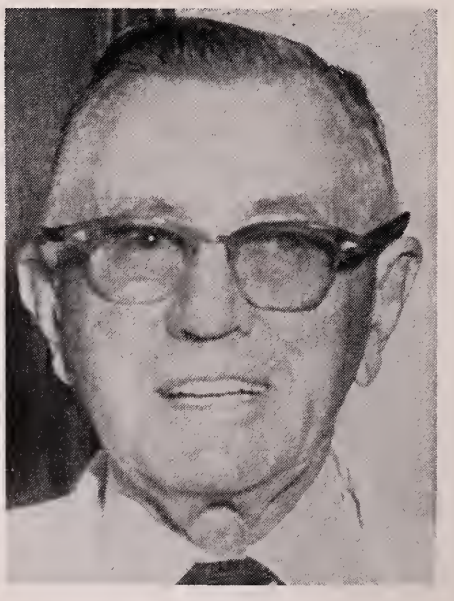
In addition to his practice, he for many years operated a farm.

He married Constance Newton in 1907 at Seymour. With her and their son, Dr. Wayland is survived by a daughter, Mrs. Louis Rader of Darien, Conn., and five grandchildren.

DR. U. B. OGDEN

Dr. Urial Bevis Ogden and his wife, Florice, were killed Feb. 20 in the crash of their plane at La Isla de Las Mujeres, Yucatan, Mexico. The physician had practiced medicine in Pleasanton since 1946.

Dr. Ogden, 57, was born in Call, Tex., on Oct. 19, 1906. His parents were Dr. U. B. Ogden and Lottie Ogden. He was educated in the Kirbyville schools and attended the University of Texas in Austin. He received



DR. L. C. WAYLAND

DR. U. B. OGDEN

his M. D. degree from the University of Texas Medical Branch in Galveston in 1938 and interned the following year at St. Mary's Infirmary in Galveston.

He began his practice in Victoria in 1939 and left it to go to war in 1942. He served four years in the U. S. Army, and upon his return to civilian life, he set up practice in Pleasanton where he had lived ever since.

He married the former Virginia Blackburn of Victoria in 1937. This marriage ended in divorce, and on Dec. 26, 1946, he married Florice Brite of Pleasanton in San Antonio.

Dr. Ogden was a former staff member at Santa Rosa and Baptist Memorial Hospitals in San Antonio and was chief of staff of Mercy Hospital of Jourdanton before his death. He was president of the Pleasanton Rotary Club, a member of the Atascosa County Medical Society, Texas Medical Association, American Medical Association, American Academy of General Practice, Flying Physicians Association, board of directors of Alamo Savings and Loan Association in San Antonio, Aberdeen Angus Association, Pleasanton school board, Knife and Fork Club of San Antonio, and First Methodist Church of Pleasanton.

Survivors include two sons, U. B. Ogden III, Victoria, and William J. Ogden, Pleasanton; three daughters, Mrs. Herman Vogt, Victoria, Sue Ann Ogden, Calif., and Sandra Lee Ogden, Pleasanton; and one sister, Mrs. E. C. Calhoun, San Antonio.

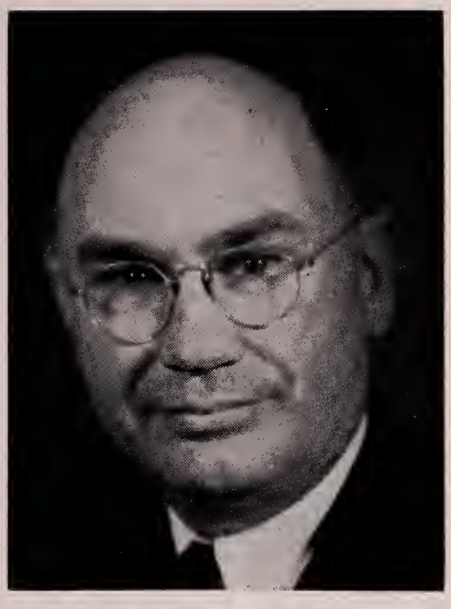
DR. C. L. PRICHARD

A longtime resident physician and surgeon of Abilene, Dr. Clarence L. Prichard, 76, died Jan. 19 after a long illness.

Associated at one time with the late Dr. J. M. Alexander, Dr. Prichard was in active practice until two years ago.

Born in Boonville, Miss., his parents were John C. and Lydia Bell Prichard. He attended Anson public schools and West Texas State University in Canyon. He received his B.A. degree from Hardin-Simmons University in Abilene where he later served as team physician.

He was graduated from Baylor University College of Medicine in 1922 and completed his internship at Harris Hospital in Fort Worth. He began his practice in Abilene in



DR. C. L. PRICHARD

1924. Throughout his career, he attended a number of postgraduate clinics.

In 1919, he married Ethal Hemphill of Waco.

A Mason and a Baptist, Dr. Prichard was a former chairman of the Masonic Chapter Council. He also served in the Texas State Guard from its beginning to 1957 and was a captain.

His hobbies were hunting, fishing, and golfing, and his business activities included farming and ranching. He belonged to the Lions Club. His philanthropies included financing young college students.

A former chairman of Hendrick Memorial Hospital Staff, Dr. Prichard was a member of the Taylor-Jones Counties Medical Society (and its president in 1928), Texas Medical Association, Southern Medical Association, American Academy of General Practice, American Medical Association, and a Fellow of the American Geriatric Society.

He had served as district secretary of the Taylor-Jones Counties Medical Society, and in 1960, he was elected to inactive membership in TMA.

Survivors include his wife; four sisters-in-law, Mrs. John A. Prichard of Dallas, Mrs. F. E. Prichard of Anson, Mrs. J. Frank Prichard of Fort Worth, and Mrs. H. D. Prichard of Wichita Falls; three nieces, Mrs. W. W. Whitcher of Corpus Christi, Mrs. Chester Riggs of Dallas, and Mrs. W. G. Prichard of Abilene and a nephew, Dr. John Franklin Prichard of Fort Worth.

DR. J. I. ALLEN

Dr. Joseph Israel Allen of Bloomburg died in a Texarkana hospital on March 21. He was 87.

Well known throughout the Four States Area, he had practiced medicine in Bloomburg since 1906. He was elected to honorary membership in Texas Medical Association through the Cass-Marion Counties Medical Society in 1957.

Dr. Allen first practiced in the Indian Territory (now Oklahoma) in 1905. He returned to school and received his degree from the Memphis Hospital Medical College, now the University of Tennessee College of Medicine, in 1906. He and 12 other members of that graduating class were honored for 50 years of service. Dr. Allen received a "Golden T" and a certificate of merit.

At the time of his death, Dr. Allen was president of the Bloomburg State Bank, a position he had held since 1930.

He was active in the community and in the First Baptist Church. He had been a member of the Masonic Lodge for 60 years, receiving his 50-year pin in 1954. He was also an active member of the Lions Club.

He is survived by his wife; two sons, Joe Bailey Allen and James I. Allen, Jr., both of Bloomburg; two daughters, Mrs. W. G. Schuhmann and Mrs. J. C. Jones, also of Bloomburg; a sister, Mrs. Pearl Davis of Atlanta, Tex.; nine grandchildren; and nine great-grandchildren.

DR. L. M. SELLERS

Dr. Lyle M. Sellers, Dallas specialist in otology, laryngology, and rhinology, died May 4.

Dr. Sellers had practiced in Dallas since 1925 and was chief emeritus of the Otolaryngology Department at Baylor Medical Center.

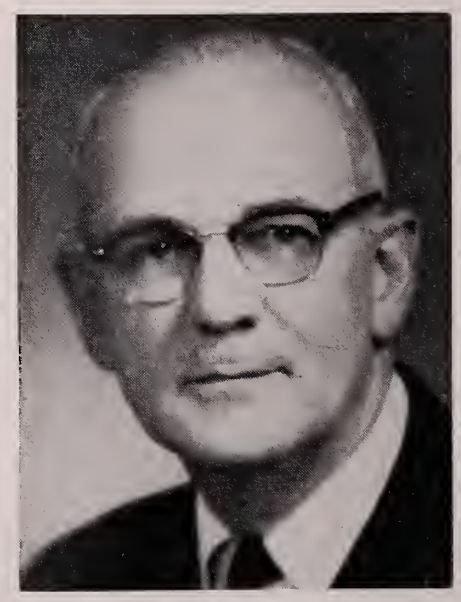
He was born in Kansas City, Mo., Aug. 29, 1894. He attended the University of Chicago and received his M.D. degree in 1919 from the University of Kansas School of Medicine. Following this, he studied the treatment of ear, nose, and throat disorders with Dr. A. J. Lorie in Kansas City.

He was a member of the American Otological Society, Inc., American Academy of Ophthalmology and Otolaryngology, American Medical Association, American Society for the History of Medicine, Triological Society, Texas Society of Ophthalmology and Otolaryngology, Texas Medical Association, Dallas County Medical Society, Dallas Southern Clinical Society, and a fellow of the American College of Surgeons.

He belonged to the Grolier Club and Temple Emanu-El.

Survivors are his wife, Ruth, and a daughter, Mrs. Herschel Jaffe, both of Dallas.

DR. J. I. ALLEN



DR. L. M. SELLERS

COMING MEETINGS

American Medical Association, Annual Meeting, San Francisco, Calif., June 21-25, 1964. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

JUNE

American College of Chest Physicians, San Francisco, Calif., June 18-22, 1964. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American Dermatological Association, Hawaii, June 27-July 2, 1964. Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Sec.

American Neurological Association, Atlantic City, June 15-17, 1964. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

Texas State Board of Medical Examiners, Fort Worth, June 22-24, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

JULY

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, San Francisco, May 12-16, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, New York, Oct. 24-29, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, Los Angeles, Oct. 8-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Oct. 2-3, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Gastroenterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Medical Association Congress on Occupational Health, Houston, Sept. 26-27, 1964. Dr. Henry F. Howe, 535 N. Dearborn St., Chicago, Sec.

American Medical Women's Association, New York, Oct. 16-20, 1964. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., New Orleans, La., Oct. 8-10, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Chicago, Nov. 6, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons. Dr. W. F. James, 1615 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico. Dr. C. Kalil, 200 East Monterey Way, Phoenix, Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwest Regional Cancer Conference, Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Waco, Nov. 8, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. John W. Chriss, 2436 Morgan, Corpus Christi, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, San Antonio, September, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. D. Hoehn, Doctor's Bldg., Fort Worth, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, San Antonio, May 1-2, 1965. Dr. Fred P. Thomas, 2342 Maroneal, Houston, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section, San Antonio, April 29-May 2, 1965. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc., Houston. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Maria P. Hathaway, 1012 State Office Bldg., Austin, Exec. Sec.

Texas Surgical Society, San Antonio, Oct. 4-6, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, Sec.

United States-Mexico Border Public Health Association, Los Angeles, April 26-29, 1965. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

TEXAS
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Devoted to the interests of the medical profession and public health in Texas

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The Splintering of the Profession

ONE OF MEDICINE'S most difficult problems—as expressed at the American Medical Association's 1964 Annual Meeting—is the splintering of the profession. The division of one group against another has weakened the profession's overall effectiveness.

The spirit of cohesiveness and unity within the profession has been impaired by a welter of meetings and by a scattering of interests. Intramural problems in some areas of the country have pitted "town against gown," specialists against general practitioners, hospital and corporate practice against private practice, and the salaried physician against those who work for fees.

While some of these problems have been evident in Texas, this state has been far more fortunate than most others. Specialty societies, related medical organizations, and the Texas Medical Association have developed a wholesome and productive relationship.

This achievement has not developed by happenstance. The problem of splintering has been tackled directly and forcefully. In 1954, Dr. F. J. L. Blasingame, then President of TMA, contacted every specialty and related medical organization in the state. He outlined the problem concisely. He pointed out that regardless of hospital staff responsibilities, specialty society meetings, and postgraduate assemblies, no group could take the place of a basic medical organization, with its county, state, and national components. Dr. Blasingame emphasized that it was the duty of every physician to contribute to the foundation of American Medicine. He invited the specialty societies and other physician organizations, while retaining their autonomy and carrying out activities for which they were established, to join the state association in mutual endeavors to strengthen the entire profession.

Dr. Blasingame's initiative has paid handsome dividends. Twenty-seven specialty and related societies now meet concurrently at the Annual Session of the Texas Medical Association. Through collaborative efforts and sharing guest speakers, they are

presenting the most comprehensive scientific program in this section of the country. These joint efforts have been responsible for doubling Annual Session attendance in ten years; registration at the 1964 meeting in Houston reached a record 4,093. This achievement has come at a time when most other large state societies have reported that annual meeting attendance is remaining stable or actually is decreasing.

This wholesome relationship with specialty and related societies also has produced several other fine by-products. Many of these societies are working together in support of scientific and public health programs such as hospital accreditation and the health hazards of cigarette smoking. Officers of specialty societies are contributing editorials and articles to the *Texas State Journal of Medicine*, thus enhancing its value to the entire profession. Officers of the Texas Medical Association as well as specialty and related medical societies recently conferred in Austin to coordinate their legislative objectives and programs in anticipation of the 59th State Legislature next January.

Texas physicians can be extremely proud of this relationship which has proved to be beneficial to specialty societies, related medical organizations, the state association, and most importantly, the entire profession.

—C. LINCOLN WILLISTON, Austin,
Executive Secretary,
Texas Medical Association.

Editor's Note: Mr. Williston is the immediate past president of the Medical Society Executives Association. The foregoing is based on a portion of his presidential address, which he gave at the MSEA meeting in San Francisco, Calif., in June.

Sunlight and Skin Cancer

SUNLIGHT is the major cause of skin cancer, and many cancers can be prevented by shielding the skin from prolonged exposure.

Prolonged exposure to the sun has an aging effect on the skin. A biopsy taken from the hand or face will show degeneration when compared with another taken from the buttock where sun exposure never occurs. This degeneration is great in the faces of farmers and others whose occupations keep them continually in the sun. People who are exposed only in the summer months are saved a certain amount of damage because their skin is able to recover during the winter months.

The number of hours in the sun has a cumulative effect on those exposed, something to be considered in Texas where we have long hours of bright sunlight from April to November. It also has been shown that the heat of the sun as well as ultraviolet light is important.

Skin cancer is the end result of aging of the skin, and since aging is increased by prolonged exposure to sunlight, some people will be more susceptible than others. Skins deficient in melanin are more easily damaged by the sun, and colored skins are so free of skin cancer that we consider them "immune." Olive skin is less prone than fair to skin cancer.

Privileged people who stay tan all summer and then go to a winter resort to continue the tan are candidates for skin cancer. Respite from the sun in the winter months prevents damage to the skins of most people.

There are substances that will protect many people from the harmful effects of the sun. Zinc oxide, talc and petrolatum are physical barriers. Chemical substances, mostly para-aminobenzoic acid derivatives found in many commercial sun tan lotions and creams, are more effective in that they absorb many of the sun's rays. Most, however, will allow enough of the sun's rays to pass through so that the individual will tan. They do not give complete protection.

An old preparation, red veterinary petrolatum (RVP), has been brought back into use. During World War II, it became standard equipment in life rafts and vehicles engaged in tropical warfare. It is applied to the skin and then rubbed off; the remaining film gives protection from sunburning ultraviolet rays for up to 20 hours of strong sunlight. RVP is stable and inexpensive, and adheres to the skin during swimming.

This is the time of year when we should instruct our patients about the dangers of excessive sunlight exposure. They should be warned that skin that burns easily will also be damaged easily by the sun, and that they should use sun screens to protect themselves if they have to be in the sun. They should be told that sun exposure early in the morning and late in the afternoon does not cause as much damage from ultraviolet light as exposure in the middle of the day. They should be instructed to wear hats and other protective clothing. Our biggest responsibility, however, is to discourage the fad of sun burning to the point of a "baked on tan," so

popular among our young people. Merely to point out that this will cause premature aging of their skin is often enough. To emphasize that skin cancer may develop when they become older should be the final argument.

—J. Franklin Campbell, M.D.,
Fort Worth.

The Cardiac Child in School

IN THE REGULAR school setting, children with cardiac disorders react much the same way as normal children do. In Houston, special programs are available for those who need them, but the children are moved from hospital, homebound, and special classes back into the regular schools as soon as their condition permits.

Impetus was given this practice by the findings of a Texas Heart Association committee, working in cooperation with three members of the supervisory staff of the Houston Independent School District, to consider the needs of the cardiac child in school. Because adequate health information about each child is obtained as he enters school, it was relatively easy to find the number of children with cardiac disease.

Of the 371 cases known to the school district's health department, 89 per cent were under the care of a physician, and of the remaining 11 per cent, several had been dismissed by the physician after heart surgery.

Many of these children are able to participate in the regular school programs, and some attend regular classes but do not participate in all physical activities, such as climbing stairs or playing certain games. Others, still in regular classes, are assigned no physical education at all, and must have rest periods during the day.

More severely limited children are placed in special classes for the handicapped, where they work only in one room and have toilet facilities and a quiet room for rest nearby. There are no stairs, and the amount of rest and exertion is indicated by the physician. The few children who are hospitalized resume their studies when they are able to attend the hospital school. Many physicians believe that starting learning experiences as soon as possible is good therapy for the child.

As for attendance, children with heart disorders generally have excellent records; this probably can be attributed to the fact that if a cardiac condition causes the illness, the child is placed in a special class appropriate for his physical capabilities.

The school marks of these children were found to be unrelated to their heart conditions, and they walk to school, ride city or school buses, or are brought in the family car in about the same percentages as other children.

Close cooperation between the health department, private physicians, hospital, and school personnel have resulted in immeasurable benefits for children with heart disease who might otherwise have had far more limited opportunities for normal education.

—Alberta Baines,
Assistant Superintendent,
Coordination of Curriculum,
Houston Independent School District.

Medicare Is Not Dead Yet

ALTHOUGH the House Ways and Means Committee adroitly sidestepped a showdown vote on the King-Anderson bill, the issue of medical care for the aged through Social Security remains volatile. The Senate still could attach such a program to other legislation.

What the House actually did—in lieu of acting on medicare—was approve a 5 per cent across-the-board increase in social security cash benefits now being paid. *It also approved mandatory entry into the Social Security System of about 150,000 doctors and interns who are exempt from the system under present law.*

To finance the proposed increase in benefits, payroll taxes would be increased gradually until 1971, at which time the rate of 4.8 per cent on the employee's first $5,400 of earnings would prevail. The current rate is 3.625 per cent on the first $4,800. A self-employed person now pays 5.4 per cent Social Security tax on his first $4,800 of earnings. The proposed new rates would be for the first $5,400, and would be raised to 5.7 per cent Jan. 1, 1965, to 6 per cent in 1966, to 6.8 per cent in 1968, and to 7.2 per cent in 1971.

The bill is expected to be considered on the floor of the House, under a closed rule (no amendments permitted from the floor), about July 20. If approved, it will move on

to the Senate Finance Committee where amendments could be made to include medical care for the aged. Such an amendment might also come from the floor of the Senate.

There is the possibility that a medicare rider could be tacked on to another piece of legislation, or that it could be completely ignored.

The Texas Medical Association is on record as being firmly opposed to compulsory Social Security coverage for all physicians. Doctors who wish to express their opinions on this and other aspects of the proposed legislation, may write to Sen. Harry Byrd, Chairman, Senate Finance Committee, Washington, D. C.

Society Must Be Protected From Unethical Physicians

IT IS REASSURING that only a few doctors are a discredit to our profession, but one rotten apple can spoil the whole barrel. It is the duty of each medical society to utilize its authority to seek out physicians whose actions are harmful to the public confidence in us.

The average person usually forms his judgment of all doctors through his contact with only one of them. If that physician is unethical in his practice it will take a great deal of kindness and efficiency by other physicians to overcome the poor impression that the patient has gained from his first encounter. Whether we like it or not, each of us is a salesman for our profession, and the impression we make is an advertisement, good or bad.

God has granted us the unique privilege of ministering to His children—this is a sacred trust which must not be abused. If we do not fulfill our obligation to protect society from unethical physicians, then we will all suffer the consequences. That is the real challenge facing our medical societies today.

The Offenders

I believe that unethical physicians can be divided into two groups:

1. *The Uninformed.*—They have never really received the message but do have the inherent ability to be ethical. This group can be salvaged if convinced that there is a better way, and we are obligated to direct their rehabilitation. Many of these physicians have never read the "Principles of Medical Ethics" and certainly are completely ignorant of the material published in "Opinions and Reports."

2. *The Pathological.*—The physicians with fulminating pathologic ethics have a completely warped attitude toward others, cannot be rehabilitated, and will utilize every loophole that can be found in our disciplinary procedures. The etiology of this condition is worship of the almighty dollar, and the predominant symptom is self-aggrandizement. The only treatment for this condition is radical amputation from the profession. There are two opinions of the Judicial Council that are usually applicable in this type of case:

> The charging of an excessive fee is unethical and contrary to Section 7 of the Principles. The physician's fee should be commensurate with the services rendered and the patient's ability to pay.
>
> One of the strongest holds of the profession on public approbation and support has been the age-old professional ideal of medical service to all, whether able to pay or not. That ideal is basic in our ethics. The abandonment of that ideal and the adoption of a principle of service only when paid for would be the greatest step toward socialized medicine which the medical profession could take.

Many excessive fees could be eliminated by asking ourselves if we have really earned the fee. Although the Principles give us freedom of choice in accepting patients, poverty should not be a criterion in rejecting patients. Charity means giving from the heart, not unpaid bills.

Interpreting the Principles

Many physicians have difficulty in the interpretation of the Principles when ap-

Dr. Robert Mayo Tenery, Past President of Texas Medical Association, presented this discussion before the AMA Legal Conference in Chicago on April 16.

plied to specific problems, especially in matters that fall into what we loosely call "gray areas." Some of the solutions may seem simple to those familiar with the opinions of the Judicial Council, but that frequently is not true for the average practicing physician. For my own use and for talks to doctors I have devised a formula that usually works fairly well even for those who do not know the principles. This formula consists of three measuring rods which can be applied to any problem of ethics:

1. *Measure of Intent.*—We must ask ourselves if the intention of the action is for the benefit of the patient, the public, the profession, or ourselves. Naturally, the primary intent must be for the benefit of the patient. If it is for the physician's own benefit, then there is a good chance that the practice is unethical. A simple example is that of holding "open house" for the opening of a new clinic. As the Judicial Council has stated, "If motivation for holding open house stems from civic and natural pride in having made a contribution to the community and to medicine, then the practice might not be objectionable." This same measure can be applied to almost every section of the Principles.

2. *Measure of the Golden Rule.*—This can be applied to the rules of medical etiquette and certainly to our relationships with patients and their families. The applications are obvious.

3. *Measure of Local Customs and Laws.*—The opinions of the Judicial Council have necessarily left many items to such decision because of variations from state to state and even from county to county. I feel, however, that there is a need at the AMA level for a minimal standard publicity code which could be adopted by local county societies with additions as dictated by local needs.

In America, the law considers an individual to be innocent until proved guilty, and we follow this same rule in disciplinary actions against physicians who may have violated the "Principles of Medical Ethics." This is as it should be; however, it does present serious difficulties in our efforts to prevent certain unethical practices. As physicians, we may know in our own minds that another doctor is guilty of a violation, yet we are frequently unable to produce sufficient evidence to prosecute the offender in a trial before his county medical society.

The law does require that we obtain permission, that is, a license, before we are allowed certain privileges such as driving a car, practicing medicine, establishing a corporation, and, if we do these things without a license, we are guilty of violating the law.

The Principles allow physicians certain privileges so long as they are in the best interest of the patient and so long as there is no exploitation of the patient. We could avoid certain offenses if the interpretations required that the physician obtain permission from his county medical society *before* utilizing these privileges. For example, Section 7 of the Principles states that a physician may dispense drugs provided it is in the best interests of the patient, but exploitation is almost impossible to prove because every physician has the right to prescribe medications that in his judgment are best for his patients. Why not require that a physician obtain permission from his county medical society *before he enters into the ownership of a pharmacy?* To obtain such license, he would have to prove that ownership would be in the best interests of his patients. This would switch the burden of proof from the county medical society to the physician who is seeking a privilege.

The Texas Plan

In Texas we have approached the problems of ethics from three directions: (1) provisional membership, (2) education, and (3) uniform disciplinary procedures.

Provisional Membership.—In April, 1956, after two years of study by the Board of Councilors and after long debate in reference committee and on the floor of the House of Delegates, the by-laws of the Texas Medical Association were amended to provide a mandatory two-year period of provisional membership. This not only applies to new applicants but also to physicians who transfer from one county society to another and to members who may have been dropped for such reasons as delinquent dues.

Following the initial enactment, several amendments were required to provide for unforeseen problems, and for about three years there were efforts to excuse the transfer members from this requirement; however, by 1960 the procedure became routine, and there have been essentially no changes since then. The Board was firmly convinced that this must apply to transfer members

because that group contains "drifters" who may be medical misfits. It is true that many ethical physicians suffer some hardship as the result of this requirement, but those who are really dedicated to the Association are willing to follow the rules for its betterment.

The provisional member has all of the privileges of his society except the right to hold elective office, endorse applications for membership, and serve as a delegate or alternate delegate to the Texas Medical Association. This has eliminated the practice in some small societies of electing an applicant to membership and to the important position of county society secretary at the same time.

Applicants for provisional membership are voted upon by the society after recommendation by the Board of Censors, and at the end of the provisional period are voted into regular membership in the same manner. At any time during this period a provisional member can be dropped by a two-thirds majority vote of the society upon the recommendation of the Board of Censors with no cause specified. The by-laws also state that "the Board of Censors will provide supervision and guidance in matters of medical ethics and etiquette for provisional members."

During the past eight years there have been several instances when physicians have been denied regular membership because of unethical practices, and without provisional membership a trial by the society would have been needed to expel them.

Education.—At the same time that provisional membership was adopted it was also decided that every provisional member must attend a one-day orientation program presented by the Texas Medical Association to be eligible for regular membership. This applies to transfer members as well as new members unless they have previously attended. The program is given three times each year and covers such subjects as ethics, etiquette, medical organization, legislation, malpractice, and medical economics. To date, more than 3,000 physicians have attended this program—this represents one-third of our membership, and it has certainly resulted in better informed members who have more interest in the work of the Association.

For the past 11 years a similar educational program has been presented to the senior classes of our three medical schools. The purpose of the program is to provide students with information which will make their introduction into the practice of medicine a little easier. Officers and staff of the Association present practical aspects of medical practice which are not included in the school curriculum. Addresses are given on public relations and public opinion, medical ethics and etiquette, legal aspects of medical practice, patient relationships, and the comparative advantages of solo, partner, and group practice. As part of this event there is always a luncheon or a dinner to which both the senior students and their wives are invited.

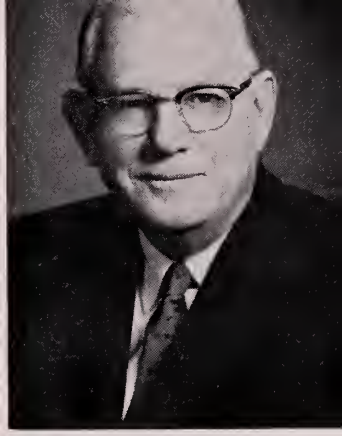
We believe that the Medical Student Day and Orientation Programs are providing our new physicians with a much better understanding and appreciation of their ethical and moral responsibilities as physicians.

Uniform Disciplinary Procedures.—Physicians are usually poorly versed in the law; thus, uniform disciplinary procedures which spell out each step are essential if county medical society verdicts are to stand up when appealed to higher authority. In 1960, this portion of our by-laws was completely rewritten by the Board of Councilors and our legal counsel. Each procedure is clear and the constitution and by-laws of each component county society must be reviewed and approved by the Board of Councilors so that there is no conflict with the Association constitution and by-laws. We believe that the revision has prevented some embarrassing and potentially dangerous situations.

Conclusion

If free enterprise in the practice of medicine is to survive, then our art must be rid of any tarnish that interferes with the relationships to those who do us the honor of entrusting their lives to our care. There is no single panacea for all members of the profession. Each physician must evaluate himself from the patient's point of view and then prescribe corrective measures. Nevertheless, one cardinal ingredient stands above all—dedication to the practice of ethical medicine.

—Robert Mayo Tenery, M.D.,
Waxahachie, Past President,
Texas Medical Association.

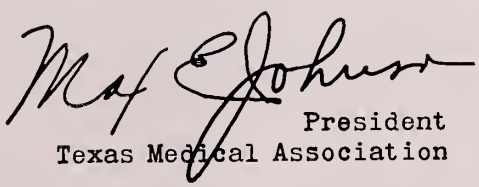


ACCEPTANCE OF REHABILITATION

Physicians in America are vitally interested in rehabilitation and most stand ready to assist. Their concern is reflected in the action of the American Medical Association and the various state associations which have appointed committees to study the problem, and in the large number of individual physicians serving on many rehabilitation boards and committees.

There are some physicians today, however, who do not understand the programs; they need education in this field. The doctor, by concept and training, is an individual entity and does not respond readily to group situations and team approaches to medical problems. The modern concept of rehabilitation involves a whole community with a team composed of professionals, technicians, administrators, specialists, and laymen. The physician is an important member on this team and must be made aware of his special role in this community program. In many instances, the physician, seeing that this program is instituted and carried out by laymen, does not concern himself with discovering the existing or planned program, services, and facilities available to his patients. Physicians have been accused of a lack of interest in rehabilitation or of reluctance in referring patients to rehabilitation centers. Recent surveys show that this is not true and that physicians are referring a fair percentage of cases to rehabilitation centers.

The physician's basic training demands that he at all times endeavor to restore the sick and injured to full health. Modern medicine has as its goal the treatment of the whole man and it is no longer satisfied in treating only the disease. The physician realizes that man, in reality, is many things. He is a physical, mental, psychological, and spiritual being. Impairment of any one component may have profound effect on the other parts. Treatment of the whole man requires not only the cooperation of physicians and those trained in all phases of rehabilitation — individuals, communities, agencies, hospitals, and rehabilitation centers — but requires also the correlation and coordination of work done by these interested groups.



President
Texas Medical Association

Differentiation of Pulmonary And Cardiac Breathlessness

REVIEW ARTICLES

LIKE PAIN, pleasure, sorrow, or fatigue, dyspnea is a subjective sensation. As such, it is difficult to define and quantitate. The degree to which it is disabling or even appreciated is greatly variable in different individuals. Nevertheless, it is a common complaint of people seeking medical help, and the physician must evaluate it to treat it properly.

Many theories have been offered as to the mechanism of dyspnea. Several of the better ones include: (1) fatigue of intercostal muscles,[3] (2) dyssynergia of intercostal muscles and diaphragm,[13] (3) excessive work of breathing,[7] (4) excessive oxygen cost of breathing,[2] (5) ratio of actual ventilation to maximum breathing capacity,[9] (6) variations in intrapleural pressure,[5] (7) elevated carbon dioxide tension,[8] (8) hypoxemia,[11] (9) pulmonary vascular engorgement,[6] (10) decreased respiratory center inhibition by stretch receptors,[12] (11) increased physiologic dead space, (12) altered balance between demand for and effort of breathing,[1] (13) length: tension inappropriateness.[1]

These definitions appear to be efforts to translate pulmonary physiologic measurements into explanations for the symptoms. Several comments may be made regarding these theories:

1. Predominantly, physiologists attempt a definition.

2. No one definition can suitably include all possible causes for dyspnea.

3. These definitions are difficult to translate into medical diagnoses.

4. Accepting their inadequacy in establishing a diagnosis, they also do not form a good basis for symptomatic treatment.

5. In summary, they are of limited assistance in the management of the problem of breathlessness.

Practically speaking, breathlessness is what a person says he is, which leaves us with the contemplation of a subjective sensation. As physicians, we can initially eliminate most of those circumstances causing breathlessness such as bona fide physical fatigue, passion or excitement, which are generally not offered in the nature of a complaint. By the same reasoning, we must assume that breathlessness sufficient to bring a patient to the physician is not without cause and that medical disease must be suspected.

By virtue of their frequency and severity, diseases of the heart and lungs are commonly prime suspects. The importance of anxiety in the genesis or the magnitude of this symptom cannot be overlooked, and one cannot forget that shortness of breath is a common, subjective expression of the sensation of one who is merely weak. Such weakness may be the result of malnutrition, chronic infection, anemia, metabolic diseases, and malignant diseases which are therefore possible etiologies for complaints of breathlessness. The need to differentiate pulmonary from cardiac etiology arises when age or physical findings suggest the presence of either or both.

Features of the history may be helpful but are not always diagnostic. The rapidity of onset can be equally striking in pneumonia, acute asthma, spontaneous pneumothorax, or atelectasis as it is in acute pulmonary edema or acute pulmonary embolus. On the other hand, it may be equally as slow in chronic emphysema, fibrothorax, or ankylosing spon-

From the Cardiovascular Section and the Department of Clinical Research, Scott and White Clinic, Temple.

The authors discuss the value and importance of the Valsalva maneuver in differentiating pulmonary from cardiac breathlessness. The maneuver during the routine physical examination is described as one of the most consistently helpful sources of information in such diagnosis.

PER H. LANGSJOEN, M.D.
DON J. LYNCH, M.D.

dylitis, as it is in chronic decompensation or multiple microembolization of the lung. The patient with cardiac asthma may wheeze or rattle as loudly as the patient with bronchial asthma or emphysema. Diaphragm attachment pain or the pain of pleural irritation is easily confused with the pain of coronary insufficiency or pulmonary hypertension. Cardiac decompensation without weight gain or orthopnea is fairly common. The patient with severe obstructive pulmonary disease may have edema without being in heart failure, caused by immobility and the prominent high intrathoracic pressure intermittently present.

Even the findings of evident cardiac or pulmonary disease on physical examination, roentgen-ray studies, electrocardiograms, or pulmonary function studies do not insure that the diagnosable disease is specifically the cause of the breathlessness. In short, there is no absolute way of differentiating cardiac from pulmonary breathlessness in all cases. Since highly refined, sophisticated, and at times costly, studies are frequently incapable of making a differentiation, it is sometimes wise to make the most of the simple, cheap, and available observations based on a few fundamental facts of cardiopulmonary physiology.

Basic Considerations

Adequate circulation is dependent upon the function of four separate, but closely integrated pumping units: the left heart, the muscles of the extremities, the right heart, and the bellows action of the thorax. Much can be learned by considering the importance of this bellows action of the thorax because of the fortunate fact that the pulmonary vascular bed is an expansile structure located within this thorax and responsive to variations in pressure within the thorax.

In good health with quiet resting respirations, the cardiac output is reasonably stable. Nevertheless, on pressure tracings taken within the pulmonary artery during cardiac catheterization in normal patients, definite respiratory variations are usually seen. It is reasonable to conclude that the bellows action of the thorax makes some contribution to the pulmonary blood flow at all times by virtue of its effect on the venous return to the right heart and on the resistance to right ventricular output. The importance of this bellows action is shown clearly in slightly abnormal circumstances in the tracing of an intra-arterial pressure recording done during surgery for a pheochromocytoma in an otherwise healthy 24-year-old woman in whom respirations were being maintained by positive bag pressure (Fig. 1). An evident decrease in cardiac output as manifested by a significant 25 mm. drop in systolic blood pressure followed shortly after the introduction of a positive intrathoracic pressure.

The equivalent of this situation can be easily accomplished by a simple Valsalva ma-

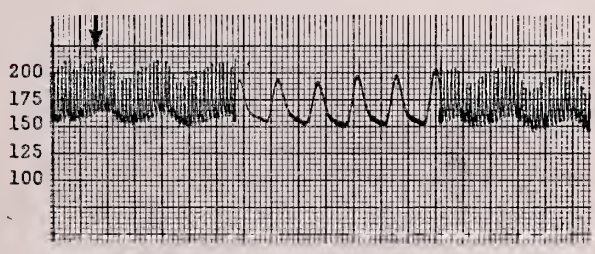


Fig. 1. Arterial pressure recording with respirations being maintained by intermittent bag compression as indicated by the arrow, showing significant pressure changes.

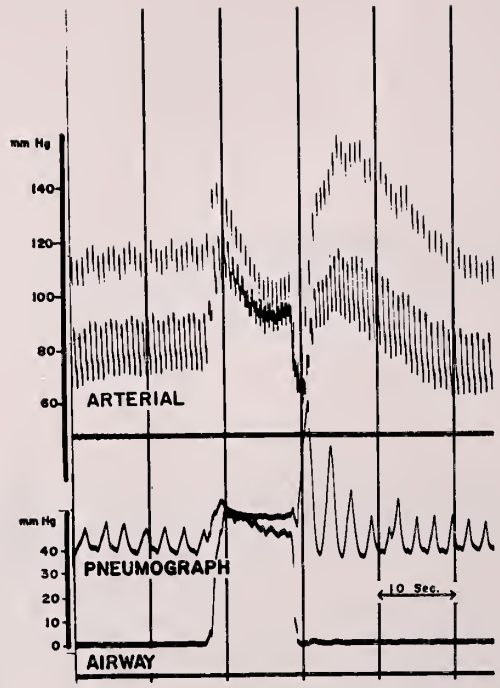


Fig. 2. Normal Valsalva response showing the four characteristic phases in a healthy young female.

neuver. In a normal person this maneuver will initially increase slightly the venous return to the left heart by increased pressure on the pulmonary vascular bed. It will also slightly increase resistance to the right ventricular output and will significantly restrict the venous return to the right heart. The release of this positive intrathoracic pressure will initially permit the pulmonary vascular bed to rapidly reconstitute its original volume at the expense of the venous return to the left heart followed shortly by an increase in the venous return to the right heart with a resulting eventual increase in cardiac output.

The normal vascular response to this maneuver has been well described with four characteristic phases:[10] phase 1, sharp rise in arterial pressure; phase 2, fall in arterial pressure as intrathoracic pressure maintained; phase 3, fall in pressure below baseline as intrathoracic pressure is released; phase 4, reflex rise in pressure above baseline followed by bradycardia (Fig. 2).

Clinical Observations

In the presence of pulmonary vascular congestion, as seen in left heart failure, there

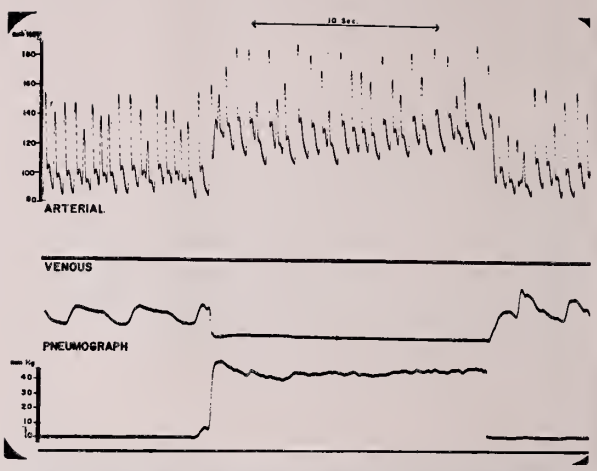


Fig. 3. Abnormal response to the Valsalva maneuver in a patient with left ventricular failure.

is a considerable increase in the blood volume of the pulmonary circulation and as a result, the Valsalva maneuver will give an exaggerated increase in the venous return to the left heart and, if the left ventricle is significantly dilated, may slightly increase its emptying which will result in a prolonged increase in cardiac output. The degree of resistance to the right ventricular output and to venous return may be reduced by the compensatory increase in venous pressure and in the right ventricular pressures, which are secondary to the left heart failure itself. The characteristic vascular response pattern is one of a sustained elevation of the arterial pressure throughout the period of the Valsalva maneuver with an absent or reduced overshoot after the release of the increased intrathoracic pressure (Fig. 3).

There are pitfalls in the interpretation of this maneuver which are obvious. If the pulmonary vascular congestion is due to severe mitral stenosis, or severe aortic valve stenosis, the fixed resistance to cardiac output on the left side may not be overcome by the increased intrathoracic pressure, and a false negative test may result. A false positive response has been seen in the presence of a large expansile aortic arch either due to aneurysm or associated with marked aortic insufficiency with significant dilatation of the left ventricle (Fig. 4). We have also noted false positive results in mitral insufficiency with massive dilatation of the left atrium (Fig. 5). A factitious positive reading may be produced if the patient performing the test actively contracts the muscles of the arm on which the blood pressure cuff is placed during the Valsalva effort. The re-

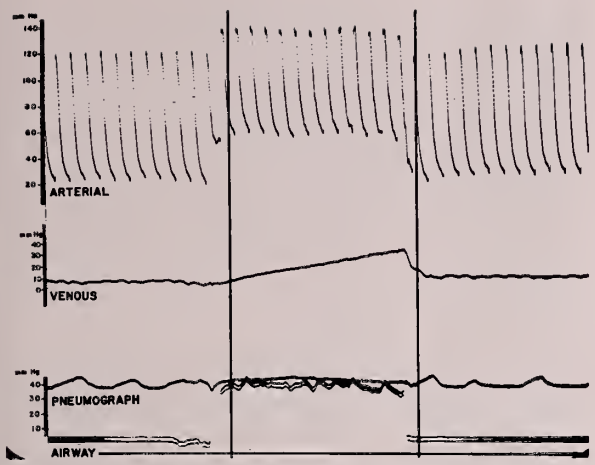


Fig. 4. Abnormal response to the Valsalva maneuver in a patient with severe aortic regurgitation and cardiac enlargement but without evidence of heart failure.

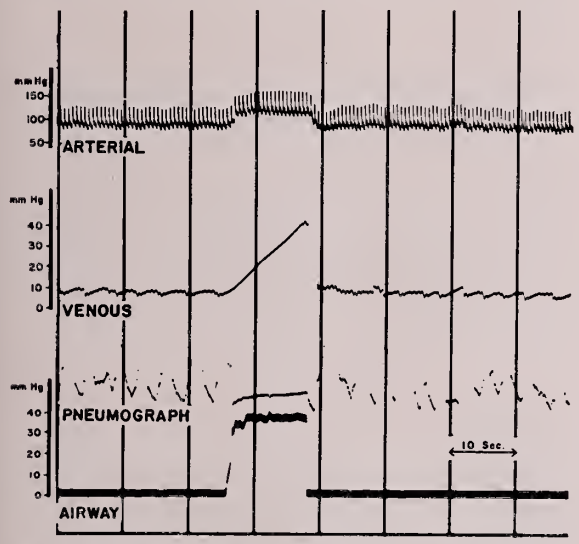


Fig. 5. Abnormal response to the Valsalva maneuver in a patient with mitral insufficiency with enlargement of the left atrium and left ventricle but who was not in heart failure.

sponse is difficult, but not impossible, to interpret in the presence of anything but a basically sinus rhythm and is, of course, totally unreliable in the presence of left to right or right or left shunts.

In some cases of acute and severe pulmonary congestion, this respiratory variation may be evident on quiet breathing. If it is associated with a grunting type respiration and cough in circumstances where heart failure is probable, it is often significant evidence of pulmonary congestion. In this latter circumstance, the lung fields may be clear because there is pulmonary congestion without pulmonary edema. The grunting

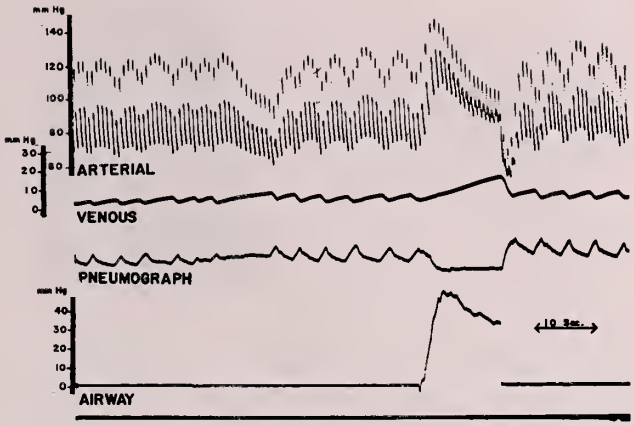


Fig. 6. Normal response to the Valsalva maneuver in a patient suffering from obstructive pulmonary disease. Variation of blood pressure on quiet breathing is exaggerated.

respirations may be merely a mechanism for increasing the intra-alveolar pressure intermittently to counteract the high pulmonary capillary pressure in order to reduce the probability of pulmonary congestion resulting in frank pulmonary edema. This principle is utilized in practice when intermittent positive pressure breathing is used for acute pulmonary congestion.

Statistically, a significant variation in the blood pressure of greater than 6 to 8 mm. of mercury with quiet respirations is more commonly associated with obstructive pulmonary disease than with left heart failure. In obstructive pulmonary disease, the bellows effect of respiration is greatly enhanced; such that the inspiratory negative pressure is lower and the expiratory positive pressure is considerably higher than normal. As a result, both pulmonary flow and the systemic flow may be phasic and at first glance may look like the pattern seen in acute pulmonary congestion with heart failure. Its differentiation lies in the fact that the pulmonary vascular bed is not engorged, nor is the left ventricle significantly dilated, and therefore, a Valsalva maneuver done at end expiration will not produce a positive increase in the cardiac output as measured by the arterial pressure recording (Fig. 6).

Problems arise mainly when both obstructive pulmonary disease and congestive heart failure are present simultaneously. When this combination is suspected, about all one can hope to do with this maneuver is to be certain as to the presence or absence of pulmonary congestion, and leave the confirmation of the pulmonary disease to other studies.

Dr. Per H. Langsjoen specializes in cardiovascular diseases and Dr. Donald J. Lynch, his co-author, is an internist. Both are with Scott and White Clinic, Temple.

The respiratory variations in blood pressure recordings due to failure or obstructive pulmonary disease may be confused with the picture of cardiac tamponade. The significant difference lies in the fact that in tamponade the blood pressure recording will be depressed from the end expiratory phase level by the act of inspiration. In pulmonary congestion, or severely obstructive pulmonary disease, the blood pressure is increased above the end inspiratory phase during the act of expiration or Valsalva maneuver. A further check on tamponade may be the failure of the venous pressure to drop on inspiration as visualized in the neck veins. This finding, however, is not always reliable since it may likewise be present in severe chronic myocardial fibrosis with significant involvement of the right ventricle.

In our laboratories, a series of 25 patients whose major problem was differentiation of cardiac from pulmonary breathlessness were studied with physiologic recording of the response to the Valsalva maneuver. The results support the validity of this interpretation of these observations.

Differential Diagnosis of Dyspnea

Twelve patients with dyspnea but without cardiac disease all had normal responses.

Seven patients who were clinically classified as cardiac had abnormal responses.

Six patients in whom a definite cardiac and/or pulmonary differentiation could not be made by other means, treated on the basis of their Valsalva responses, had satisfactory responses.

Methods

These recordings were made with an indwelling catheter in an arm vein, a Cournand needle in a radial artery, a pneumograph in place, and a mouthpiece arrangement which permitted obstruction of the airway, with a manometer to record intrathoracic pressures, all connected to strain gauges, recorded simultaneously on a physiologic recording system (Fig. 7). This arrangement is clearly cumbersome, costly, and not applicable in many cases. Fortunately, however, it can be done effectively with a little practice using only a blood pressure cuff and a stethoscope.

Initial observations must be made of the stable systolic peak pressure and any variations in respirations noted. It has been recommended that the cuff pressure then be set 20 mm. above this level to measure the Valsalva response.[4] We have found that this level will give many false negative responses and therefore have used a setting closer to 15 mm. of mercury above the peak systolic pressure. The Valsalva maneuver is then performed for approximately ten seconds. A sustained elevation of the blood pressure above this new level as set on the blood pressure cuff, is a positive Valsalva response. Within the limitations listed previously, this is good evidence of pulmonary vascular congestion, and as such, presumptive evidence of left heart failure. We have found it equally as applicable in the seated or prone position.

Conclusions

The use of the Valsalva maneuver in the course of the routine physical examination has been the most consistently helpful source of information in differentiating pulmonary

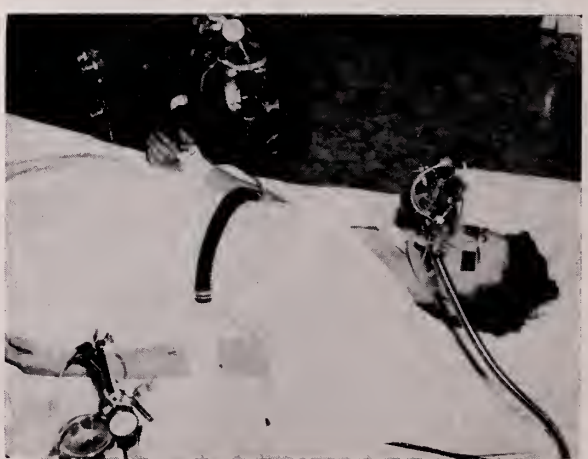


Fig. 7. Laboratory setup used to measure directly central venous pressure, arterial pressure, airway pressure, and respirations during the Valsalva maneuver.

from cardiac breathlessness in our experience. The meticulous recording of the blood pressure and an understanding of the great amount of physiologic information that can be detected thereby is one of the most important parts of an adequate physical examination, and not a chore to be performed by the office nurse.

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Fiber Glass Vaulting Pole Risky

High school pole vaulters have been warned by the AMA of the possible hazards associated with fiber glass poles due to their "apparent vulnerability to breakage."

"The potential hazard from falls and possible impaling of the vaulter justifies serious concern by all of those interested in the health and safety of athletes," according to a joint statement by the Committee on the Medical Aspects of Sports of the American Medical Association and the National Federation of State High School Athletic Associations.

"Too often, in an attempt to achieve the publicized whiplike action, the aspiring athlete turns to a pole lighter than that specified for his weight," the statement said.

"Each fiber glass pole is rated by the manufacturer through control tests on flexural, tensile, and compressive strength for a definite weight limit. Manufacturers warn that the poles must not be overloaded as much as five pounds or breakage is apt to occur."

Most of today's 16-foot vaulters use a pole which is specified for a vaulter weighing five pounds more to get the pole strength they need for the kick to get over the top, the statement continued. These vaulters know how to put extra bend in the pole, it said, but the average high school vaulter doesn't have this skill nor is he going to a height that requires the whip action.

"Therefore, there is no excuse whatsoever . . . for the coach to allow his vaulters to use a pole lighter than that specified for their weight," the statement concluded. "Moreover, the athlete must share this responsibility by respecting the need for the fundamentals of a progressive training program rather than seeking glamorous unsound shortcuts to competitive success."

The two groups also recommended conscientious care of the fiber glass pole to avoid breakage.

INHERITANCE In Essential Hypertension

RESEARCH & INVESTIGATION

ESSENTIAL HYPERTENSION is assuming an ever greater role in the health of the nation, but in our search for its causes we must first consider the host, and recognize heredity as an important etiologic factor. Earlier investigations indicate that hypertension is conveyed by a dominant gene.[1, 5, 10, 12, 18] There is wide divergence in the figures in these reports. However, Allan's come close to fulfilling the requirements for dominance.[1] Pickering,[9] and Hamilton and associates,[4] believe that hypertension is inherited as a graded characteristic and that the inheritance is multifactorial. The writer supports this view herein, and also deals with the influence of a positive family history of hypertension on the blood pressure in the offspring, as well as the influence of hypertensive sibs on other members of the sibship.

Method

This analysis avoids the use of a clinic or hospital population, and represents a three-year survey of 319 persons in all walks of life. There is no selection except that all subjects are from a medium-sized city, over 20 years of age, and their parents must have lived long enough so that the anamnesis is of value. Hypertension caused by a known exogenous factor is not included. However, this is difficult and one or two instances may have been overlooked. The subjects are the propositi, or the persons with whom the genetic investigation begins. An investigation of their pedigrees includes an anamnesis as to the presence of hypertension, stroke, and related diseases in their first-degree relatives (father, mother, brother, sister, child), parental sibs, and grandparents. When a history of hypertension or stroke in a relative is obtained, further information is secured by another series of questions to verify the diagnosis. In nearly all instances the diagnosis was made by a physician. Brothers and sisters are grouped together,[12] since there is only a difference of 1 or 2 mm. of mercury between the sexes in any of the adult age brackets.[3] The difference is also small for age, and there is evidence that the blood pressure of normotensive adults does not rise much after age 30. The figures of the Society of Actuaries on the general population show for men a rise of about 5 mm. of mercury for the diastolic pressure between 27 and 62 years of age. Morrison and Morris[8] believe that the apparent rise in blood pressure with age is an artifact, that the blood pressure of normotensive persons rises little with age, and the apparent rise is due to the emergence of a hypertensive group in the ages past 50 years. The same thing is observed in our own controls. For these reasons, differences in age do not introduce much error. However the ages of the propositi are fairly evenly distributed; from 20 to 39 years, 29 per cent; 40 to 59 years, 38 per cent and 60 to 79 years, 33 per cent. When subgroups are compared as in the tables, a check reveals that the error attributable to age differences is not over 1.5 mm. of mercury in the blood pressure averages.

The dividing line between hypertension and normal blood pressure is taken as 150 mm. of mercury systolic and 95 mm. of mercury diastolic. A stroke is taken as evidence of hypertension.[12, 15] A previous check on stroke alone revealed a blood pressure increment similar to hypertension. Whenever possible, blood pressures of the relatives are checked also. The term hypotension refers

A three-year study of 319 patients indicates that essential hypertension can be inherited, on a multifactorial basis, as a graded characteristic.

S. K. ROBINSON, M.D.

to blood pressure that is below the figure of the Society of Actuaries for that age and sex.[3] The blood pressure averages and the distribution of hypotension in the groups are used also for comparison, since if the blood pressure is inherited as a graded characteristic, it is inherited not only as hypertension, but in the range of normal blood pressure as well. This is best brought out in the average blood pressure and in the distribution of hypotension.

The blood pressure is taken in a sitting position, five readings on each subject, one every three minutes. An average of these is used in the analysis. Whether one such series of blood pressure readings is sufficiently reliable in a study of this kind was tested by rechecking many propositi a second time, several weeks later. There were differences, but not enough to change the final average much more than 1 mm. of mercury. Søbye, who made a similar investigation, stated that it made little difference in the results that the blood pressure was checked only once.[12] Family size may also have a bearing on a study of this kind and will be referred to in the discussion.

Analysis

One hundred propositi with negative family histories make up the controls. In checking the pedigree for the controls the following criteria are required: both parents must have reached a fairly old age and all first-degree relatives must have been free from hypertension and stroke. Whether such persons passed insurance examinations is also of aid. The controls have an average blood pressure of 125.5 mm. of mercury systolic and 78.5 mm. of mercury diastolic, and include three instances of hypertension. The rise in the diastolic pressure with age is small, there being only a difference of 1 mm. of mercury between those above and those below 50 years of age. For the systolic pressure, the rise is about 2 mm. of mercury for each decade. The low incidence of hypertension in the controls speaks well for the accuracy of the anamnesis.

Following the method used by Thomas[14, 15] for different matings, we have in Table 1 a comparison of the prevalence of hypertension in three types of matings. In type 1, both parents are affected; in type 2, one parent; and in type 3, neither parent is affected. The descending gradation of prevalence from type 1 to type 3 shows a strong hereditary influence. Later it will be shown that the single gene hypothesis is not a suitable explanation for the inheritance of hypertension. However, it may be mentioned that in Table 1 the requirements for dominance are not fully met. Type 3 and type 2 matings come close to fulfilling the requirements for dominant inheritance, but type 1 falls considerably short. Few investigators have been able to prove dominance in all three types of mating, and our figures do little better.

TABLE 1.—*Comparison in Three Types of Mating.*

Mating	No.	Parents	Offspring: Hypertension %
Type 1	38	H+ and H+	60.52
Type 2	111	H+ and H—	47.74
Type 3	100	H— and H—	3

(H—) Hypertension negative.
(H+) Hypertension positive.

The low incidence of hypertension in type 3 mating is further highlighted by the fact that the incidence of hypertension in the

TABLE 2.—*Blood Pressure Average and Increment, and Chance of Affection in Propositi with History of Hypertension in One Relative.*

Relative with Hypertension	No.	Blood Pressure Average Syst.	Blood Pressure Average Diast.	Blood Pressure Increment Syst.	Blood Pressure Increment Diast.	Chance of Affection %
None	100	125.5	78.5			3
Sister	20	156	95	30.5	16.5	60
Brother	17	156.3	92.5	30.8	14	53
Mother	69	149.9	95.4	24.4	16.9	47.8
Father	42	149	92.3	23.5	13.8	47.6
Grandparent	17	124.2	84.2	–1.3	5.7	11.8

general population is between 12 and 40 per cent, depending on the authority.[11] Yet our low figures in type 3 mating have been confirmed.[2, 15] This, along with other evidence, supports the view that hypertension is genetically influenced; some investigators consider the disease a genotype.[2, 12, 18] The influence of environmental factors, although important, is probably limited by the hereditary background.[18]

Table 2 gives a comparison of the part the different hypertensive relatives play in conveying hypertension to the offspring. For instance, with a hypertensive father, what is the possibility for a child to develop hypertension? If we take all the propositi with hypertensive fathers and determine the percentage of hypertension in the group, it gives the answer. It may be expressed as the "chance of affection." The "chance of affection" varies with the different first-degree relatives; the sibs contribute more than the parents, and in each pair, the females are ahead of the corresponding males. In the order of importance, the "chance of affection" is for a sister 60, brother 53, mother 47.8, father 47.6, and grandparent 11.8 per cent. Søbye,[12] who introduced the term, gave the "chance of affection" for a sister as 68.5, brother 55, mother 45.6, father 43.66, aunt 26.8, and uncle 20.6 per cent.[12] He did not include grandparents.

The increment in the blood pressure for each group above that of the controls is given also in Table 2, and it bears a relation to the "chance of affection." The systolic blood pressure bears approximately a 50 per cent relation and the diastolic blood pressure a 25 per cent relation to it. There is approximate agreement in these figures for the first-degree relatives except for the mothers, where the diastolic figure is closer to one-third, but for a grandparent there is little agreement. The grandparent is a more distant relative and there is a greater chance for error. When there is more than one hypertensive relative in a family, the increment in the blood pressure is not the sum of the two or three relatives, but considerably less. To understand this factor better the propositi in Table 3 are arranged in groups depending on the number of first-degree hypertensive relatives in the family.

Table 3 reveals an ascending gradation in the average blood pressures of the propositi as the number of hypertensive first-degree relatives rises. The rise for one hypertensive relative is 24.1 systolic and 14.5 diastolic; with additional hypertensive members the increase is not in exact multiples of the first, but somewhat less, especially in grade C.

The number of hypertensive relatives in a family is an index of the intensity of the blood pressure for all members of the family. Where other family members are also examined, as was done in another study, it is observed that all the members of a family with three hypertensive relatives have, as a rule, a higher average blood pressure than those in a family with two hypertensive relatives. As the blood pressure of the propositus rises the incidence of hypertension in the family rises. This suggests that there is an ascending gradation in the hereditary conveyance of hypertension, and that the higher the blood pressure, the greater the expressivity of the genes.

Discussion

In Table 2 it is brought out that the sibs show a greater index of severity than the parents. They are of course younger than their parents and their blood pressures are lower because blood pressure is lower in young persons, particularly those under the age of 30. Then too, they are less likely to have reached the age when hypertension becomes manifest and therefore less likely to

TABLE 3.—*Blood Pressure Average and Increment, Chance of Affection, Hypotension, and Age of Propositi as Number of Hypertensive Relatives Rises.*

Grade	No.	No. Hypertensive Relatives	Blood Pressure Average Syst.	Blood Pressure Average Diast.	Blood Pressure Increment Syst.	Blood Pressure Increment Diast.	Chance of Affection %	Hypotension %	Av. Age
A	100	0	125.5	78.5			3	45	48
B	144	1	149.6	93	24.1	14.5	49.7	15.3	53.6
C	48	2	155.4	96.2	29.9	17.7	56.2	6.3	47.2
D	13	3	190.3	105.3	64.8	26.8	92.3	0	49.1
E	14	4	203.9	114.7	78.4	36.2	92.9	0	50.7

have high blood pressure. Thus a high blood pressure in the parents is less likely to indicate a severe familial disposition to high blood pressure than is the same level of blood pressure in the sibs.

As seen in Table 3, hypotension bears an inverse relation to the "chance of affection." Whereas hypotension is present in 45 per cent of the controls, it falls off sharply when one family member has hypertension, and disappears in all propositi with three or more hypertensive relatives. This decline in hypotension in grades B and C is greater than it should be, and we can obtain similar information in still another way. Starting with those propositi who have one hypertensive parent, we know that a little less than 50 per cent inherit hypertension, but what are the findings in the other half with the lower blood pressures? If we examine this lower group, we find that they show also an increase in the average diastolic pressure of 3.5 mm. of mercury over the controls. This suggests that the blood pressures of most, if not all, propositi are affected by the hypertensive parent. This is confirmed in a later study.

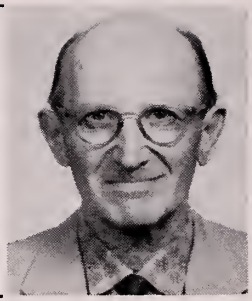
The different grades of blood pressure in Table 3 appear to be distinct groups that are fairly true to type. In each group the propositi resemble each other in the range of the blood pressure. This favors the idea that the individuals inherit a certain level of blood pressure. In the controls shown in group A of Table 3, there are only two instances of a blood pressure of the order in group E. Contrariwise in Group E there are only two instances of the lower type blood pressure seen in the controls. In some primitive peoples hypertension is rare and an average of 60 mm. of mercury diastolic has been reported for an entire tribe, which means a lower expressivity than in our controls. The reason hypertension is rare in this tribe is probably because the expressivity is of too low an order to produce it, and environmental factors are not conducive for it. It is therefore difficult to see how the conventional ideas of heredity can be applied to the blood pressure and hypertension. Our evidence favors the idea of multiple genes, and that the inheritance is therefore most likely multifactorial, as in longevity or height.[7]

Another question that arises is what effect family size has on these results. Such a study[17] was undertaken and an analysis was made separately on families with equal numbers of sibs, and all blood pressures were recorded. A similar result was obtained in all families irrespective of size, thus confirming the findings in Table 3 that there is an ascending gradation of the blood pressure as the number of hypertensive persons in the family increases.

There is also a difference between inherited and acquired hypertension which is of clinical value. The hereditary pattern is the more basic and more difficult to alter; the acquired hypertension is more easily changed. This was brought out in the work of Speransky and Beljaeva[13] who say that hypertension with a negative family history responds better to treatment. Langley and Platt[6] collected 26 instances where nephrectomy was done to relieve hypertension. In those with a positive family history there was only 1 success in 13; in those with a negative history, 8 successes in 13. The presence of a genetic factor points away from a physical cause such as renal disease, but not necessarily from pheochromocytoma, since this has also been shown to be inherited.[16] In obtaining a family history, the presence of hypertension in the sisters or brothers is just as important as is its presence in the parents.

Conclusions

In this study, the following conclusions seem warranted:



Dr. Robinson practiced in Chicago before moving to Brownsville where he specializes in internal medicine.

1. There is an ascending gradation of the blood pressure in the propositus with each additional first-degree hypertensive relative.

2. The increase in the blood pressure is 24.1 mm. of mercury systolic and 14.5 mm. of mercury diastolic for the first hypertensive relative. For the second and third hypertensive relatives the rise is less than the multiples of these figures would indicate.

3. The "chance of affection" with one first-degree hypertensive relative is given in order of importance. It is for a sister 60, brother 53, mother 47.8, father 47.6, and grandparent, 11.8 per cent.

4. The number of hypertensive relatives in a family serves as an approximate index for the height of the blood pressure in the propositus.

5. Several distinct grades of blood pressure and hypertension exist in the population.

6. The evidence favors the idea that the inheritance of the blood pressure is multifactorial.

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Down-to-Earth Excitement Greater

The heart of a man at the wheel of a sports car may beat much faster than the heart of an astronaut, a Houston experiment reported in the March 16 issue of *Modern Medicine* has shown.

Telemetry techniques transferred to a Houston speedway from Project Mercury have shown that the driver's heart often hits 200 beats per minute; no astronaut has exceeded 184 beats even during reentry, the toughest part of an orbital flight.

Studying stress and performance relationships, the National Aeronautics and Space Administration has found that the astronaut in his capsule undergoes less physical stress than sky divers, hockey players, runners, and polo players during competition.

Paraphysial Cyst of Third Ventricle

Walter Krohn, M.D., K. P. Wittstruck, M.D., and Harry W. Slade, M.D.

Two patients died suddenly following excruciating headaches associated with nausea and vomiting. One had a three-weeks' history of headaches and the other apparently had suffered no previous serious headaches or other cerebral symptoms. Paraphysial cysts of the third ventricle were found at autopsy.

CASE REPORTS

PARAPHYSIAL CYSTS are characteristically located in the anterior, superior portion of the third ventricle, just below the fornices. The cystic tumor can easily obstruct the interventricular foramina causing acute dilatation of the lateral ventricles, followed by coma and sudden death. The first case of paraphysial cyst was reported in 1858 by Wallmann.[9] Up to now 142 case reports have appeared in the literature. Most of these were coroner's cases and discovered at autopsy because of sudden, unattended, and unexplainable death. In two of our medicolegal autopsies we discovered paraphysial cysts of the third ventricle obstructing the interventricular foramina causing acute dilatation of the lateral ventricles followed by coma and death. These cases are reported and discussed.

Case Reports

Case 1.—A 12-year-old white boy was brought to the emergency room of Providence Hospital, Waco, at 8:15 a.m. At the time of arrival the boy was pronounced dead. Rigor mortis and livor mortis were fully developed and the time of death was estimated at 4 to 6 hours prior to arrival at the hospital. The history revealed an acute onset of severe and excruciating headaches the afternoon prior to death. These were associated with nausea and vomiting. The parents stated that there was no previous history of headaches or other cerebral symptoms. The boy went to bed that evening and was found the next morning by his parents without any vital signs. An autopsy was ordered by the justice of the peace.

At autopsy the brain was heavy and soft, weighing 1530 gm. The gyri were flattened with the sulci being obliterated. The leptomeninges were delicate, smooth, and clear. The blood vessels over both hemispheres were moderately congested. A pressure cone was evident at the base of the brain and at the cerebellum, secondary to increased intracranial pressure. Sectioning through the corpus callosum revealed a large cyst completely filling the third ventricle. This cyst was protruding and herniating into the lateral ventricles and obstructed the interventricular foramina of Monro. Both lateral ventricles were acutely dilated. The cystic tumor measured 2.5 cm. in maximum diameter, was thin-walled, round, and of pearl-gray color. On section the cyst contained a soft, gray, colloid type material. Microscopically, the cyst wall was composed of thin bands of fibrous tissue which were covered along the inner surface by cuboidal-type cells. The contents of the cyst consisted of a homogeneous pink-staining colloid-type material. Other autopsy findings were massive pulmonary edema (weight of lungs: 1160 gm.) and generalized passive congestion.

Case 2.—A 35-year-old white housewife was brought to the emergency room of Providence Hospital, Waco, at 10:30 p.m. in a comatose condition. Five minutes after arrival her respirations ceased, and heroic efforts to resuscitate failed. The history revealed an acute onset of severe and excruciating headaches associated with nausea and vomiting while watching television one hour before death. The patient took aspirins which failed to relieve the headaches. She became dizzy and disorientated and went into coma with convulsive-type seizures. She was rushed to the hospital and died five minutes after arrival. The past history revealed frequent and intermittent episodes of severe headaches over the past two to three weeks. The husband stated that sometimes these headaches were spontaneously relieved by abrupt and rapid change of position of the head. Aspirins did not relieve the headaches. She did not seek medical attention. An autopsy was ordered by the justice of the peace.

At postmortem examination the brain was swollen, soft, and wet with the gyri being flattened and the sulci completely obliterated. The brain weighed 1580 gm. Around the cerebellum and brain stem there was a deep pressure cone. The blood vessels over both hemispheres were massively congested. The meninges were smooth and glistening. On serial

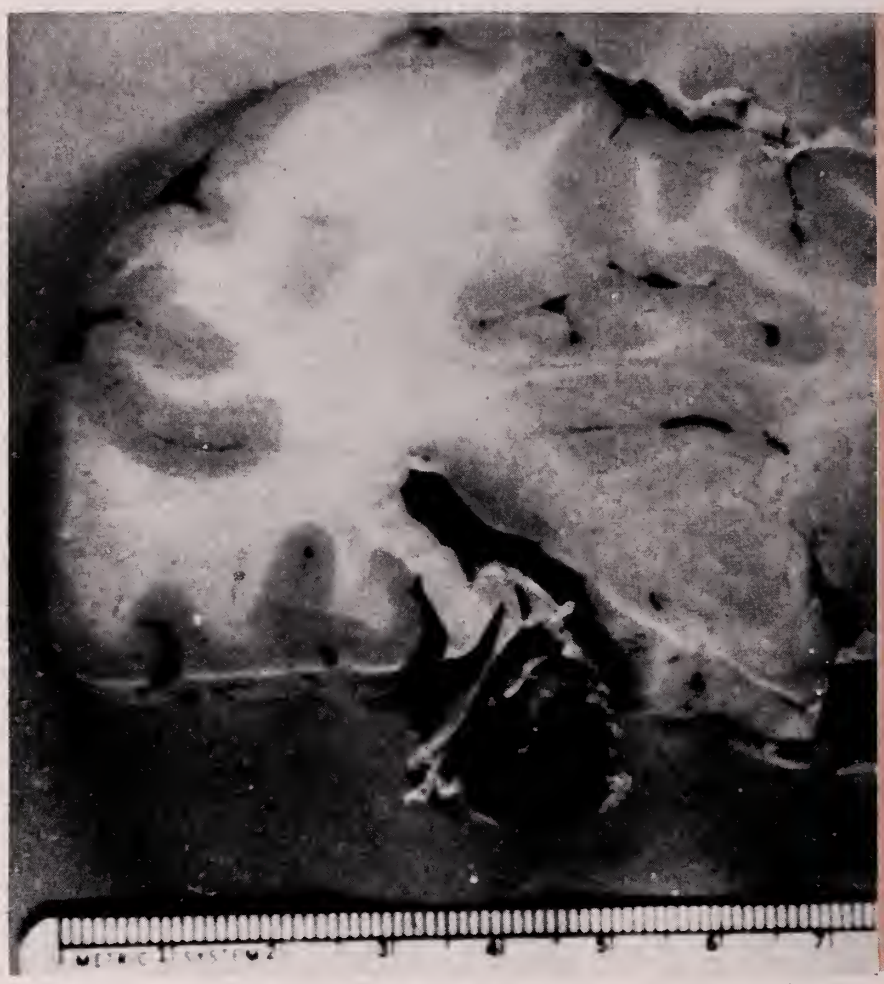

Fig. 1. Case 1. Longitudinal section of brain through corpus callosum showing impacted cyst in the third ventricle.

frontal sections both lateral ventricles were massively dilated and filled with clear, watery cerebrospinal fluid which was under pressure. The third ventricle was also dilated, but much less than the lateral ventricles. In the third ventricle there was a grayish-white, round, cystic mass, measuring 2.2 cm. in maximum diameter. This cyst was lodged in the dilated opening of the aqueduct of Sylvius which communicates with the fourth ventricle. On section this cyst was filled with clear, watery fluid. The cyst wall was thin and transparent. The inner lining was smooth, grayish-white and glistening. Microscopically, the wall of the cyst was composed of thin bands of fibrous tissue. The inner lining consisted of a single layer of cuboidal-type cells. Other postmortem findings were pulmonary edema and generalized passive congestion.

Embryology

In the past these cystic tumors were thought to arise from the ependyma or choroid plexus. It is now believed that these cysts are derived from remnants of the paraphysis. Sjoevall[6] was the first to suggest this possibility of origin. The paraphysis is a rudimentary structure found in all vertebrate embryos[3, 5] and developed to a high degree in amphibians.[2] In human embryos the paraphysis has been identified as an evagination of the telencephalon in the mid-line from the roof of the third ventricle in the region of the rostrum. This outgrowth appears at the 75th day of gestation and normally disappears thereafter. In some individuals the evagination or outgrowth of the telencephalon apparently persists and slowly enlarges, probably due to retention of material secreted from the lining epithelial cells.[8] The exact function of the paraphysis is not known, although it appears to be secretory in nature.[1]

Symptomatology

In our two cases, severe and excruciating headaches associated with nausea and vomiting were the only significant symptoms. In all 142 reported cases in the literature

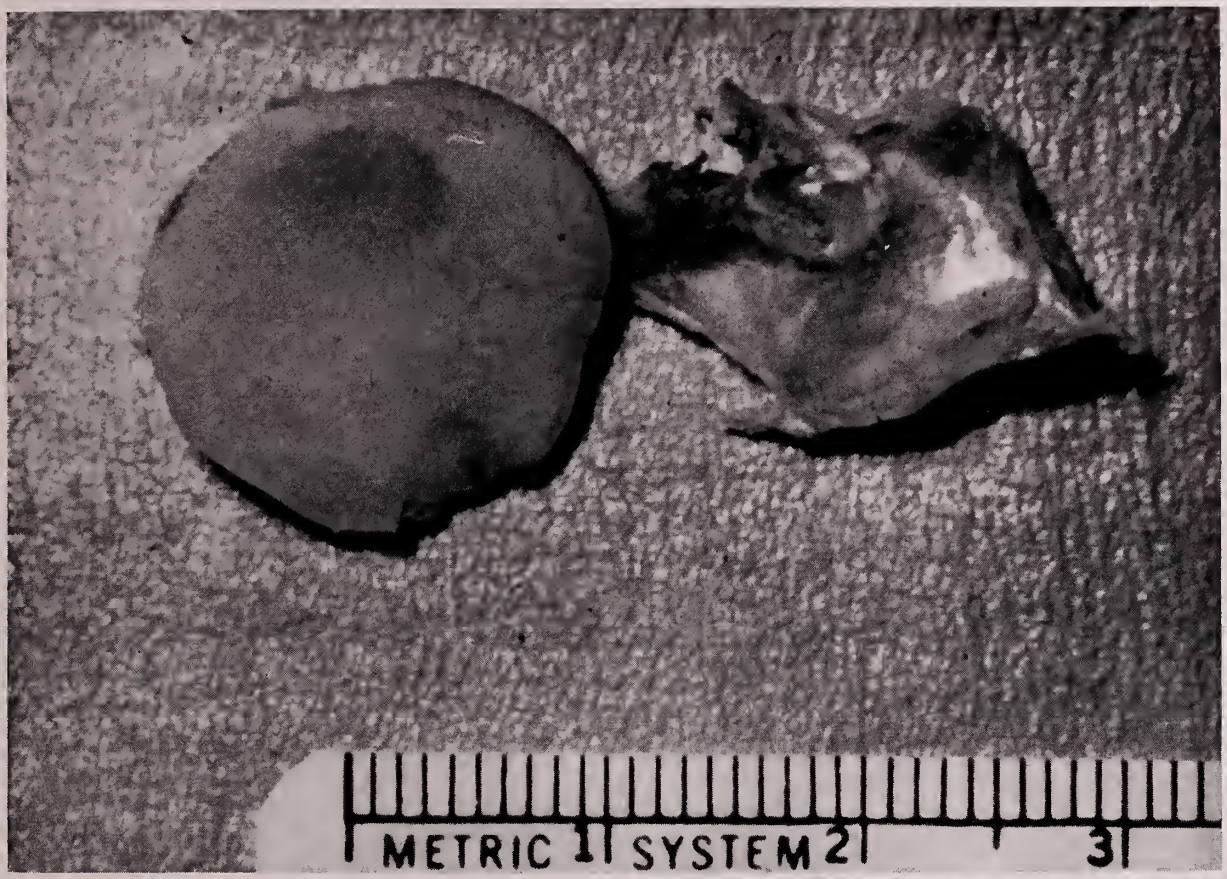

Fig. 2 (above). Case 2. Cross-section of the cyst with coagulated protein contents and thin fibrous wall.

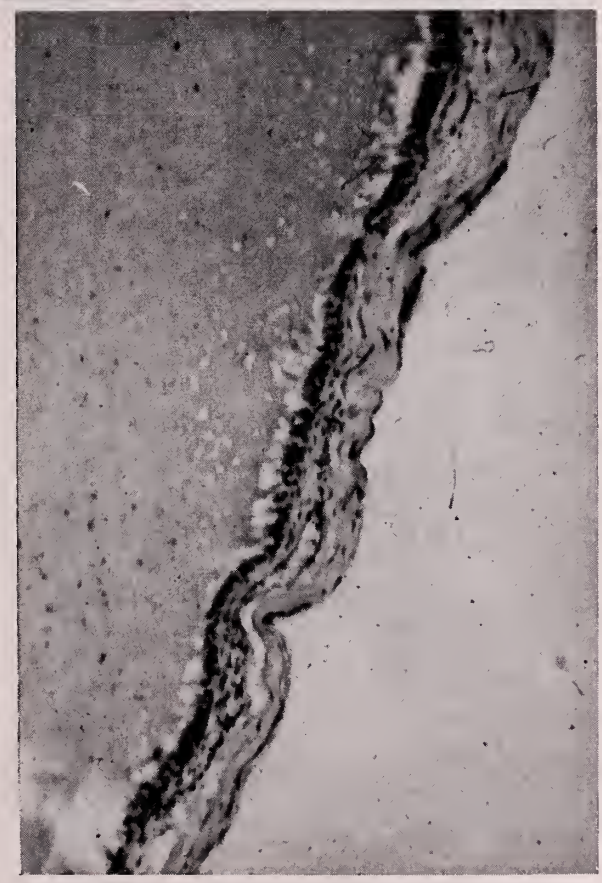

Fig. 3 (right). Microsection of cyst wall (X 200) showing the thin fibrous wall with a low columnar epithelial lining.

headaches associated with nausea and vomiting were the most striking and consistent features. These were either intermittent in character over a period of months or brief and terminal. Neurologic findings were not constant, and varied except for a significant incidence of papilledema in those who had the benefit of medical study during life.[7]

Pathology

The paraphysial cyst of the third ventricle is a smooth, thin-walled, pearl-gray, cystic structure of varying size. Sometimes the choroid plexus of the third ventricle is adherent to or stretched over the surface of the cyst.[4] Microscopically the outer layer of the cyst wall is composed of thin bands of fibrous tissue which may be vascular. The inner layer is lined in most cases by cuboidal-type cells and occasionally by columnar cells with rare goblet cells. The contents of the

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cyst vary from a colloid-type material to a clear watery fluid.

Summary

Reported are two cases of paraphysial cysts of the third ventricle which came to medicolegal autopsy because of sudden, unattended, and unexplainable death. The pertinent autopsy findings are presented and the embryology, symptomatology, and pathology are discussed.

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*Their paper was presented originally to the V. International Congress of Clinical Pathology in Mexico City in October, 1963.

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Don't Blame the Weather

Weather apparently has no significant relationship to murder, or for that matter, to suicide, says Veterans Administration psychiatrist Dr. Alex D. Pokorny of Houston.

With the cooperation of the Houston Police Department, Dr. Pokorny supervised the abstraction of all cases of suicide and suicide attempts during 1960.

Each of the 91 suicides and 400 suicide attempts was reviewed to establish the time of the act.

Weather information, obtained from the U. S. Weather Bureau Station at the Houston Airport, was made available on punched cards, one for each hour; 8,784 cards for the (leap) year. Eleven weather variables were studied: temperature, windspeed, wind direction, barometric pressure, relative humidity, visibility, ceiling height, rain, fog, thunderstorms, and cloudiness.

At the completion of an exhaustive study, no single significant relationship was found between suicide and weather conditions.

The same procedure of charting and correlating homicides and weather conditions led to a similar conclusion—"No significant relationship was found."

The Cardiac Athlete

Recent Studies in Sports Medicine

ALBERT SALISBURY HYMAN, M.D.

With careful observation and training, many young people with heart disorders may participate in athletic activities, and some may even achieve high levels of performance.

REVIEW ARTICLES

THE CARDIAC ATHLETE is a relatively new conceptual entity in sports medicine.[6] The traditional belief that children and young adults with disabilities of the cardiovascular system are unlikely candidates for athletics has been changed by recent studies in compensatory physiologic reactions of the heart and blood vessel system following improvement in general physical fitness. The former classification of functional capacity based upon structural pathology has been supplanted by a new concept concerning intrinsic physical fitness of the cardiovascular system.[9] Emphasis is now placed upon actual work performance rather than *a priori* reasoning that valvular heart disease, for example, automatically precludes a boy from engaging in competitive athletics.

It has long been recognized by cardiologists that certain patients with advanced types of heart disease are able to perform amazing tasks of physical endurance while other individuals with minor or only with suspicious cardiac disabilities are unable to carry on simple activities without showing signs of dyspnea, tachycardia, palpitation, and heart pain. As an illustration it may be pointed out that mitral valvular insufficiency is the most common type of cardiac disability seen in school children and college students; individuals with identical objective physical findings may exhibit a wide range of physical fitness. Many top-level athletes have such valvular heart lesions. It is, therefore, no longer acceptable to reject candidates for school and college athletics on the simple diagnosis of mitral valvular heart disease. On the other hand, and this is important to remember, irreversible damage may develop in certain cardiac patients who have a low level of functional capacity when forced to engage in physical activities for which they are not adequately prepared.[4]

Classification of Patients

The basic problem in every case involving cardiac disability is the determination of functional capacity or physical fitness of the cardiovascular system;[8] this applies both to the asymptomatic group who may be totally unaware of any cardiac involvement as well as to those who have a long history of heart disease in childhood or in men who have experienced a previous coronary episode. As a rule, athletic team physicians will have limited contact with the latter group except in relation to professional athletes where the age period of contestants may be as high as 55 years. Our attention will therefore be directed primarily to individuals 14 to 25 years old.

The determination of physical fitness of the cardiovascular system has been greatly simplified since the introduction of the Multidiscipline Exercise Tolerance Tests developed by the Cardiovascular Research Committee of the American College of Sports Medicine, aided by grants from the Daitz Research Fund of New York.[7] In these tests based upon work performance on the Har-

From the Cardiovascular Research Committee, American College of Sports Medicine.

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HOW TO ESTIMATE FUNCTIONAL CAPACITY OF THE CARDIOVASCULAR SYSTEM*

MULTIDISCIPLINE POST-EXERCISE TOLERANCE CLASSIFICATION**

Measurement Technique	GRADE 1 Superior Physical Fitness[a]	GRADE 2 Normal Functional Capacity	GRADE 3 Fair Functional Capacity	GRADE 4 Poor Functional Capacity	GRADE 5 Hazardous Functional Capacity
PULSE RATE	No change	+5%	+10%	+25%	+50%
BLOOD PRESSURE	No change	+10%	+20%	+30%	+60%
VITAL CAPACITY	No change or better	−5%	−10%	−25%	−50%
BREATH HOLDING	−10%	−20%	−30%	−50%	−75%
MAXIMUM EXPIRATORY PRESSURE	No change or better	−5%	−10%	−25%	−50%
MAXIMUM BREATHING CAPACITY	No change or better	−5%	−10%	−20%	−35%
ELECTROCARDIOGRAM	No change	No change	Questionable changes	Definite changes	Marked changes
BALLISTOCARDIOGRAM	No change	No change	No change	Definite changes	Marked changes
VIBROCARDIOGRAM	No change	No change	Questionable changes	Definite changes	Marked changes
PHONOCARDIOGRAM	No change	No change	Questionable changes	Definite changes	Marked changes
ARTERIOGRAM	No change or DCI drop[b]	No change or DCI drop	No change	No change or PA[c]	No change or PA
HEART SIZE (X-RAY)	No change or smaller	No change	+5%	+15%	+30%
DYNAMIC RESPIRATORY INDEX	−5%	−10%	−15%	−25%	−50%
CARDIO-PULMONARY INDEX	−5%	−10%	−15%	+15%	−60%

(a) Top level athletes (b) Dicrotic Index (c) Pulsus Alternans
*Cardiovascular Research Committee, American College of Sports Medicine
**Based upon the 5,000-foot-pounds-of-work test and correlated with observed physical fitness.

Fig. 1.*

vard-Ash steps, a calculated amount of physical work, usually in the 5,000 foot-pounds-of-work range, is utilized in recording the results obtained in a selected number of physiologic parameters. In the order of relative importance and simplicity these parameters are (1) pulse rate, (2) blood pressure, (3) vital capacity, (4) breath holding, (5) maximum expiratory pressure, and (6) maximum breathing capacity. The instrumental parameters include (7) electrocardiogram, (8) ballistocardiogram, (9) vibrocardiogram or apex cardiogram, (10) phonocardiogram, (11) arteriogram, (12) rheogram, and (13) roentgenogram of heart size. Finally, certain objective formulas like (14) the Dynamic Respiratory Index, and (15) the Cardio-Pulmonary Index are included. All of these parameters may be determined (1) after a suitable resting period, (2) immediately after the tests, and (3) after a five-minute resting period.

Not all of these parameters may be necessary in the examination of cardiac candidates for athletic activities; good to excellent results in the first half of the list (parameters 1 to 6 plus 14 and 15) will be sufficient to classify a given individual. On the other hand, in doubtful and poorly-defined cardiac disabilities, all of the parameters may be essential in establishing an objective physical fitness diagnosis.[2]

Fig. 1, a guide to estimating the functional capacity of the cardiovascular system, is based on more than 4,500 cardiovascular-pulmonary studies. Five grades of cardiovascular physical fitness or functional capacity are defined:

Grade 1 includes the data from high-level athletes representing a state of superior or better than normal performance while grade 2 is the average results found in normal (non-cardiac) subjects. Grade 3 represents the data from individuals with only fair functional capacity while grade 4 includes the group with poor functional capacity. Grade 5 includes patients with such limited capacity that dyspnea, anginal pain, or congestive failure appears even on less than the calculated 5,000 foot-pounds-of-work performance.

A word should be said here about conditioning exercises in subjects with cardiovas-

*From Hyman, A. S.: Clinical Estimation of Functional Capacity in Heart Disease, Consultant 5:28 (Aug.) 1962.

cular disabilities and the estimation of serial or repeated functional capacity tests. Experience has shown that nearly every individual in grades 2 to 4 has definitely increased his functional capacity; sometimes the improvement has been as much as 60 per cent based upon the Cardio-Pulmonary Index. Both isometric and isotonic exercises may be employed in cardiac athletic candidates; grade 5 patients will benefit from the isometric routine.

It has already been stated that valvular heart disease is the most common cardiovascular disability seen in high school and college students. Mitral valvular insufficiency leads the list, followed by mitral stenosis and insufficiency; aortic valvular defects have been seen less frequently in the past decade compared to 20 and 30 years ago. Suppression of rheumatic infections and associated streptococcal control by the antibiotics and other therapeutic measures have greatly decreased the incidence of this type of valvular pathology. Serious congenital anomalies are becoming rare as they benefit from surgical intervention in early childhood; the team physician may, however, discover a number of asymptomatic congenital lesions like atrial septal defects, coarctation of the aorta, and an occasional dextrocardia. Such suspected cases should be referred to cardiac clinics equipped to perform catheterization and angiographic procedures.

Murmurs

Most of the valvular lesions are accompanied by murmurs;[11] murmurs are by far the chief reason for labeling a given individual as a cardiac suspect. The apical systolic murmur has been a clinical challenge for the past 75 years; among school children it may occur in rates as high as 40 per cent of the entire group. Our studies have shown that more than half of these can be classified within the so-called "functional systolic murmur" group.

What is a functional systolic murmur? According to the American College of Sports Medicine's Cardiovascular Research Committee, such murmurs are usually solitary findings with no evidence of clinical changes ordinarily associated with pathologic involvement of the mitral valve. Loudness or intensity of the murmur, its duration in the cardiac cycle, its distribution or radiation, and its changes during breath holding or after exercise may offer no accurate determination concerning its etiology. Open heart surgery has long demonstrated that as such, the auditory criteria of any murmur may have no relation to the actual structural change present.[5]

Tachycardia

Tachycardia is the next most common cardiac disability seen in school and college candidates for athletic activities. A fast pulse rate is not uncommon in top level athletes *before* a given competitive sport; rates as high as 160 have been noted but the usual range is within the 110 to 120 beats per minute. At rest, *after* the activity the rates ordinarily return to the 52 to 68 range. Directly after the exercise and during the final stages of the activity rates as high as 180 to 200 have been recorded by telemetric monitoring. Sinus tachycardia rarely exceeds the 210 level but paroxysmal atrial fibrillators may exhibit rates as high as 240. Persistent tachycardia with rates of 110 to 138 at rest require careful cardiologic study; a number of etiologic factors may be involved here, the chief of which is thyroid overactivity. Psychosomatic tachycardia is rarely persistent and must be differentiated from certain types of autonomic nervous system syndromes.

Atrial Flutter

Paroxysmal atrial flutter and/or fibrillation[10] are not uncommon in well-conditioned athletes both at rest and after strenuous exercise. Pure atrial flutter may not be recognized when functional A-V block develops; under such circumstances the atrial rate may be 300 beats per minute while the ventricular and radial pulse rates are only 75 as when a 4:1 ratio occurs with all of the aspects of a normal sinus rhythm. Instrumental examination will quickly reveal the diagnosis when it is suspected from rapid unusual pulsations seen in the jugular area of the neck. Paroxysmal atrial fibrillation, on the other hand, is easily diagnosed by the grossly irregular heart beat and pulse rate. In athletes this rate may be very high, as in the 200 to 240 range, but certain individuals may exhibit a relatively slow rate in the 60 to 80 range.

Episodes of paroxysmal atrial flutter and/or fibrillation usually last only for a short period, ordinarily from a few min-

utes to a half-hour. Longer attacks require a reevaluation of the cardiovascular status. During the interval of flutter or fibrillation, ventricular output falls and physical performance declines. Athletes recognize the condition by their inability to obtain their usual maximum response rather than by perceiving any change in the activity of the heart itself. Under conditions of good to excellent physical fitness, congestive failure rarely occurs, but acute pulmonary edema has been found in contestants with less functional capacity.

The most frequent disturbance of rhythm seen in athletic candidates are premature beats; these are ordinarily ventricular in origin and may occur from 5 to 20 times per minute. Many causes are given for these extrasystoles—gastrointestinal dysfunction, psychosomatic problems, and certain drugs like caffeine in coffee or the cola drinks may play a part. I doubt that smoking is a factor in this group although it is well recognized that smoking lowers vital capacity. Premature beats may be unnoticed by certain individuals even when they occur very frequently; on the other hand, many athletes suffer from various degrees of heart consciousness when they develop. Palpitation is a common complaint in the extrasystolic arrhythmias, sometimes with painful sensations over the entire precordium.

Premature Beats

Premature beats which lead to paroxysmal ventricular tachycardia have considerable clinical significance; whereas atrial paroxysmal tachycardia is more or less limited by the AV nodal mechanisms, the ventricular tachycardias have no such physiologic control and rates as high as 240 to 280 may occur with the rapid onset of congestive failure. In certain instances, cardiac arrest has developed secondary to ventricular fibrillation. Prompt cardiac resuscitation measures are required at this time; restoration of the cardiac cycle is the rule. These are the hearts which are said to be "too good to die" since the cause of the arrest is a simple dysfunction of the electrical system of the heart.

Blood Pressure

Elevated blood pressure levels are not an uncommon finding in the routine physical examination given to all athletic candidates.[3] Systolic levels are most commonly involved; in school children levels as high as 128 to 158 have been found while in college students levels up to 220 are known. When are systolic levels considered to be clinically elevated in these asymptomatic groups? Our experience with the Police Athletic League of New York Golden Gloves contestants suggests that up to the age of 14, normal systolic levels should be within the 105 to 120 mm. of mercury range; from 15 to 21 years the range is 108 to 128. Perhaps 134 mm. of mercury should be adopted as the upper limits of normal blood pressure; certainly, *constant* systolic levels above 140 must be considered as abnormal. The qualifying word, *constant,* must be emphasized here; many well-qualified athletes may on occasion exhibit higher systolic levels secondary to certain psychosomatic or emotional factors, as for example, just before a given contest. When at rest, however, these high levels promptly return to a normal figure.

Systolic levels which remain elevated after strenuous competitive sports have been under close observation by the Cardiovascular Research Committee; after heavy and fatiguing athletic events as in some track and field events, a number of well-conditioned athletes have shown systolic levels as high as 200 to 240 mm. of mercury. These high levels usually return to normal within the oxygen debt recovery period which may require 15 to 20 minutes. Under certain conditions there may be a considerable delay in the return to the pre-exercise levels; if this delay reaches an hour or longer, a number of important implications must be considered, the chief of which may be related to subclinical metabolic syndromes.

Elevated diastolic blood pressure levels have far more prognostic significance. School children may exhibit rather low diastolic blood pressure levels in the 30 to 48 mm. of mercury range; college students average 54 to 72. Diastolic levels above 88 at rest should be evaluated; in many instances subclinical or clinical evidence of kidney dysfunction may be discovered. Appropriate laboratory and other renal function tests are indicated. After exercise, diastolic pressure may remain unchanged or become slightly elevated; this is in contrast to the usual systolic rise. In a typical college student resting blood pressure was 116/78; after strenuous athletic activity the levels became 188/82. If diastolic pressure rises in any unusual de-

gree after exercise, metabolic dysfunction secondary to anaerobic muscular work must be suspected.

Hypertensive Athletes

Should hypertensive candidates engage in athletic activities? This problem is still under intensive study; certain available statistics indicate that hypertensive athletes appear to have a better longevity curve than their opposite numbers who have maintained a sedentary type of existence. These figures are offset, however, by the recognized physiologic data that anaerobic muscle metabolism throws a far greater burden on renal function than when no oxygen debt occurs. The implication here is that hypertensive individuals should take part only in those athletic activities which produce a minimum of per minute anaerobic ratios; golf, bowling, and those sports which have resting periods may be permitted.

Heart Pain

Heart pain is a relatively infrequent finding among athletic candidates but the symptom carries significant implications. Heart pain may occur at the end of a very strenuous physical event; it is probably due to myocardial ischemia secondary to the high level of oxygen debt which develops. Anaerobic muscle metabolism may contribute a number of degraded products which alter normal myocardial physiology; the catecholamine groups are known to interfere with the systolic factors of functional coronary insufficiency and may represent a stage in the development of the anginal syndrome. When such pain occurs, most athletes stop all activity; occasionally, the "second wind phenomenon"[12] develops with reduction or complete subsidence of the painful sensations. Death is rare in these instances but again prudence and good judgment should be the guide in permitting such athletes to continue in the strenuous events.

Dr. Hyman, associate clinical professor of medicine (emeritus) at New York Medical College, presented this paper at the April 23, 1964, meeting of the Texas Society of Athletic Team Physicians.

Drugs

Finally, the use of drugs in *cardiac athletes* must be considered. *Cardiac athletes* is emphasized here to differentiate this group from all other athletes. I believe that all authorities in sports medicine are definitely opposed to the employment of drugs in amateur athletics; the use of the amphetamines, Coramine, Wyamine, and a number of other parasympathomimetic preparations have no place in honest athletic activities.[1] A drugged athlete, like a drugged laboratory animal, is not a normal individual, notwithstanding a possible increase in physical performance. The question of using certain drugs in cardiac athletes is based upon an entirely different concept which involves likely beneficial treatment of the disabilities resulting from heart disease. Patients with cardiovascular syndromes receive medication to restore as much as possible their normal functional capacity. Such a cardiac individual should not lose the right to receive medication if he decides to engage in athletic activities. Many of the disabilities discussed previously may be considerably ameliorated by selected drugs under the administration of a cardiologist or competent physician.

Athletes, for example, who are subject to paroxysmal atrial flutter and/or fibrillation should receive the quinidine preparations in sufficient dosage to suppress the episodes. The persistent sinus tachycardias caused by thyroid effects will benefit from any one of the various iodine combinations. Premature beats may be suppressed by the reserpine group. The early hypertensive candidates may find some relief from the hydrochlorothiazide drugs. In short, the use of such therapeutically indicated drugs may change the entire physical performance of a given cardiac athlete.

Conclusion

The purpose of this article is to open the door to a greater measure of athletic enjoyment to many who were formerly believed to be doomed to a life of sedentary activities. The individual with cardiac disability may reach a high level of functional capacity under conditions of careful observation and training; physical fitness programs can be

tailored to serve as a welcome and healthful outlet for those generally considered to be poor athletic material. The proof of this concept is readily at hand; the number of known cardiac athletes with high-level performance continues to increase with each passing year. It should be our duty as well as our pleasure to see that the young victim of heart disease shares an equal spot in our athletic programs with those who have been more fortunate. This is one of the chief goals of sports medicine.

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Snake Fright Sometimes as Bad as Snakebite

Fear of snakes will keep many a would-be camper at home this summer, even though statistics show that in the U. S. more people are killed or seriously injured in their bathtubs than by snakebites.

An unreasoning fear or ignorance may cause more harm than an actual snakebite. For example, a man who once trod on a rattler still wakes up screaming in the night. And a hunter, spiked by barbed wire thought he had been bitten by a snake and very nearly died of shock.

True, these are extremes. But many regard the snake as a vicious and cunning brute with a deep-seated hatred of man. This is a mistake. Instead, it is timid and retiring. The earth-bound reptile will react to man as it would to any big and threatening creature, but it much prefers to slide unobtrusively away.

Should one encounter a rattler, the safest thing to do, usually, is to remain perfectly still or edge gingerly away. Do not run for this will only excite the snake, making it more apt to strike in self-defense. On the other hand, do not assume that it will shake its rattles as a signal of attack or that it only strikes when coiled. A rattling tail indicates nervousness rather than a sign of aggression. And, a rattler may strike in any direction from any position.

Effect of Usual Triparanol Dosages On Adrenocortical Function

RALPH V. FORD, M.D.

RESEARCH & INVESTIGATION

THE PROBLEM of stress in various clinical disorders is frequently seen and it is of importance especially in cardiovascular diseases. Usually, as a therapist, the physician will resort to tranquilizing drugs.

A new approach, however, has been suggested by the observation of the effect of triparanol as an inhibitor of adrenocortical activity—a possible mediator of stress.

Triparanol was first observed to have a hypocholesterolemic action due to inhibition of cholesterol synthesis predominantly in the liver[2, 3, 6] but which could also occur in the adrenal and the epidermis.[7 9] Toxicity of this drug is mostly limited to nausea, alopecia,[1] and dermatitis.[9] A recent report[7] using 1,000 mg. of triparanol daily indicated a severe inhibition of adrenocortical steroid excretion, but in an earlier report,[5] the use of 250 mg. of triparanol daily did not produce any significant change in adrenocortical steroid excretion following a period of six weeks of drug administration. The current report is concerned with the further evaluation of the long-term effect of triparanol, in the usual clinical (hypocholesterolemic) dosage (250 mg. daily), on adrenocortical steroid excretion.

Material and Methods

The subjects of this study were 30 patients who were divided into two groups. Group 1 consisted of ten patients who had urinary 17-ketosteroid and 17-ketogenic steroid determinations before and after stimulation of the adrenal gland with Corticotrophin Zinc as described previously,[5] as well as serum cholesterol determinations in the control state and following six months or more therapy with triparanol, 250 mg. daily. The urinary steroid excretion values are based on a single determination. Group 1 was further divided into five patients (1-A) with a diagnosis of "early functional hyperadrenocorticism" in addition to arteriosclerotic heart disease and five patients (1-B) with arteriosclerotic heart disease, alone. Group 2 consisted of 20 patients with arteriosclerotic heart disease who had serum cholesterol determinations prior to and following six months or more of therapy with triparanol, 250 mg. daily. In this group, urinary steroid determinations (before and after stimulation of the adrenal gland with Corticotrophin Zinc) were done only following six months or more of triparanol therapy.

Results

Cholesterol Findings (Tables 1 and 2).

In group 1-A (five patients with early functional hyperadrenocorticism and arteriosclerotic heart disease), the mean serum cholesterol decreased 37 $\pm$16.9 mg. per 100 ml. ($P > 0.05$, that is, not significant). However, if patient B.C. is omitted, the mean serum cholesterol decrease is 21 $\pm$4.7 mg. per 100 ml. ($P < 0.05$). In group 1-B (five patients with arteriosclerotic heart disease, alone) the mean serum cholesterol decreased 35 $\pm$8.4 mg. per 100 ml. ($P < 0.05$). In group 2 (20 patients with arteriosclerotic heart disease), similar and slightly more significant decreases in serum cholesterol levels

In a small study, the author has found evidence that triparanol may serve as an inhibitor of adrenocortical activity.

TABLE 1.—*Effect of Triparanol on Adrenocortical Function and Serum Cholesterol.*

Group	Age	Sex	Before Triparanol Therapy 17-KS C	17-KS D	17-KGS C	17-KGS D	Cholesterol mg.%	Following Triparanol Therapy 17-KS C	17-KS D	17-KGS C	17-KGS D	Cholesterol mg.%	Duration of Therapy (mos.)
1-A Early functional hyperadrenocorticism (5 patients)													
J.A.	53	M	19	51	45	130	265	6	9	15	9	246	10
B.C.	34	F	29	11	10	70	259	13	17	24	27	156	7
R.D.	45	F	14	29	14	47	208	6	22	18	33	182	6
J.J.	41	F	5	25	10	53	226	10	27	10	48	218	9
P.F.	31	F	2	13	24	82	259	8	12	19	52	230	8
Subtotal Average	41		14	26	21	76	243	9	17	17	34	206	8
1-B Arteriosclerotic heart disease (5 patients)													
F.L.	59	F	3	23	15	86	351	14	37	20	55	292	9
M.R.	50	F	7	5	7	41	246	8	13	9	30	208	10
R.H.	45	F	5	22	11	108	258	11	9	20	33	252	8
F.D.	30	F	11	18	19	5	396	15	32	4	35	364	10
S.B.	48	F	20	24	20	33	288	16	19	12	20	250	6
Subtotal Average	46		9	18	14	55	308	13	22	13	35	273	9
Total Average	44		12	22	18	66	276	11	20	15	35	240	8.5

KEY:
17-KS (17-Ketosteroids) and 17-KGS (17-Ketogenic steroids)—Urinary excretion expressed in mg. per 24 hours.
C—Control 24-hour urinary excretion.
D—Steroid excretion following stimulation with Corticotrophin Zinc.

TABLE 2.—*Effect of Triparanol on Adrenocortical Function and Serum Cholesterol in Group 2.*

Patient	Age	Sex	Before Triparanol Therapy Cholesterol mg.%	Following Triparanol Therapy 17-KS C	17-KS D	17-KGS C	17-KGS D	Cholesterol mg.%	Duration of Therapy (mos.)
J.C.	67	F	273	6	11	23	91	200	11
D.C.	51	F	196	14	28	16	44	176	6
W.D.	54	F	273	12	23	18	35	259	8
C.D.	53	F	226	7	19	20	174	169	8
C.F.	57	F	312	23	16	26	85	252	9
D.F.	45	M	400	22	20	24	60	320	12
V.F.	55	F	280	12	28	16	48	214	11
W.H.	58	F	299	16	33	18	42	267	9
A.K.	79	M	176	9	14	20	44	126	20
G.M.	35	F	324	18	26	20	39	273	19
J.P.	57	F	280	13	16	18	32	161	9
G.S.	50	F	273	10	28	20	51	192	12
W.S.	56	F	287	18	31	18	63	233	15
J.O.	57	F	200	14	24	27	42	182	13
R.C.	53	F	287	13	20	18	41	280	12
L.C.	55	M	170	11	21	14	39	164	16
D.K.	63	M	233	17	31	26	48	218	12
E.W.	47	M	267	12	24	15	32	246	7
T.G.	50	F	310	13	19	21	54	269	11
R.H.	65	F	296	21	29	29	41	222	9
Average	55		268	14	23	20	55	221	11

KEY: See Table 1.

were observed following triparanol (Table 2). The mean decrease was 47 ±6.1 mg. per 100 ml. ($P < 0.001$). Here, of course, there was a greater number of patients.

Steroid Excretion (Tables 1 and 2).

There was no significant difference ($P > 0.05$) between the pre- or post-Corticotrophin Zinc excretion of 17-ketosteroids or 17-ketogenic steroids in any group following triparanol therapy. While the decreased excretion of 17-ketogenic steroids (following stimulation of the adrenal gland with Cor-

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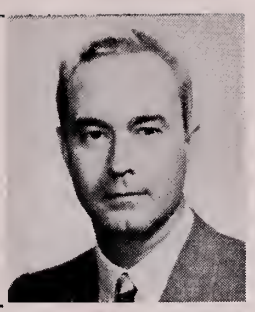

ticotrophin Zinc) in groups 1-A and 1-B appears significant on first appraisal, statistical analysis shows that the variation of the individual decreases from the mean decrease is so great as to make the result statistically insignificant. However, if patient J.A. of group 1-A is omitted, the mean decrease in 17-ketogenic steroid excretion is 23 $\pm$7.3 mg. per 24 hours ($P < 0.05$).

Discussion

Prior to the reports of Melby and others,[7] the therapeutic usefulness of triparanol was limited to the attempt to impede the atherosclerotic process in patients with hypercholesterolemia. His report indicates that triparanol, or an analogue, may be a valuable adjunct in the therapy of various states of hyperadrenocorticism. In consonance with the earlier report,[5] our current data indicate that triparanol, used in the dosage (250 mg. daily) which produces a hypocholesterolemic effect in a high percentage of patients,[4, 8] has no significant effect on adrenocortical steroid excretion. However, a trend was noted toward a reduction in 17-ketogenic steroid excretion following stimulation of the adrenal with Corticotrophin Zinc and it is possible that with a larger series of patients this trend might become significant. Therefore, in view of the fact that stress is associated with various clinical disorders and that there is a trend toward the inhibition of adrenocortical activity by triparanol, further study perhaps will indicate that triparanol would be a useful therapeutic agent in the prevention (or at least partial prevention) of the harmful effects of stress.

Summary

When triparanol was administered in a dosage (250 mg. daily) which produces a significant decrease in serum cholesterol, statistically significant decreases in urinary 17-ketogenic steroids (following Corticotrophin Zinc) were not observed. However, the possibility should be recognized that patients refractory to smaller doses but responsive to larger doses (more than 500 mg. daily) of triparanol may have a statistically significant decrease in the excretion of adrenocortical steroids following stimulation of the adrenal gland with Corticotrophin Zinc.

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Annular Pancreas in Adults

Preoperative Diagnosis

CASE REPORTS

ANNULAR PANCREAS is a congenital anomaly which exhibits constricting encirclement of the duodenum by the head of the pancreas (Fig. 1). It is probably the only congenital defect of the gastrointestinal tract that usually produces symptoms late in life.[9]

Although this entity was described as a rare medical curiosity as recently as 1944, increased awareness and improved diagnostic techniques have led to a steeply rising number of case reports. At least 271 cases have been reported in world literature, most of them in the last ten years, indicating the anomaly is more common than previously believed. Some medical centers recently reported several cases simultaneously. Widespread medical interest is indicated by the 97 articles which have appeared in the world literature, 36 of these articles since 1957; and yet, the delay in diagnosis is still disturbing.

In 23 adult cases collected from the literature between 1957 and 1962, the average duration of symptoms was 7.5 years, and the longest duration was 27 years.[10] A number of patients had multiple laparotomies, and the lesion still was not found until subsequent examination or operation. Apparently, no article concerning this subject has appeared in the *Journal*. This case report presents a summary of available etiologic, diagnostic, and therapeutic information. The scope of the paper is directed to the entity in adults.

Case Report

History: The 47-year-old white male was first examined on Jan. 17, 1958. He complained of vomiting and abdominal pain of 15 months duration, which had begun first with postprandial vomiting lasting five days and stopping suddenly. Prior to this attack he had been in excellent health. An upper gastrointestinal roentgenogram series done in another city was said to have been negative, and because of considerable business strain at that time the diagnosis was "nervous tension." Then monthly, he had three- to four-day episodes of vomiting and right upper quadrant pain, generally associated with increased business tension. The intervals between these episodes were completely free from symptoms. Seven months after onset he was hospitalized, and numerous laboratory studies and x-rays were negative. Twelve months after onset he experienced unusually severe vomiting immediately following each meal, and a severe, constant, right upper quadrant pain radiated into his back. With this he had afebrile jaundice and generalized pruritis for three days, and on hospitalization then, the diagnosis was "jaundice, possible hepatitis." He had been told that he had "an enlarged gallbladder without stones and a narrow place in his intestines." In the absence of a specific diagnosis, he refused exploration. All symptoms then completely disappeared except for intermittent, severe eructation during a ten-week period at the end of which he entered into a two-week episode characterized by vomiting 30 minutes after every meal. He vomited what he recognized as "old food" eaten the meal before, the night before, or even the previous day's lunch, and he lost 18 pounds. Belched gas seemed to have a fetid odor.

Physical Examination.—The patient was surprisingly well nourished and hydrated, and he appeared only slightly ill. Skin and mucous membranes suggested faint icterus. The temperature was 98.6 F.; pulse, 80; and respirations, 16. Blood pressure was 100/70 mm. of mercury. The liver, which descended to two fingerbreadths below the costal margin on deep inspiration, was not tender, and it had a smooth, sharp edge. There was no abdominal tenderness or distention, but a succussion splash was present. The remainder of the physical examination was within normal limits.

Laboratory and Roentgenogram.—The hemoglobin was 12 gm. per 100 ml. and the hematocrit, 39 volumes per cent. The white blood cell count was 13,100 with a normal differential, and the sedimentation rate was 26 mm. per hour, Wintrobe method. Urinalysis revealed a specific gravity of 1.025 and a pH of 7.5. The stool was a grayish tan and guaiac was negative.

Results of x-rays performed in the author's office were as follows: The intravenous cholangiogram showed delay in visualization of the common bile

The author reports the first case of symptomatic adult annular pancreas correctly diagnosed preoperatively in Texas and the 26th case in American literature.

MAURICE S. GROSSMAN, M.D.

duct which was dilated to 14 mm. The gallbladder visualized but exhibited noticeable delay in contraction after fatty stimulus. There were no stones.

Barium meal showed moderately dilated stomach. There was a conspicuous pressure defect on the lateral aspect of the second portion of the duodenum measuring 4 cm. in length and causing the width of this portion of the duodenum to be narrowed to 1.5 cm. By obstructing the flow of barium in the third portion of the duodenum with the fluoroscopic glove the pressure defect was found to have well-demarcated borders inferiorly as well as superiorly. This was demonstrated with difficulty after multiple positioning. Significantly, the lesion compressed but did not invade the mucosa of the duodenum. Follow-up films showed gastric retention (Figs. 2 and 3).

Diagnoses.—

1. Annular pancreas with partial duodenal obstruction.
2. Secondary recurrent edematous pancreatitis causing low-grade common bile duct obstruction.

Surgery.—The patient was admitted to Spohn Hospital on Jan. 27, 1958, and laparotomy confirmed the preoperative diagnoses.* Internal exploration of the annulus was performed through a duodenotomy followed by transverse closure of the duodenum and pylorus. Division of the annulus anteriorly was necessary to release obstructing pressure from the distal common bile duct, and gastrojejunostomy was created.

Subsequent Course.—Six weeks postoperatively, a barium meal showed the duodenoplasty and a well-functioning gastroenterostomy. Approximately two-thirds of the barium traversed the stoma, while the remaining one-third passed along the duodenal loop. The second portion of the duodenum filled out well, although it still showed a slight lateral pressure defect (Figs. 4 and 5).

The patient was not seen or heard from again for 19 months. In the meantime, he had three widely spaced attacks of mild epigastric pain following alcohol and dietary indiscretions. Then during a six-day period the patient indulged in alcohol, and he had daily attacks of more severe epigastric and right upper quadrant pains lasting about 20 minutes each. On admission to the hospital, he had an unbearably crushing epigastric pain which girdled the midabdomen and penetrated to the back. The patient was ashen gray and sweating profusely from the pain.

The elevated white blood cell count of 25,200 was accompanied by a shift to the left. Serum amylase was 6,400 Somogyi units per 100 ml. Two days later, this figure dwindled to 1,066 units, and four days later, to 160 units. Scout films of the abdomen showed calcification of the head of the pancreas.

The patient responded to nasogastric suction, meperidine hydrochloride, antispasmodics, and sedation. Stool studies revealed steatorrhea, which responded to pancreatic replacement medication. After two weeks the patient was discharged on a low fat diet, multiple small feedings, triple strength pancreatin, chlorpromazine hydrochloride, propantheline bromide, calcium lactate, potassium chloride, acetazolamide, and antacid.

The diagnosis was relapsing pancreatitis. The diet was increased gradually and medications decreased over the next month, and the patient was back to full activity in another month.

He did well until two years later, when following overwork and dietary indiscretion he had a mild attack of relapsing pancreatitis and was hospitalized for one week. Since that time, he has had no further attacks of pancreatitis.

Fig. 1. Line drawing depicting annulus (arrow) of annular pancreas constricting the descending duodenum.

*Color movies of the procedure are available from the author.

Fig. 2. Barium meal demonstrates a "cutoff" point in the descending duodenum, but fails to visualize the full annulus defect without counterpressure because the small barium stream quickly empties from the duodenal loop beyond the partial obstruction.

Fig. 3. Pressure defect of annular pancreas is demonstrated in spot film of descending duodenum by blocking escape of thin stream of barium beyond obstruction with gloved finger (see check in right lower corner). Note the large "notch" (arrows) with well-demarcated superior and inferior borders. Compression without invasion or destruction of underlying mucosa is a critical point.

Fig. 4. Barium meal six weeks postoperatively showing gastroenterostomy and duodenoplasty. Barium now flows freely through annular area (Close-up in Fig. 5).

Fig. 5. Close-up (spot film) of annular region showing much less obstructing "notch." Note persistent retraction of descending mid-duodenum toward pancreas.

More Cases Discovered

Tiedemann described the first case in 1818, but there is controversy[9] as to whether he discussed the actual lesion or theoretical possibility of such a lesion. The term "annular pancreas" was proposed by Ecker in 1862, and the condition was first treated surgically by Vidal in 1905. Lehman rendered the first preoperative diagnosis in 1942, bringing the number of known cases up to 50; however, only 13 cases had been treated surgically at that time. During the next five years, the number of cases requiring operation increased by only five. Momentum gained in the next five-year period with 35 new operation cases, and from 1953 through 1957 no less than 127 operation cases were added.[6] Thus, surgically treated cases in this recent five-year period more than doubled the number treated surgically in the previous 50 years. These statistics include infants.

Embryology and Pathology

The normal pancreas develops in the seven-week-old embryo before rotation of the gut occurs. One theory is that the annular pancreas develops because the ventral anlage of the pancreas fails to rotate with the foregut, so that it is "fixed" anteriorly, and the gut stretches a ring of pancreas around itself.[10] Usually the encirclement is complete, but occasionally there is an anterior gap. The annular pancreatic tissue drains through a duct which passes around the entire duodenum. This duct may enter the duct of Santorini or Wirsung, or it may open separately into the common bile duct or the duodenum. The annular pancreas preserves continuity with the pancreas proper.

"The gross lesion in annular pancreas is essentially one of varying degrees of obstruction of the descending duodenum,"[2] with or without associated peptic ulcers and their complications, and the annulus may produce obstruction of the common bile and pancreatic ducts.[7] Microscopically, the annulus consists of normal-appearing pancreatic tissue unless acute or chronic pancreatitis is present.

Clinical Clues to Diagnosis

The history and physical examination can accurately localize the area of difficulties, that is, the pyloric and duodenal region, and focus attention to the type of trouble, that is, high intestinal obstruction, either partial or complete.

Most surgical literature dismisses symptomatology with "ulcer-like symptoms," or "recurrent acute indigestion"; however, more detailed analysis is necessary to suspect the diagnosis. Characteristic abdominal pain is located predominantly in the epigastric region, although occasionally it may be in the right upper quadrant. Usually the pain does not radiate, but occasionally it is directed posteriorly. Most patients describe the pain as intermittent cramping whereas few complain of a burning sensation which is relieved by alkali or food. One-fourth of the cases experience postprandial fullness.

Vomiting occurs in two-thirds of the cases and characteristically relieves the pain. Generally, this is without hematemesis unless there is an associated ulcer, but old food in the vomitus is characteristic. Nocturnal aggravation of pain and vomiting is an important diagnostic clue. It is believed that a supine position causes obstruction because the duodenum is more sharply compressed by the annulus.

Weight loss is surprisingly moderate in most cases and ranges from 5 to 10 pounds. This relatively good nutritional status is maintained in spite of the severe and usually long duration of symptoms.

Symptoms of lesser frequency and importance include nausea without vomiting, belching, and anorexia. There have been a few cases of hypoglycemia, which have been attributed to hyperinsulinism in a hypertrophic pancreas.

The presenting picture may be acute pancreatitis with pronounced abdominal pain and tenderness and an elevated serum amylase. On the other hand, the condition may simulate chronic pancreatitis with steatorrhea, diarrhea, and even diabetes mellitus.[10]

Annular pancreas may give the appearance of peptic ulcer and its complications in as many as one-third of the patients,[12] and at least three known factors may be related to this. Constriction of the annulus prevents regurgitation of alkaline duodenal contents into the duodenal bulb for neutralization of the acid chyme. This acidity in the cap, as well as the dilatation of the area, maintains a reflex closure of the pylorus, leading to gastric retention. Mechanical stimulus by the retained food on the antral mucosa in-

duces increased liberation of gastrin which increases acid secretion by hormonal mechanisms. In a few instances that hypoglycemia has been noted, gastric secretion is increased by means of the vagus nerve.

In the adult group, ages have ranged from 17 to 81 years, with a mean age of 39 years. Although there is no racial predilection, annular pancreas occurs twice as often in males as in females.

Physical examination may reveal: a succussion splash due to retained fluid in the stomach, tenderness related to a peptic ulcer or pancreatitis, jaundice related to biliary tract obstruction, or dehydration and stigmata of hypochloremic alkalosis due to prolonged, recurrent vomiting.

Laboratory data is generally of little value except in cases of elevated amylase, icterus, electrolyte changes, or guaiac-positive stools. Measurement of gastric acidity is of no help.

Diagnosis

The upper gastrointestinal x-ray series is the most valuable procedure in establishing the diagnosis. Yet, there may be difficulties with this procedure, and a first set of films may be inconclusive. In such a case careful review of the patient's history can lead to suspicion of annular pancreas. In many instances this suspicion has led to a second upper gastrointestinal series which yielded the correct diagnosis.

The "double bubble" sign, which is pathognomonic in the newborn, is of no value in the adult. Obstruction is generally not complete in the adult, and x-ray examination with barium or water-soluble opaque media is almost always feasible. Poppel and Beranbaum,[8] as well as Dodd and Nafis,[1] presented good discussions of the roentgenographic criteria. Gastric dilatation may or may not be present. One of the first significant features is a reflex pyloric spasm; however, the partial obstruction is located not in the pylorus, but farther down in the descending portion of the duodenum. Proximal to this are the nonspecific changes of dilatation, frequently a giant "megabulbous" duodenal cap and reversed peristalsis.

Five points characterize the radiologic complex at the annulus level:

1. A notch-like pressure defect, 1 to 5 cm. in length, lies on the outer border of the descending duodenal loop, and this loop is retracted toward the pancreas.
2. The mucosa underlying this defect is smooth or compressed, not destroyed.
3. The duodenal lumen above the annular band bulges.
4. There are no spastic phenomena.
5. Repeated examinations show no change in the defect.

Diagnostic Errors

Erroneous clinical diagnoses of cicatrizing peptic ulcer, duodenal diverticulum, congenital duodenal band, primary neoplasm of the duodenum or pancreas, arteriomesenteric ileus, cholecystitis, and choledocholithiasis have been made frequently. Even in the past three years there have been cases incorrectly diagnosed preoperatively as carcinoma of the duodenum, peptic ulcer for 20 years, duodenal ulcer with obstruction, and infiltrating pancreatic malignancy. This latter error also occurs at surgery.

Neoplasm and postbulbar ulcer are the lesions most frequently confused with annular pancreas on x-ray examination. Postbulbar ulcer produces a smooth, round indentation on the lateral wall, causing a narrowing of the lumen similar to that seen in annular pancreas. The presence of a crater within or opposite the deformity is the best differential sign. When this crater is not present, one should look for secondary signs of diffuse irritability of the proximal duodenal segment, mucosal edema, localized derangement of the valvulae conniventes, or absence of obstruction.

Malignant disease presents destruction of the mucosa, and this is the most outstanding sign for differentiation from annular pancreas. Duodenal neoplasm reveals an eccentric filling defect, and in addition to eccentricity, pancreatic neoplasm shows external pressure defect and lateral view increase in retrogastric space. Mucosa over a benign lesion may stretch or even ulcerate, but it never shows true destruction. Lipoma produces a smooth, round or oval defect, while aberrant pancreatic tissue produces a circumscribed and irregular, contrast defect similar to a vacuole. A broad-base polyp may be distinguished by peristalsis through the involved area.

Contrary to the infant situation, congenital lesions such as diaphragms, aberrant ves-

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sels, and atresia give no difficulty in the adult patient. Cholecysto-duodenocolic bands involve the bulb area, proximal to the annulus, and in the right anterior oblique position, the bands disappear. This maneuver accentuates the findings from annular pancreas.

In the early literature, a number of cases were erroneously diagnosed as large, duodenal diverticula because of duodenal dilitation. Recently this diagnosis has been ruled out, however, by demonstrating the pedicle of the diverticulum with contrast material. Primary chronic or tuberculus duodenitis can be distinguished by widespread irregular changes and stricture of the duodenum, but inflammatory adhesions may cause a picture difficult to differentiate from annular pancreas.

Management

Annular pancreas can be treated only with surgery, and recent surgical reviews agree that some type of bypass procedure should be performed.[4, 10] Direct attack upon the annulus may produce complications such as duodenal stenosis, pancreatitis, abscess, fistula, or pseudocyst. Hence, this previously recommended procedure is not advised. In the absence of complications a simple duodenojejunostomy posterior to the transverse colon carries negligible morbidity and mortality and usually produces complete and permanent relief from symptoms. This simple procedure advantageously places the bypass just proximal to the obstruction. Thus the operation precludes the possibility of postoperative dumping syndrome, avoids the danger of marginal ulceration accompanying simple gastroenterostomy, decompresses the dilated duodenum, and relieves reflex pyloric closure.

When serious peptic ulcer is a complicating factor, gastroenterostomy with partial gastrectomy and/or vagotomy is recommended. If the common bile duct is obstructed, common duct exploration and T-tube choledochostomy with cholecystectomy should be performed. If pancreatitis exists, gastric resection may be advisable.

Medical management of pancreatitis is important, and patients should be advised against drinking alcoholic beverages.

Discussion

In 1957 Drey[2] published a complete world literature review of symptomatic annular pancreas in adults; this was the most extensive and current report available at that time. Of 62 adult surgical cases known then, Drey found that a correct preoperative diagnosis had been made in only ten American cases and in two cases from outside the United States. However, he credited it a correct diagnosis if it was mentioned anywhere in the differential. Simultaneous 1957 reports included Mast, Telle, and Turek,[7] four cases; Whelan and Hamilton,[12] one case, and Friedman,[3] one case; Regan and Wren[10] brought the review of literature up to 1960, adding two American cases. Between 1960 and 1963 only Seigman and Thorp,[11] four cases, and Huebner and Reed,[4] one case, correctly diagnosed the condition prior to surgery. Thus the case presented here is the 26th American adult symptomatic case in the literature with a correct preoperative diagnosis, and it seems to be the first Texas case report to fit into this category.

In infants, either acute annular pancreas obstruction occurs or else the defect does not become symptomatic until decades later.[4] Newborn cases are associated frequently with other anomalies, and since they present an entirely different diagnostic and therapeutic problem, they are not discussed in this paper. In a few cases symptoms may be minimal or absent throughout life and are found accidentally at autopsy or surgery.

Why a constricting lesion such as this may be present from birth, escape symptoms for 40 years or more, and then become severe enough to demand surgery is interesting and peculiar. It has been speculated that aging produces an increasing ptosis of the stomach and duodenum which forms an angle at the fixed ring. This gradually increasing angulation is thought to cause gradually progressive obstruction which may or may not be accelerated by acid-ulcer mechanisms. Recurrent mild attacks of pancreatitis may be

induced by slight obstruction of the annulus duct or they may be secondary to ulcer inflammation. Either way, the gradually swelling pancreas behaves like a slowly tightening noose around the neck of the duodenum.

Summary

Annular pancreas is a unique obstructive congenital anomaly which may not present symptoms until late adult life. The condition is unusually prone to preoperative and even surgical diagnostic and management error. Absence of a diagnosis in this case when three previous x-rays of the upper gastrointestinal tract were made during symptoms illustrates the difficulties in recognition. Rapidly increasing numbers of reports indicate that the condition is much more common than previously suspected. Clinical familiarity and improved radiographic techniques are apparent keynotes to identification, and preoperative diagnosis is the touchstone to proper surgical management.

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Laboratory Animals Essential

The introduction of a new drug and indeed the continued use of some established drugs would be impossible without the use of experimental laboratory animals. Only when a drug has passed stringent initial tests on animals for toxicity, clinical safety, and usefulness can it pass into the hands of the clinical investigator for use in man. These tests constitute a vital chain in the long process from the time a new drug is prepared until it finds its way into the hands of the physician for use in the alleviation of human disease, writes Dr. Arthur Grollman in *Southern Medical Bulletin,* December, 1963.

Cavernous Hemangioma Involving Segment Jejunum and Mesentery

C. D. Bussey, M.D., William S. Reynolds, M.D., and George J. Race, M.D.

The authors present a case of a patient with a hemangioma involving a segment of jejunum and its mesentery which is considered unusual since there was only solitary involvement of the jejunal wall and massive involvement of the corresponding jejunal mesentery.

CASE REPORTS

HEMANGIOMA occurs in all tissue of the body. In 1960 Rissier[1] reviewed the world literature on the subject of hemangioma of the intestinal tract which at that time contained 114 cases, and he recorded two additional cases. Of these 116 cases, only 36 involved the small intestine alone and of the 36, only 11 patients had single lesions whereas 25 patients had multiple lesions. The most common types were cavernous or capillary, or mixed capillary and cavernous.

In the patients reported, no age was exempt. The most common symptom was that of hemorrhage from the gastrointestinal tract. Other symptoms were far less frequent and usually related to intestinal obstruction produced by a space-occupying hemangioma itself or secondary to hemangioma acting as an intussusceptum.[2]

The following report of a patient with a hemangioma involving a segment of jejunum and its mesentery is considered unusual in that there was only solitary involvement of the jejunal wall and there was massive involvement of the corresponding jejunal mesentery. The authors have not been able to find a case in the literature which includes massive jejunal mesentery involvement.

Case Report

A 39-year-old man was seen by one of the authors on Jan. 16, 1963, with complaints of weakness, headache, shortness of breath, and palpitation of the heart. These symptoms had gradually increased in severity over a three-month period.

A review of systems was negative except as related to the symptoms mentioned. Specifically, there were no abdominal symptoms and no knowledge of gross melena.

Physical examination revealed obvious anemia. There were no visible hemangioma nor birthmarks. The lungs were clear. The heart was normal except for a rate of 92. The abdominal findings were entirely normal and no masses were palpable. The rectal examination was negative except for the dark brown fecal matter on the examining finger. Guaiac test was strongly positive for blood.

Laboratory examination showed the following values: hemoglobin, 6.3 gm.; hematocrit, 23 per cent; red cell count, 2,500,000; white cell count 7,400; bleeding time, one minute, and coagulation time, 13 minutes. The blood smear showed hypochromatic microcytic anemia. Proctoscopic examination was normal. Gastrointestinal roentgenograms of the stomach, small bowel, and colon were normal. No phleboliths were noted in the scout films of the abdomen. Stool specimens were positive (four plus) for blood.

The patient was given two 500 cc. transfusions of packed red blood cells and one 500 cc. transfusion of whole blood.

A working diagnosis of chronic small bowel hemorrhage, probably from Meckel's diverticulum, was made and an exploration was advised.

On Feb. 14, 1963, the abdomen was explored by one of the authors through a right mid-rectus incision. There was a cavernous hemangioma involving a segment of distal jejunum and its mesentery. A segment of jejunum approximately 30 cm. in length, including the entire hemangioma was excised (Fig. 1). An end-to-end jejunojejunostomy was done. Recovery was uneventful. One month after surgery the hematocrit was 42 per cent and the stool specimen contained no blood.

Pathologic examination of the surgical specimen revealed a 24 cm. segment of small bowel and its mesentery (Fig. 1). The mesentery was an irregular, poorly delineated, highly vascular structure measuring up to nine cm. from the bowel wall, and followed the bowel for a 14 cm. segment. The bowel wall as well as the pattern of the mucosa was essentially normal grossly, except for large submucosal vascular channels projecting into the lumen of the bowel on its mesenteric border (Fig. 2).

Histologic section showed a portion of the mesentery consisting of fat. It contained numerous dilated blood vascular channels. The vascular chan-

Fig. 1. Gross photograph of operative specimen, hemangioma segment jejunum and its mesentery.

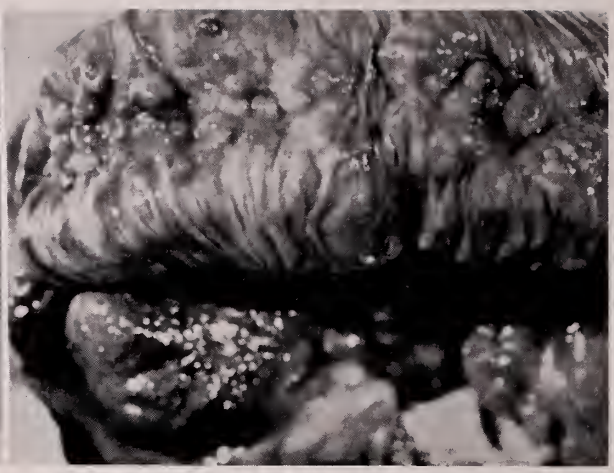

Fig. 2. Gross photograph of jejunum showing projection of large submucosal vascular channels into the lumen.

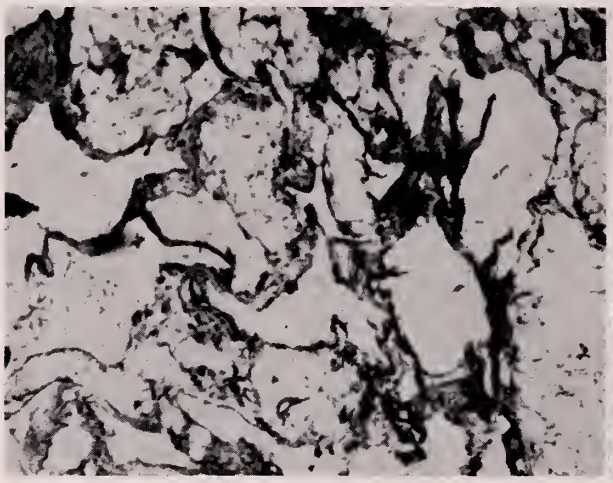

Fig. 3. Photomicrograph of the mesentery showing vascular channels lined by endothelial cells.

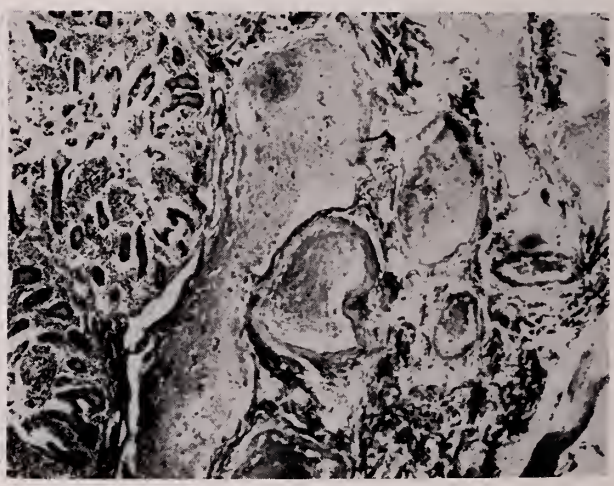

Fig. 4. Photomicrograph of jejunal wall showing vascular channels involving muscularis and submucosa.

nels were both large and small. They were lined by endothelial cells (Fig. 3).

Histologic sections of the small bowel showed numerous vascular channels which involved the muscularis and the submucosa and extended into the mucosa focally (Fig. 4).

Diagnosis.—Resected small intestine and mesentery; cavernous hemangioma, massive, involving the submucosa and muscular wall of the small bowel and its mesentery.

Summary

A case is reported of a patient who had a massive cavernous hemangioma of a segment of jejunum and its mesentery successfully removed surgically.

Chronic melena with severe secondary anemia and related symptoms were the only clinical and laboratory findings.

The report is unusual in that a single segment of small bowel (jejunum) was involved and there was massive involvement of the corresponding jejunal mesentery.

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Dr. C. D. Bussey, the senior author, is a Dallas surgeon. Dr. William Reynolds is a Dallas internist and Dr. George J. Race is a pathologist there.

Inadvertent Gastroileostomy

R. E. MAURER, M.D.

This patient was the victim of a serious surgical accident. Fortunately a second operation corrected the mishap of the first, and the patient regained his health.

CASE REPORTS

INCIDENCE OF gastroileostomy associated with partial gastric resection is fortunately far less than that with the short-circuit procedure without resection. The consequences are less serious if the error is made in attempting a simple gastroenterostomy than if the gastrectomized stump is sutured to the ileum, thus deactivating the entire small bowel with all its enzymatic actions. As long as the pylorus remains patent, a sufficient amount of gastric content will continue to pass into the small intestine to nourish the individual, but with a partial gastrectomy, the food passes through the gastroileostomy stoma directly into the colon and results in severe malnutrition.

A review of the literature reveals seven cases of gastroileostomy associated with partial stomach resection. Moretz summarizes 25 cases of inadvertent gastroileostomy and finds that 22 of them involved patients on whom a simple gastroenterostomy was being done for peptic ulcer. In only three instances was this condition associated with partial gastrectomy, one of them being a case in which malrotation of the midgut was largely responsible for the error.[3] More recently, Marshall and O'Donnel have reported seven cases of gastroileostomy, four of which occurred during partial gastric resection and three during the attempted performance of a gastroenterostomy.[2] McKenzie and Robertson report a case of an 81-year-old man who had had a simple gastroileostomy 36 years before his death and in whom the error was an incidental autopsy finding.[1] Obviously a faulty anastomosis of the stomach to the ileum is compatible with comparatively good health as long as the stomach has been left intact and the pylorus is patent. Such is not the case, when the gastrectomized stomach is sutured to the ileum. The case reported herewith is believed to be the eighth reported instance of gastroileostomy associated with partial gastric resection.

Case Report

A 50-year-old white male farmer was hospitalized on Nov. 27, 1961. He complained of diarrhea, great loss of weight, edema, and fecal belching of six months' duration. On May 6, 1961, he had elsewhere undergone partial gastrectomy for duodenal ulcer. On the third postoperative day, diarrhea had developed and had never ceased. The stools were light tan in color, frothy, contained undigested food particles, and had a foul odor. The number of defecations varied anywhere from eight to ten per day and usually occurred 15 minutes after eating. In spite of the fact that he had a voracious appetite and consumed large quantities of food, his weight had decreased from 230 to 154 lbs. in the six months following his surgery. He complained of a constant fecal taste and had been forced to resort to chewing tobacco to relieve it. He felt bloated much of the time and occasionally had gas pains, but he did not have epigastric distress which he had prior to his surgery in May. He felt weak and as a result had not been able to work since his discharge from the hospital. He had become progressively weaker and was bedridden for part of the three months prior to his admission to the hospital. During this time he had begun to notice edema, first of both ankles and feet, and then of the upper extremities and face. He thought perhaps he had kidney disease and had consulted a physician who assured him that his kidneys were in good order.

Examination revealed an emaciated white male who looked older than 50 years. He had pitting edema in the lower and upper extremities. The face also appeared swollen and the abdomen was visibly distended. About an inch above the umbilicus a transverse scar 5½ in. long was noted. He had a blood pressure of 90/60 and a pulse of 100. The lingual papillae were atrophied and some red-

ness of the tongue was observed.

Fluoroscopy and roentgen examination (Fig. 1) after a barium meal revealed a gastrectomized stomach. The size of the gastric stump could not be determined as the barium emptied rapidly into the colon with little remaining in the stump itself. Roentgen plates taken five minutes after ingestion of the meal showed it to be in the ascending and transverse colon as far as the splenic flexure. A loop of small bowel approximately 18 in. long was seen connecting the gastric stump to the cecum.

Laboratory tests revealed the following: Blood count and urine were normal; total protein was 5.6 gm. per 100 ml.; albumin, 2.4 gm. per 100 ml.; and globulin, 3.2 gm. per 100 ml.; and A/G ratio, 2:3. Mazzini test was negative.

Bleeding time was 2 minutes; clotting time, 3 minutes. Gastric contents examined by the Ewald method revealed free hydrochloric acid of 6 degrees and a total acidity of 27 degrees. Electrocardiogram was normal. Chest x-ray was normal.

The patient was prepared for surgery. He was given 75 gm. of Albumisol (Merck Sharp & Dohme) intravenously, with frequent feedings of a diet high in calories, carbohydrates and proteins. He also received daily injections of vitamin B complex and vitamin C.

Fig. 1. Immediate plate after ingestion of the barium meal. The stomach is seen to contain a small amount of barium. The greater portion of the barium has passed through the stomal orifice into the terminal ileum, filling the ascending and transverse colon. Some of the barium has also entered the afferent loop.

At operation three days after admission, it was found that the large gastric stump had been anastomosed to the ileum 18 in. from the ileocecal valve. There was no evidence of a gastroileal ulcer. Many adhesions were encountered, mainly in the region of the stoma and distal ileum. The liver appeared slightly enlarged and the small bowel showed extensive edema. No other significant findings were noted. After lysis of adhesions the transverse colon was delivered and the jejunum identified at the ligament of Treitz. After mobilizing the smaller and greater curvatures, the stomach was transected at a higher level leaving approximately one third. The gastric remnant was anastomosed to the jejunum approximately 10 cm. from ligament of Treitz, placing the efferent loop at the greater curvature. This anastomosis was made anterior to the transverse colon. The resected segment of stomach still attached to the ileum was then excised at the anastomotic site, and transverse closure of the ileum accomplished. The abdomen was closed without drainage.

Postoperatively, the patient had an uneventful convalescence. Parenteral feedings were given daily. The stomach was kept empty by gastric suction tube, which was removed on the third postoperative day. Water and liquids in small quantities were started by mouth. The patient tolerated this well, and by the fourth day he was taking soft foods. The edema had subsided to a great extent by the time he left the hospital on the ninth postoperative day. He actually was discharged two days earlier but elected to stay. The fecal taste was gone and he remarked that he felt much better than he had

Fig. 2. Four weeks postoperatively. Five minutes after ingestion of the barium meal, the gastric stump has filled out well and the barium is passing through the stomal orifice into the efferent loop of the upper jejunum.

Dr. Maurer is a Littlefield surgeon.

for a long time. His stools were beginning to become formed and were dark. He no longer was plagued with diarrhea. Four weeks later he was seen in the clinic. He had gained 15 pounds, ate well, and was gaining strength.

The pathologic diagnosis read: Segment of stomach and ileum, removed for correction of gastro-ileo anastomosis following subtotal gastric resection.

Comment

In an attempt to determine the cause of this surgical error, the surgeon who performed the original operation was contacted and hospital records were reviewed. Several factors contributed appreciably to this mishap:

1. Poor anesthesia resulted in poor relaxation. The patient vomited and aspirated during anesthesia.

2. The incision was poorly placed. It was transverse, placed too low at about an inch above the umbilicus, and was 5½ in. long. This incision could not possibly have given adequate exposure for a proper subtotal gastric resection. At the second operation an upper left rectus incision approximately 7 in. long was made. This gave excellent exposure.

3. The original operation lasted nine hours. The patient went into shock, and in an attempt to terminate the procedure, the surgeon proceeded too rapidly.

4. The surgeon was inadequately assisted.

Summary

A case of inadvertent gastroileostomy following partial gastric resection with all its dire consequences is presented. The patient lost 76 lbs. in a period of six months and exhibited the syndrome of severe malnutrition. Corrective surgery, which consisted of lysis of the faulty anastomosis, with higher resection of the stomach and proper anastomosis of the jejunum to the gastric remnant brought dramatic relief to the patient. By the end of four weeks all previous symptoms had subsided and the patient had gained 15 pounds.

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Dr. Maurer, 500 LFD Drive, Littlefield.

Role of Potato Chip in Staple Diet

Those who absentmindedly eat potato chips from the bag have been known to ingest staples as well.

Dr. Robert L. Ruskin reports in the *New England Journal of Medicine*, April 16, that he spotted a staple in the gastrointestinal tract of a patient during a routine x-ray examination of the abdomen.

A visit to the patient's room established where the staple came from: lying on the bedside table was an opened bag of potato chips.

Texas Snakebite Statistics

RESEARCH & INVESTIGATION

MORE PEOPLE ARE bitten by poisonous snakes every year in Texas than in any other state in the United States. Furthermore, Texas led the nation in the number of snakebite deaths during the ten-year period, 1950-1959.[7] Approximately 43 per cent of all poisonous snakebites occurring west of the Mississippi River happened in Texas. These statements refer to numbers of snakebites and numbers of deaths. A more meaningful measurement is the annual rate of bites in a state, which takes into account the population at risk to snakebites. The leading annual poisonous snakebite rates per 100,000 population for the states west of the Mississippi River are found in Arkansas (17.19), Texas (14.70), Louisiana (10.25), and Oklahoma (8.85). They comprise all the member states of the West South Central Region of the United States.

Most of the early studies for evaluating snakebite treatment were done in Texas. Amaral[1] and Hutchison[2] of the Antivenin Institute of America evaluated the use of antivenin in treating poisonous snakebites from 1926 to 1928. Jackson of San Antonio was the first to demonstrate experimentally the value of incision and suction for treating rattlesnake bites.[3]

Reliable statistics about the incidence of poisonous snakebites in Texas have not been published. Therefore, it seemed worthwhile to make a statewide study for the following purposes: (1) to define the epidemiology of poisonous snakebites in Texas; (2) to relate some medical findings associated with these bites; and (3) to review briefly current concepts of snakebite treatment.

Poisonous Snakes

Werler[10] authored an excellent booklet about poisonous snakes of Texas that was published by the Texas Fish and Game Commission, Austin. According to Werler[10] there are 16 species and subspecies of poisonous snakes indigenous to Texas including ten kinds of rattlesnakes, four kinds of copperheads, one kind of cottonmouth moccasin, and one kind of coral snake. See Fig. 1 for photographs of poisonous snakes of Texas.

He lists the following species and subspecies of poisonous snakes: the western diamondback rattlesnake *(Crotalus atrox)*, the timber rattlesnake *(Crotalus horridus horridus)*, the canebrake rattlesnake, *(Crotalus horridus atricaudatus)*, the prairie rattlesnake *(Crotalus viridis viridis)*, the banded rock rattlesnake *(Crotalus lepidus lepidus)*, the mottled rock rattlesnake *(Crotalus lepidus klauberi)*, the blacktailed rattlesnake *(Crotalus molossus molossus)*, the Mojave rattlesnake *(Crotalus scutulatus scutulatus)*, the western pigmy rattlesnake *(Sistrurus miliarius streckeri)*, and the western massasauga *(Sistrurus catenatus tergeminus)*.

Also, the southern copperhead *(Agkistrodon contortrix contortrix)*, the northern copperhead *(Agkistrodon contortrix mokeson)*, the broadbanded copperhead *(Agkistrodon contortrix laticinctus)*, the Trans-Pecos copperhead *(Agkistrodon contortrix pictigaster)*, the western cottonmouth moccasin *(Agkistrodon piscivorus leucostoma)*, and the Texas coral snake *(Micrurus fulvius tenere)*.

Texas has each of the four major kinds of venomous snakes found in the United States. With the exception of the coral snake, they are all pit vipers. They are so named because of a characteristic pit that is located between the eye and nostril on each side of the body. Pit vipers also are identified by elliptical pupils and by two well-developed fangs that protrude from the maxillae when the snake's mouth is opened. Rattlesnakes have rattles that are attached to their tails. Copperheads, cottonmouth moccasins and harmless snakes do not have rattles. Harmless snakes do not have facial pits, they have round rather than elliptical pupils, and while they have teeth, they lack fangs.

Oftentimes people will chop off the head

Prior to this study* there were no reliable statistics about the snakebite problem in Texas. Readers may be surprised to learn how effective snakebite treatment is. State case fatality rate is 0.25 per cent.

HENRY M. PARRISH, M.D., Dr.P.H.

of a snake that has bitten someone and bring the snake's body in for identification. Pit vipers can be identified by turning the snake's belly upwards and noting a single row of subcaudal plates just below the anal plate. Harmless snakes have a double row of subcaudal plates. Fig. 2 depicts the characteristic features of pit vipers and harmless snakes.

The coral snake is a beautifully colored small snake that has broad rings of scarlet and black separated by narrow rings of yellow. An easy way to remember this is "red next to yellow will kill a fellow." It is important to remember that the coral snake's *snout is always black.* Several harmless snakes resemble coral snakes, but the yellow and red rings on their bodies are separated by black rings. Coral snakes have round pupils, and they lack facial pits.

*From the Department of Community Health and Medical Practice, School of Medicine, University of Missouri, Columbia, Mo., this investigation was supported in part by Public Health Service Research Grant GM 11268-02 from the Division of General Medical Sciences, Public Health Service.

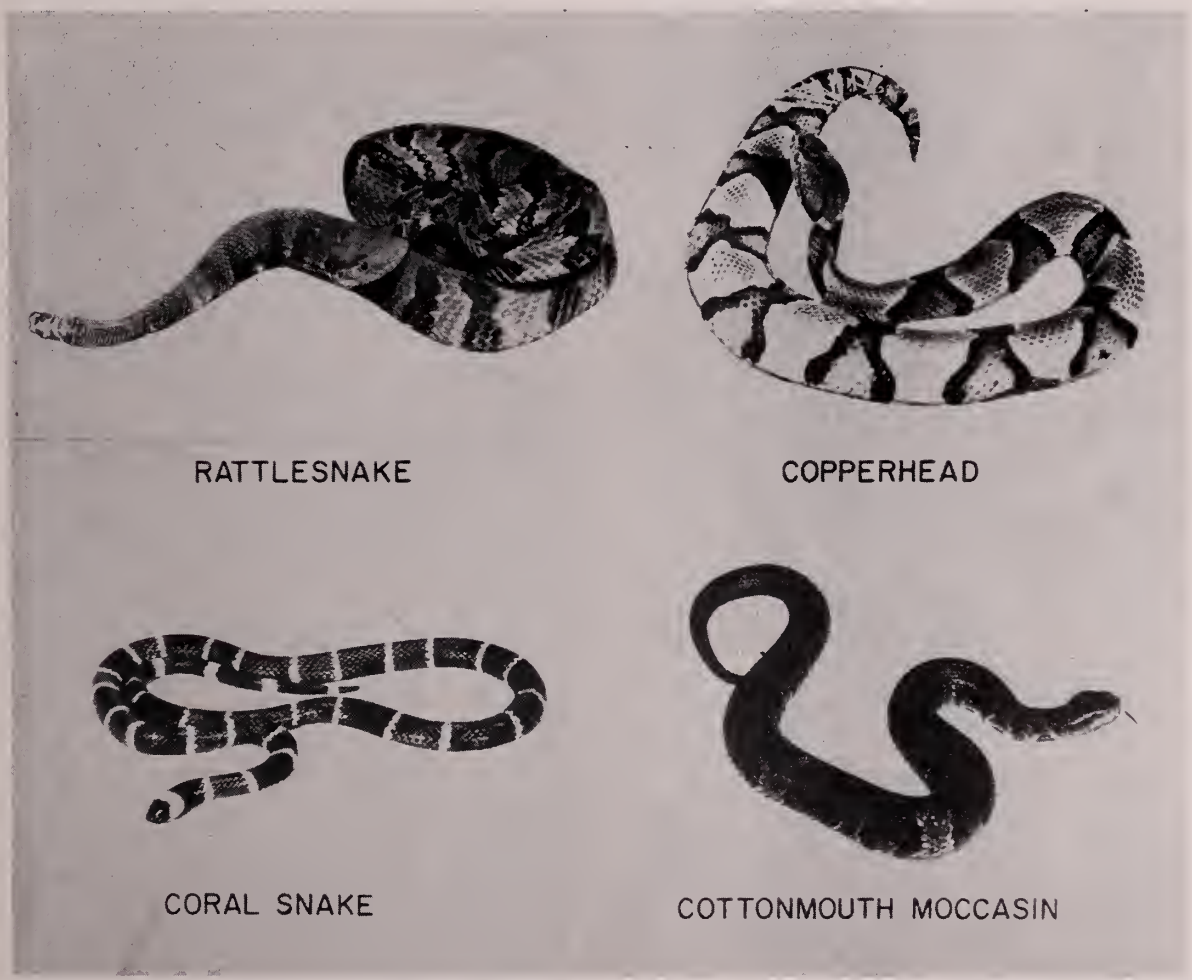

Fig. 1.

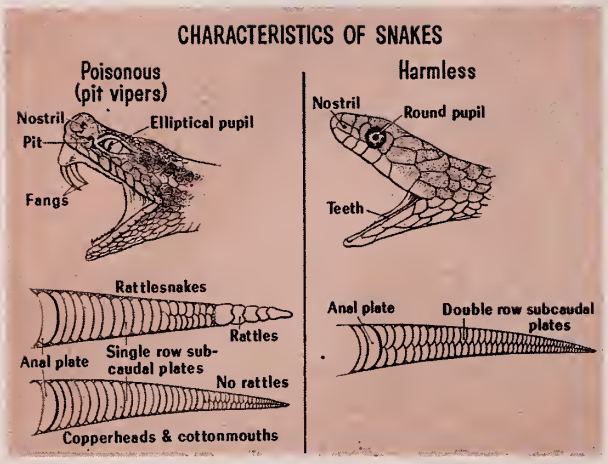

Fig. 2.

Method of Study

A questionnaire and letter explaining the purpose of this study were mailed to a "selected" group of Texas hospitals listed in *Hospitals Journal of the American Hospital Association* guide issue. The hospitals selected for this study were general hospitals, children's hospitals, and college infirmaries. Army, Navy, Coast Guard, Public Health Service, Air Force and Veterans Administration hospitals also were sent questionnaires. Maternity, tuberculosis, and mental hospitals were omitted as they would not be expected to treat snakebite victims. A total of 519 Texas hospitals comprise the study group. Each hospital was requested to report all in-patients admitted to the hospital for snakebite treatment during 1958 and 1959.

Most hospitals do not code and tabulate the diagnoses of emergency room and out-patient clinic visits. Since some snakebite victims are not admitted to the hospital as in-patients, it seemed essential to ask a sample of practicing physicians how many snakebite victims they treated on both an out-patient (office, home, emergency room, and other) and on an in-patient basis. Previous surveys,[6, 9] have shown that most people with venomous snakebites are treated by general practitioners, surgeons, internists, pediatricians, and orthopedic surgeons. Therefore, a random sample of one-third of all the Texas physicians in these categories of practice who were listed in the "American Medical Directory," published by the American Medical Association, were sent questionnaires.

Death certificates for fatal snakebite cases were obtained from the Texas State Department of Health.

Results

This report is based on questionnaires returned by 475 (92 per cent) of 519 Texas hospitals. It is supplemented by questionnaires returned by 1,644 (74 per cent) of 2,218 practicing physicians in the State. The Texas State Department of Health indicated that there was one snakebite death during 1958 and two during 1959.

Incidence.—Texas hospitals reported a total of 559 in-patients treated for poisonous snakebites during 1958 and 1959. There were 256 cases in 1958 and 303 cases in 1959, an average of 279.5 cases per year. Of the 559 snakebites reported during 1958 and 1959, detailed case reports were received for 461 patients and only numbers of bites were reported for 98 cases. *All of the analyses in this paper, excluding the estimate of incidence, were based on the 461 detailed case reports received from hospitals.*

Physicians' reports, when adjusted to account for all Texas physicians in the practice categories mentioned, indicated that approximately 784 in-patients and 624 out-patients were treated for snakebite accidents each year. The difference between the estimate of 784 in-patients treated by physicians and the average of 280 in-patients reported by hospitals can be explained, in part by the following facts: (1) 44 Texas hospitals did not participate in the study; (2) nine counties from which physicians reported snakebites did not have hospitals listed in the *Hospitals-J.A.H.A.* guide issue; (3) there was evidence of under-reporting snakebite in-patients from 39 hospitals that participated in the study; and (4) physicians indicated that many in-patients were treated in small clinics and hospitals not listed in *Hospitals-J.A.H.A.* guide issue. Taking all of these various reports into consideration, it is estimated that approximately 1,408 (784 in-patients and 624 out-patients) people are treated annually for poisonous snakebites in Texas. This provides an incidence of 14.70 bites per 100,000 population per year.

Geopathology.—The geographical distribution of snakebites reported in Texas during 1958 and 1959 may be seen in Fig. 3. The lightly shaded counties are those from which hospitals reported in-patients treated for snakebites. An appropriate symbol is used to mark each hospitalized patient who was bitten by a specific kind of snake. The darker shaded counties are those counties from which physicians reported snakebite cases,

but from which no cases were reported by hospitals.

Of 461 people hospitalized for snakebite treatment for whom detailed records were available, 215 (47 per cent) were bitten by rattlesnakes, 104 (22 per cent) by copperheads, 33 (7 per cent) by cottonmouth moccasins, four (1 per cent) by coral snakes, and 105 (23 per cent) by unidentified poisonous snakes.

Fig. 3 shows that poisonous snakebites were reported from all sections of the state. However, they were most frequent in the eastern and central two-thirds of Texas and less frequent in the western one-third of the State. Copperhead, cottonmouth moccasin, and coral snake bites were confined to the eastern and central two-thirds of Texas, whereas rattlesnake bites had a statewide distribution. Rattlesnake bites, however, were the only snakebites reported from the western one-third of the state. Bites were especially frequent in and around Austin, Beaumont, Port Arthur, El Paso, Fort Worth, Houston, and San Antonio.

The geographical distribution of bites by various kinds of snakes are consistent with the ecological ranges for snakes described by Werler.[10] One may conclude from the distribution of snakebite cases that the following species of snakes do not inflict many bites on human beings in Texas: the Trans-Pecos copperhead, the timber rattlesnake, the banded rock rattlesnake, the mottled rock rattlesnake, the blacktailed rattlesnake and the Mojave rattlesnake.

Temporal Relationships.—The monthly distribution of snakebite accidents is shown in Table 1. Snakebites were infrequent during the colder months of the year—December, January, February and March. In general, snakes are usually inactive and/or hibernating during the colder months. Most snakebites in Texas occurred from April through November when 449 (97 per cent) of the 461 bites were inflicted. This striking seasonal distribution of bites coincides with the time that snakes are most abundant and active and with the time that people have greater exposure due to out-of-doors occupations and recreation. Similar "seasonal epidemics" of venomous snakebites have been observed in New England and South Carolina.[6, 9]

The time of day when most snakebite accidents happened was the six-hour period from 3 to 8 p.m. when 201 (44 per cent) people were bitten. The number of bites by three-hour periods of time were: 6 to 8:59 a.m., 50 bites; 9 to 11:59 a.m., 63 bites; 12 noon to 2:59 p.m., 77 bites; 3 to 5:59 pm., 111 bites; 6 to 8:59 p.m., 90 bites; and 9 to 11:59 p.m., 19 bites; 12 midnight to 2:59 a.m., five bites; and 3 to 5:59 a.m., one bite. For 45 cases the time of the bite was not recorded.

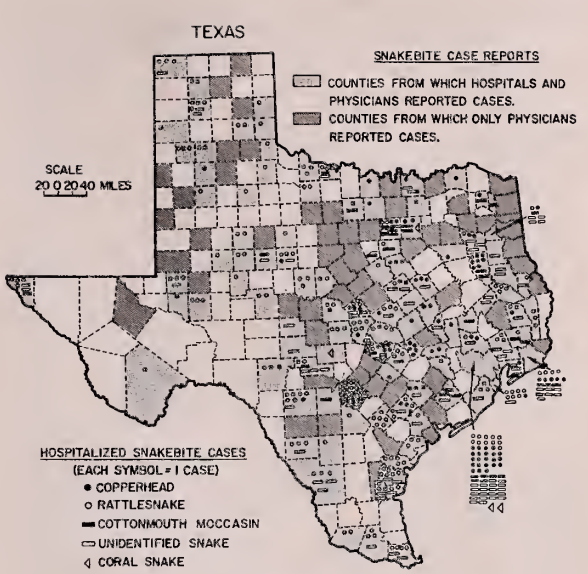

Fig. 3.

Bite Victims.—There were 285 white males, 144 white females, 22 nonwhite males and ten nonwhite females admitted to Texas hospitals for snakebite treatment during 1958 and 1959. All of the nonwhites were Negroes. Using the 1960 census for the population of Texas, the bite rates per 100,000 population were: 6.85 for white males, 3.76 for nonwhite males, 3.42 for white females, and 1.61 for nonwhite females. Thus, whites had higher snakebite rates than nonwhites and males had higher rates than females.

The age distribution of Texas bite victims is shown in Table 2. The largest number of bites happened to children and youths 10 to 19 years of age (111 bites) and those 0 to 9 years of age (103 bites). Indeed 46 per cent of all snakebites were inflicted on children

TABLE 1.—*Seasonal Distribution of Poisonous Snakebites in Texas, 1958-1959.*

Month	No. Bites	Month	No. Bites
Jan.	2	July	58
Feb.	0	Aug.	64
March	10	Sept.	77
April	38	Oct.	48
May	73	Nov.	24
June	67	Dec.	0

TABLE 2.—*Age Distribution of Hospitalized Snakebite Victims in Texas, 1958-1959.*

Age Group (years)	Population at Risk*	No. Bites	Rate per 100,000**
0- 9	2,236,530	103	4.61
10-19	1,682,133	111	6.60
20-29	1,250,280	55	4.40
30-39	1,301,793	57	4.38
40-49	1,125,524	45	4.00
50-59	906,451	52	5.74
60-69	614,372	27	4.39
70 or more	462,594	11	2.38

*Based on the 1960 census of the population of Texas.

**These rates are only on hospitalized patients for whom information was available.

and young adults less than 20 years of age. Age-specific bite rates are much more meaningful since they take into account the population at risk in a particular age group. The highest biennial bite rate per 100,000 population was: 10 to 19 years of age (6.60). The lowest bite rate was found for people 70 or more years of age.

An analysis of the occupations of the patients showed that 193 were children, 66 were housewives, 54 were farmers or farm laborers, 27 were laborers other than farm laborers, and 16 were craftsmen.

Activity and Place.—Sixty-two people were bitten while working or walking in their own yards. An additional 55 children were bitten while playing, 30 in their yards and 25 elsewhere. Forty-seven people were bitten while working on a farm or ranch, 25 while hunting or fishing, 18 while engaged in recreation other than hunting and fishing, 17 while handling a poisonous snake, ten while working around a barn or hen house, eight while working or walking on a highway, six while picking up a log or lumber and three while reaching into an obscure place. The activity was not coded for the remaining patients.

The place where the bite accident occurred is closely related to the activity at the time of the bite. The largest number of bites, 92, happened right in patients' own yards. Forty-seven people were bitten on a farm or ranch not near the house, 36 were bitten in or near a lake, river or other body of water, 16 were bitten in or under a building or house, and 14 were bitten in the woods. Ten people were bitten inside a barn or hen house, nine in a field away from the house, and eight on or near a highway. The place where the bite happened was not classified for the other patients.

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Site and Severity.—The anatomical sites on human beings where venomous snakes inflicted their bites are shown in Table 3. Ninety-six per cent of the bites were inflicted on the extremities, 36 per cent on the upper extremities and 60 per cent on the lower extremities. The feet and lower legs, including the ankles, were the parts most frequently bitten on the lower extremities. Five people were bitten on the head or face and two were bitten on the trunk of the body. The site was not recorded for 13 patients.

A modification of the clinical classification of pit viper venenation by Wood, Hoback and Green[11] was used to determine the severity of bites. Bites were classified as follows:

Grade O, No Venenation.—Fang or tooth marks, minimal pain, less than 1 in. of surrounding edema and erythema. No systemic involvement.

Grade I, Minimal Venenation.—Fang or tooth marks, severe pain, 1 to 5 in. of surrounding edema and erythema in first 12 hours after bite. No systemic involvement.

Grade II, Moderate Venenation.—Fang or tooth marks, severe pain, 6 to 12 in. of surrounding edema and erythema in first 12 hours after bite, systemic involvement may be present, nausea, vomiting, giddiness, shock, or neurotoxic symptoms.

Grade III, Severe Venenation.—Fang or tooth marks, severe pain, more than 12 in. of surrounding edema and erythema in first 12 hours after bite, systemic involvement usually present as in Grade II.

TABLE 3.—*Anatomical Sites of Bites Inflicted by Poisonous Snakes in Texas, 1958-1959.*

Anatomical Site of Bite	Side of Body		Total No. of Bites
	Right	Left	
Head, face and neck	3	2	5
Trunk, front	0	0	0
Trunk, back	1	1	2
Upper arm	1	2	3
Forearm	13	4	17
Hand	36	23	59
Fingers	53	34	87
Upper leg	5	2	7
Lower leg and ankle	87	48	135
Foot	56	53	109
Toes	17	7	24
Not stated			13

The severity of venenation (venom poisoning) was classified as follows for 409 hospitalized cases, 122 (30 per cent) were Grade O; 140 (34 per cent) were Grade I; 82 (20 per cent) were Grade II; and 65 (16 per cent) were Grade III. For the remaining 52 hospitalized cases the severity of venenation was not stated. There were three deaths in this series of 409 hospitalized cases, providing a case-fatality rate of 0.65 per cent. However, when one takes into account the 1,408 (in-patients plus out-patients) people who were bitten annually the case-fatality probably is less than 0.25 per cent. This is confirmed by the fact that there were 24 snakebite deaths in Texas during the ten-year period, 1950 to 1959, an average of 2.4 deaths per year. Of the fatalities occurring during 1958 and 1959, one was a one-year-old child bitten while playing in his yard by a large unidentified snake. The child died 12 hours later. A 30-year-old housewife, while picking berries on a farm, was bitten by a rattlesnake. She died 13 hours later. The third fatality involved a 71-year-old housewife who was bitten at home by a rattlesnake. She died two days later. Contrary to popular belief, most people do not die within the first few hours following a snakebite. About 70 per cent of them die from 6 to 48 hours after venenation takes place.[5] Rattlesnakes of the genus *Crotalus* cause more deaths than any other kinds of snakes in the United States.[5]

Although more people are bitten by poisonous snakes annually and more people died from snakebites in Texas from 1950 to 1959, the case-fatality rate from snakebites in Texas is low and compares favorably with those other states having a serious snakebite problem. This finding, it is believed, can be explained by the following facts: (1) the effectiveness of snakebite treatment; (2) the availability of prompt and proper medical care; and (3) snakebites are not as lethal as some people formerly thought, especially if they are treated properly.

Treatment

The current treatment of North American pit viper (rattlesnake, cottonmouth moccasin, and copperhead) bites includes both minor surgery and medical forms of treatment. A constricting band (tourniquet) should be applied lightly to the involved extremity several inches proximal to the bite. The constricting band should be applied only tight enough to occlude the superficial venous and lymphatic flow. *It should not occlude the arterial circulation,* and it should be released every 10 to 15 minutes for a minute or two. As edema resulting from venom poisoning spreads, the constricting band should be advanced to keep just ahead of the swelling. The purpose of the constricting band is to impede the spread of venom until incision and suction can be used to remove the venom mechanically and/or until antivenin can be administered to neutralize the venom.

Incision and suction (I.S.) is effective in removing venom from experimental animals up to about 120 minutes after the venom is injected. The sooner it is used, the larger the amount of venom that can be removed. Suction should be used for about one hour. The suction cups supplied in the Cutter and the Becton-Dickinson snakebite first-aid kits have been found effective for removing pit viper venom. Incisions, one-quarter inch long and one-eighth to one-quarter inch deep, are made into the subcutaneous tissues over the fang punctures. A few (3 to 5) additional incisions may be made in the surrounding edematous tissues. A large number of incisions are not needed. Immobilization aids in limiting the spread of venom. However, if one must decide between immobilization or seeking prompt medical treatment, the latter should be sought.

The "three A's" (antivenin, antibiotics, and tetanus antitoxin and/or toxoid) are recommended, in addition to I.S., in treating all serious pit viper bites. Crotaline Antivenin Polyvalent (Wyeth) is effective in neutralizing the venoms of all North American pit vipers. It is not protective against coral snake venom. Since antivenin is manufactured from horse serum, the patient should receive a skin test before antivenin is given. For Grade I venenations antivenin may be administered in the deltoid or gluteus muscles. In Grade II and Grade III venenations, antivenin diluted in 1000 cc. of normal saline may be given intravenously.[8] Studies with radioisotopes have shown that antivenin accumulates at the site of the bite more rapidly after intravenous administration than after intramuscular administration.[4] Injection of antivenin into the local bite area is not a particularly effective way to administer antivenin. We have found the following amounts of antivenin useful in treating the various grades of venenation:

Grade O (no venenation) requires no antivenin; Grade I (minimal venenation) may require 10 cc. (one ampoule) of antivenin; Grade II (moderate venenation) requires 30 to 40 cc. of antivenin; and Grade III (severe venenation) requires 50 cc. or more of antivenin.

Since snakes' mouths and venoms may harbor pathogenic organisms, antibiotics and tetanus antitoxin, and/or toxoid should be given prophylactically. Gram negative organisms predominate, hence a broad spectrum antibiotic is indicated. Penicillin used by itself is not adequate treatment.

Cortisone and ACTH do not affect the survival rate of animals poisoned with pit viper venom. They probably should not be used during the first few days after venenation, although they may be beneficial later in treating serum sickness resulting from antivenin therapy. Antihistamines are contraindicated as they shorten the survival time of animals poisoned with pit viper venoms. Shock resulting from venom poisoning should be treated with infusions of blood, plasma, saline solution, and vasopressor drugs. Meperidine hydrochloride and other analgesics may be given to relieve pain. Recently there have been reports of excessive tissue necrosis and amputations associated with cold therapy such as packing an extremity in ice or using ethyl chloride.[4] In the opinion of this writer, cold therapy should not be used in treating pit viper bites.

Summary

More people are bitten by poisonous snakes annually in Texas than in any other state in the United States. However, because Texas has a large population, it has only the second highest annual incidence of poisonous snakebites of the states west of the Mississippi River. An estimated 1,408 (784 in-patients and 624 out-patients) people were bitten by snakes annually, an incidence of 14.70 bites per 100,000 people. However, the estimated case fatality rate was less than one-fourth of 1 per cent.

Of 461 in-patients reported in detail by Texas hospitals during 1958 and 1959, 215 (47 per cent) were bitten by rattlesnakes, 104 (22 per cent) by copperheads, 33 (7 per cent) by cottonmouth moccasins, four (1 per cent) by coral snakes, and 105 (23 per cent) by unidentified poisonous snakes. "Seasonal epidemics" of snakebites occurred with 97 per cent of the bites inflicted from April through November.

Males had higher bite rates than females and whites had higher rates than nonwhites. Forty-six per cent of the cases were among children and young adults less than 20 years of age. Ninety-six per cent of the bites were on the extremities, 36 per cent on the upper extremities, and 60 per cent on the lower extremities. Current snakebite treatment is discussed.

A. C. Stimson, Honorary Curator of Reptiles of the Houston Museum of Natural Science, and John E. Werler, Director of the Houston Zoological Gardens, assisted and encouraged this study. Judi Pummill, Genevieve Calescibetta, and Linda Hinson gave technical assistance.

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Clinicopathologic Conference

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The patient, 84, had abdominal pain, indigestion, and vomiting intermittently for four years, and atrial fibrillation for two years. Shortly thereafter, roentgenograms showed the upper GI tract to be normal; the gallbladder was not visualized.

Clinical Summary

DR. JACK F. GLOVER: This 84-year-old Negro man was admitted to Parkland Memorial Hospital for the second and last time on March 16, 1964. He was admitted at this time with a history of lethargy, poor food and fluid intake, no bowel movements, and pain on motion of both lower extremities during the preceding week.

The first admission was in 1960, when he was observed because of abdominal pain, "indigestion," and vomiting of one day's duration. At that time his blood pressure was 138/84 mm. of mercury, and pulse was 50. The remainder of the physical examination was recorded as normal. The electrocardiogram (ECG) revealed sinus tachycardia, left axis deviation, and T wave inversion in leads II, III, and AVF with biphasic T waves in V_2 through V_6. Chest roentgenogram revealed slight cardiomegaly with slight left ventricular predominance. He was discharged without treatment.

The patient was seen approximately one month later in the emergency room with a history of abdominal cramps and vomiting for two weeks, and was referred to the medical clinic. He did not report, however, and was not seen again until March, 1962, when he came again to the emergency room because of two episodes of abdominal pain and vomiting in the preceding ten days. Again he did not keep his appointment for further investigation, but reappeared in the emergency room three weeks later complaining of dyspnea on exertion and paroxysmal nocturnal dyspnea. Physical examination revealed auricular fibrillation. The ECG showed multiple and multifocal premature ventricular contractions. He was referred to the cardiac clinic where evaluation revealed blood pressure, 140/90 mm. of mercury, pulse 80, and respirations 32. The heart beat revealed continuous arrhythmia with rate of 150. Point of maximum impulse (PMI) was in the sixth intercostal space 2 cm. left of the midclavicular line. In addition, the patient complained of vomiting regularly approximately three hours after going to bed. No mention of abdominal pain was made. The diagnosis was atrial fibrillation, etiology undetermined.

Following this, the patient was seen fairly regularly in the outpatient clinic. He was digitalized but continued to fibrillate and no other attempt at conversion was made. Roentgenographic studies revealed a normal upper gastrointestinal (GI) tract and a nonvisualized gallbladder. The patient intermittently discontinued his digitalis and was redigitalized on several occasions. In September, 1962, edema was described for the first time, and he was begun on Naqua and potassium chloride in addition to his digitalis. In October, 1962, the liver was palpable 4 cm. below the right costal margin. From that time on, edema was noted at each clinic visit. The patient was last seen in the clinic on March 3, 1964, 13 days prior to his last hospital admission. He was confused and had been eating poorly. Blood pressure was 120/65 mm. of mercury, pulse 80 and irregular, and Cheyne-Stokes respiration was present.

On admission, examination revealed an elderly, thin, Negro man who was obtunded mentally, responding only with moans to voice commands. He appeared dirty and ill-cared for. Blood pressure was 88/50 mm. of mercury, pulse 68, Cheyne-Stokes respirations, and temperature was 99 F. Bilateral arcus senilis and bilateral lens opacities were

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present. The fundi were not seen. The neck was stiff but could be flexed slowly without obvious pain. No neck vein distention was noted. The lungs were clear. The heart revealed continuous arrhythmia. The PMI was not localized and heart sounds were distant. There were no murmurs or thrills. There were bilateral guarding of the abdomen, with generalized hyperresonance and questionable generalized tenderness. Bowel sounds were normal. No organs or masses were palpated. The lower extremities were held in flexion and the patient cried out in pain when these were extended. There was what appeared to be some tenderness over the left calf, but there was no increase in heat, redness, or swelling. The skin and mucous membranes were dry and skin turgor was poor. Neurologic examination revealed generalized muscle wasting with "lead-pipe" rigidity. Deep tendon reflexes were strong and equal bilaterally. No pathologic reflexes were elicited. The patient responded with loud cries to painful stimuli and would withdraw all extremities. The cranial nerves were difficult to evaluate, but there was a questionable central type of weakness of the left facial muscles.

Laboratory studies were as shown in Table 1.

The patient was initially treated with 5 per cent dextrose by vein. Despite what appeared to be adequate rehydration he failed to excrete normal quantities of urine, and salt solutions were given intravenously. He was evaluated by a surgical consultant because of rising bilirubinemia, but surgery was thought inadvisable at that time. On the fourth hospital day the patient became febrile to 101 F. Tetracycline was begun. The patient began to deteriorate slowly and urine volumes fell to 300-600 cc. per day. Intravenous salt solutions were administered until a trace of presacral edema was obtained. On the ninth hospital day, the surgical consultant felt that surgical intervention was indicated, but the family refused permission to operate. Because of the rising BUN and potassium, in the face of a falling plasma bicarbonate, peritoneal dialysis was contemplated, but again the family would not give permission. The patient continued to deteriorate and died on the 14th hospital day.

Radiologic Findings

DR. JAMES P. WAGNER: Initial chest films are dated March 30, 1962. There is no acute inflammatory disease of either lung field or pleura. The heart is enlarged with left ventricular predominance. The aorta is uncoiled and somewhat tortuous, and the intrapulmonary vasculature is at the upper limits of normal. The bony framework is unremarkable.

A film of the abdomen dated March 29, 1962, reveals a normal distribution of bowel gas with no visualized mass lesions or intra-abdominal calcifications.

A cholecystogram performed April 5, 1962, reveals nonvisualization of the gallbladder on a single dose study. Residual contrast material was noted in the sigmoid and rectum.

An upper GI series in March, 1962, was normal. Films of the chest were obtained intermittently through 1964 and revealed episodes of recurrent congestive heart failure. On the final admission in March of 1964, the lungs were clear of consolidation and pleural effusion. The heart is again noted to be enlarged and mainly left-sided in nature although there is probably diffuse chamber involvement. The films were obtained in the supine projection and could not be compared adequately to the previous erect films. There were no dilated loops of bowel within the abdomen, and no evidence of free intraperitoneal or biliary air. Again no gross intra-abdominal calcifications are visualized. The bony structures are unremarkable except for some degenerative arthritis of the lumbar spine. The final films on March 19 are essentially unchanged.

Because of the clinical features of this case, I will briefly discuss the radiologic interpretation of the cholecystographic study. The oral cholecystogram examination is a highly accurate method of determining gallbladder disease. There are several prerequisites, aside from normal gallbladder function, for visualization. These are (1) essentially normal liver function and (2) intestinal absorption of the contrast material. Several causes of intestinal malabsorption exist; namely, primary bowel malabsorption defects, obstructing lesions in the pyloroduodenal area, and severe diarrhea. Some observers feel that diarrhea itself is not a hindrance to absorption. Certainly the contrast material must be taken to observe the gallbladder and, at times, this fact is overlooked when interpreting films. Residual contrast material in the bowel is a definite aid in determining whether or not the tablets have

TABLE 1.—*Laboratory Findings.*

	1/19/60	11/2/62	6/18/63	2/26/64	3/16/64	3/17/64	3/18/64	3/19/64	3/21/64	3/23/64	3/25/64	3/26/64	3/27/64	3/29/64
Hemoglobin	12.6		15.2	16.0	15.5	13.8	12.8				10.7	11.0		
WBC, thousands	10.2	5.5	6.0	5.4	11.4	13.0	8.6				23.7	14.2		
Neutrophils/Lymphocytes	63/24	48/37		80/12	78/13	89/5					81/13	92/7		
BUN, mg./100 cc.		20		21	111	107	106		66	67			95	122
Creatinine, mg./100 cc.					3.7									
Na mEq./liter				141	158	147	147	141	133	131	133	133	132	137
K mEq./liter				4.1	4.6	3.9	4.2	4.5	5.1	5.4	6.7	6.0	5.8	5.6
Cl mEq./liter	100			97	117	109	111	103	99	100	99	96	95	90
CO_2 mEq./liter	26			24	24	21	23	23	22	20	19	22	22	16
Total protein, gm./100 cc.		6.9	5.9			4.8				6.5			5.5	
Albumin		3.8	3.5			2.8				2.9				
Globulin		3.1	2.4			2.0				3.6				
Glucose, mg./100 cc.	117			160	163	150								
Serum glutamic oxaloacetic transaminase, units			17		27		16			25				
Bilirubin														
Total, mg./100 cc.			0.9		5.9		4.3				7.0		9.0	
Direct, mg/100 cc.			0.5		2.0		2.2				3.8		2.8	
Cephalin flocculation, units, 24/48 hr.		1+/1+	N/1+		N/1+		N/2+			N/1+			1/2+	
Thymol turbidity, units		2.2	3.6		1.7		1.9			1.5			2.4	
Alkaline phosphatase, Bodansky units		18.6	10.0		8.9		8.5			13.1			14.4	
Serum amylase, Somogyi units					<320									
Serum lipase, units					0.69									
Prothrombin time, sec.								13.5						
Urine:														
pH					6.0	5.0								
Specific gravity					1.015	1.015								
Albumin					1+	Trace								
Sugar/Acetone					N/N	N/N								
RBC					10-20	0-3								
WBC					50-100	50-60								
Casts					30-40	Occasional								

been taken. If these prerequisites are met, the single dose oral cholecystogram is accurate in over 90 per cent of the cases of cholecystitis with or without calculus formation.

If there is nonvisualization, several other approaches may be tried utilizing oral contrast media. The double dose and "four-day" Telepaque studies are all useful. The gallbladder will opacify with the double dose in a significant number of these patients and be normal. Calculi will become visible in a few others.

The examination of choice in the jaundiced patient is the "four day" Telepaque test. It is especially helpful in detecting common duct stones. The patient is placed on a relatively fat free diet and given two tablets (1 gm.) after each meal for four days. Paregoric may be given to control diarrhea. Radiography is performed on the fifth day. The ductal system and gallbladder themselves may not visualize, but in a certain number of patients, lucent stones may subsequently be demonstrated. The calculus is constantly being bathed by the iodinated opaque material, which allows interaction with biliverdin. This may produce the so-called "ring sign" which is a rim of radiodensity on the surface of the stone.

If the oral methods do not help, one may attempt intravenous or transhepatic cholangiography. If the cystic duct is obstructed, the gallbladder will not opacify with any of the aforementioned techniques.

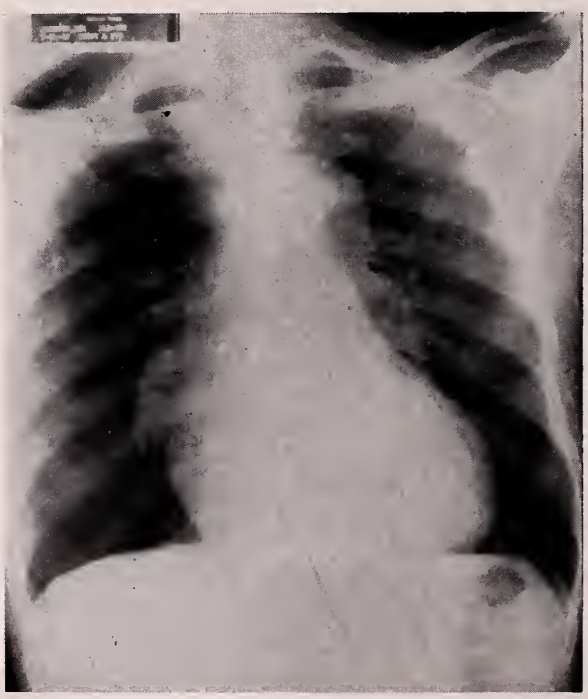

Fig. 1. Anteroposterior roentgenogram of patient's chest.

Clinical Discussion

DR. BURTON COMBES: Biliary tract disease was a major cause of death in this 84-year-old patient. Scrutiny of the protocol reveals that evidence of such disease was present, probably, for at least four years. A diagnosis of symptomatic biliary tract disease should have been made two years ago. If the patient had undergone appropriate surgery at that time, it is likely that we would not be discussing his case today. It seems appropriate then to reexamine the information we have before us, in order to determine when the diagnosis of biliary tract disease and its therapy could have been accomplished.

The first indication of possible gallbladder disease is recorded in 1960, when the patient was observed because of abdominal pain and vomiting of one day's duration. Physical examination was negative, apparently. Slight cardiomegaly and ECG abnormalities were noted. Further investigation was not carried out. One month later, the patient was seen again with a history of abdominal cramps and vomiting for two weeks. Such a story should have directed attention to a disorder of a hollow viscus; and with vomiting, disease of the upper GI or biliary tract should have been suspected. Unfortunately, although the patient was referred to the medicine clinic for work-up, he did not keep his appointment. Two years later, the patient reappeared with similar complaints of two episodes of abdominal pain and vomiting in the preceding ten days. We cannot be certain that the patient had been symptom free in this two-year interval. However, such quiescent periods frequently intervene in the natural history of chronic biliary tract disease.

Three weeks later, our attention was directed to the heart, for now the patient appeared with symptoms of left-sided heart failure. He was found to have cardiomegaly and an arrhythmia described as multifocal premature ventricular contractions on one occasion and auricular fibrillation at other times. I shall not discuss the nature of the cardiac disease, since this will be done by others. Presumably because the patient had a history of vomiting approximately three hours after going to bed, abdominal roent-

genograms were finally obtained and revealed a normal upper GI tract and a nonvisualized gallbladder. The interpretation of the latter observation is extremely important, and Dr. Wagner's comments on this finding are pertinent.

Thus, if we can be assured that the patient took his gallbladder dye, did not lose the tablets in vomitus or have them scurry through the intestinal tract, and if liver function had been appraised and found to be satisfactory, the finding of a nonfunctioning gallbladder would have been strong presumptive evidence that this man had chronic cholecystitis. Furthermore, the history of colic and vomiting would have strongly suggested that he had stones in his common duct, even in the absence of jaundice. A definitive diagnosis of biliary tract disease could, and should, have been made at this time.

The next observation that probably pertains to the biliary tract was the finding of an alkaline phosphatase of 19 Bodansky units in November, 1962. It is not clear why this was not pursued any further. An elevation of serum alkaline phosphatase is an important finding which always needs to be explained, particularly in terms of whether such an elevation is caused by a disorder of the liver and biliary tract, or of bone. When an elevated alkaline phosphatase level is an isolated finding, determination of leucine amino peptidase (LAP) activity of serum is helpful in diagnosis. An elevated LAP value indicates that the liver is responsible for at least some of the alkaline phosphatase rise, whereas a normal LAP value indicates that bone is the probable source of the elevated serum alkaline phosphatase. Even though the patient was in heart failure in November, 1962, and heart failure may of itself be associated with some rise in alkaline phosphatase levels, this degree of elevation (19 Bodansky units) is most unusual for failure alone. By June, 1963, the alkaline phosphatase had decreased to 10 Bodansky units —strong evidence that a malignant lesion of the liver or biliary tract had not been present in 1962. Rather, this finding supports the contention that an elevated alkaline phosphatase was due to incomplete obstruction of the biliary tract in this patient.

Finally, we come to the recent episode in March, 1964, when our patient was seen, first with confusion and poor food intake, followed by lethargy, lack of bowel movements, and pain on motion of his lower extremities. Physical examination disclosed low blood pressure, signs of dehydration, and neurologic findings which could be attributed to a combination of decreased cerebral blood flow resulting from dehydration, and cerebral vascular disease. On March 16, he had azotemia, hypernatremia, and hyperchloremia, in large part the consequence of dehydration due to inadequate water intake. Despite a marked stimulus for water conservation, urine specific gravity was only 1.015, indicating impaired renal function. At this time, hyperbilirubinemia, normal flocculaton tests, and an alkaline phosphatase of 9 Bodansky units were found; these are the findings of an incomplete obstructive jaundice. Subsequently, after an initial decrease in bilirubin, coincident with rehydration, serum bilirubin and alkaline phosphatase levels rose, while flocculation tests remained normal. These are the chemical findings of progressive obstructive jaundice.

When we first saw this patient, his water deficit had been replaced. Despite this, urine output did not increase and only when sodium-containing fluids were administered did urine output and clinical status improve. This response to sodium administration suggested that extracellular fluid had been sequestered in an area of inflammation, presumably in the region of his biliary tract. At this time, even though the diagnoses of chronic cholecystitis and cholelithiasis with common duct obstruction were made, surgical intervention was not recommended because disorientation persisted even though vital signs were stable, rehydration had been accomplished, and fever and evidence of cholangitis were absent, suggesting that irreversible cerebral damage had occurred. Later on, however, when fever and leukocytosis were present, cholangitis was considered likely, and cholecystectomy was recommended. Our rationale was that the immediate complications of obstruction could be treated by decompression of the biliary tract. Elective cholecystectomy could then be performed in the future if further clinical improvement followed. The patient's family decided against surgery, and I believe the patient died from complications of biliary tract disease.

Before addressing ourselves to the question, "Should this patient have been offered cholecystectomy at least two years ago?," let us examine some of the information available about the natural history of cholelithi-

asis. Gallstones are common. Their incidence increases with age, and in an autopsied population, gallstones are found in some 25 to 40 per cent of persons so examined. The incidence is even higher in certain groups, for example in diabetics. Of those with stones, up to 60 per cent have had no symptoms recognized as attributable to stones by either the patient or his attending physician. The few reports concerned with the subsequent history of living patients shown to have gallstones by roentgenogram, or at the time of laparotomy for other causes, but without symptoms of cholelithiasis previously suspected, demonstrate that approximately 20 per cent develop symptoms of biliary pain or colic, and an additional 30 per cent develop symptoms of dyspepsia over a 10- to 20-year period. About half remain asymptomatic. What about the risks involved in cholecystectomy? Elective cholecystectomy in medical centers probably entails an operative mortality of 1 to 1.5 per cent. An additional 0.5 per cent sustain injuries resulting in common duct stricture, a serious complication. Once symptomatic biliary tract disease ensues, mortality increases to approximately 3 per cent. Thus the risks of waiting definitely rise. However when confronted with a middle aged or older person with silent gallstones, who has a 50 per cent chance of escaping complications of the stones, it seems reasonable to me *not* to recommend that elective cholecystectomy be performed. On the other hand, once symptoms of gallstones appear, surgery should be recommended, since there is no medical cure for cholelithiasis. Our patient appears to have had symptomatic cholelithiasis dating back at least to 1962. Despite the presence of cardiac disease, elective cholecystectomy should have been offered at that time.

DR. FOUAD A. BASHOUR: The first phase of this patient's cardiac illness known to us was in 1960 when we saw him with the ECG finding of T-wave changes in leads V_2 to V_6. The axis deviation then was left; and, not having the ECG's for study now, it can be guessed that there might also have been some evidence of myocardial injury. There was, however, no history of chest pain, or no such history was given to us. Since the patient was not hypertensive, nor hyperthyroid, nor was there any other clinical evidence to aid in the interpretation of the clinical picture, we have to assume that he most likely had arteriosclerotic heart disease. It is noteworthy that the patient was 81 years old at that time.

The second phase of the clinical picture was from March to September, 1963, during which time the patient developed atrial fibrillation, and the ECG showed progressively decreased voltage of the QRS complex. The left axis that was described in 1960 had reverted to normal. In addition, the patient had developed a small R′ in AVR. With the history of repeated attacks of heart failure, as evidenced by the chest roentgenogram and the clinical picture described in the outpatient department, it is also possible that the patient was having repeated small pulmonary emboli and possibly increased pressure in the right ventricle.

With this background in mind we come to the last episode, which was heralded by the patient's admission to the hospital because of upper GI problems, probably gallstones. He then had no evidence of heart failure. Venous pressure was 10 cm. of water, the neck veins were flat, and no peripheral edema was present.

In this last admission, the ECG's revealed progressive decrease in the QRS voltage. If you add leads I, II, and III, the QRS voltage would come to about 5 mm. on paper. Therefore, it can be suggested that the patient either had pericardial effusion that was missed by the roentgenogram and clinically, or that he went progressively into heart failure and then developed some degree of pleural effusion.

The clinical picture on admission indicated that the patient was dehydrated. As far as we can detect, from the record, for this he was given salt and water. There is one sentence in the clinical summary that deserves some comment. It was stated that salt solution was administered intravenously until a trace of presacral edema was obtained. I wonder if we did not push fluids too much then. Since the patient was unattended before he died, it is quite possible that he had developed pulmonary edema. I doubt that the patient died from gram-negative bacteremic shock. We have to assume from the record that the blood pressure was within normal limits. I can only conclude from the clinical record that the patient had atrial fibrillation which was most likely due to arteriosclerotic heart disease. I believe that he died from heart failure, and this was

possibly aggravated by the extra fluids which were given.

One other point I would like to add about his ECG. As you notice, we have no reliable clinical history. The main clinical evidence of heart disease was the finding of atrial fibrillation by the ECG. Atrial fibrillation can be divided into two types: the coarse and the fine types. The fine atrial fibrillation means that the f-waves are barely seen; and coarse atrial fibrillation means that there are prominent f-waves. Atrial fibrillation of the coarse type occurs in hyperthyroidism, in rheumatic heart disease, and in early stages of hypertensive cardiovascular disease. The fine type of atrial fibrillation occurs mainly in arteriosclerotic and luetic heart disease. Therefore, having a fine atrial fibrillation, this patient probably can be said to have had arteriosclerotic heart disease.

DR. JEAN D. WILSON: For completeness, I think that one other possible etiology should be considered in the differential diagnosis of congestive heart failure of unknown etiology, and that is primary amyloidosis. In fact, a moderately strong case could be constructed for the diagnosis of primary amyloidosis in this case. This man was originally from Ellis County which is the home of a very large kindred of patients with primary familial amyloidosis, and he might be related to that family. In addition he had evidence of renal failure and albuminuria, as well as congestive heart failure. Unfortunately, opacities of the lens prevented any examination of the vitreous for the vitreous bodies which sometimes occur in this condition, and he did not have either biopsy or electrophoretic studies to confirm or exclude such a diagnosis. Nevertheless, the diagnosis of primary amyloidosis remains an attractive possibility in this case.

Pathological Findings

DR. BRUCE D. FALLIS: Our attention in this case was directed basically to three problems, (1) the nature of the biliary tract disease, (2) the nature of the heart disease, and (3) the immediate cause of death. As you might anticipate in a case of this type, occurring in a very aged person, there were many necropsy findings, and I shall try to simplify these as I present the case. The gallbladder contained several mixed stones and was the seat of severe chronic cholecystitis. The cystic duct was somewhat dilated, and through the cystic duct there had passed five faceted, mixed gallstones. These stones were in the common bile duct and had more or less completely obstructed the duct. All the extrahepatic bile ducts were dilated, and the intrahepatic ducts were also dilated and bile-stained. The liver weighed 1,620 gm. and showed the changes of chronic passive congestion, compatible with long-standing heart failure. Microscopically there was an inflammatory infiltrate in the periportal connective tissue, composed largely of neutrophilic leukocytes (Figs. 2 and 3). However, the degree of this acute ascending cholangitis was slight. There were no abscesses in the portal tracts or elsewhere in the liver. The central veins were somewhat dilated as were the paracentral sinusoids as a reflection of chronic heart failure. There was marked bile stasis. Dark greenish brown pigment was present in the bile canaliculi between adjacent liver cells and also in the Kupffer's cells. In summary, the patient did have an obstructive jaundice from gallstones in the common duct, and accompanying this a slight degree of acute ascending cholangitis. There was no evidence of biliary cirrhosis.

The heart was enlarged to 700 gm., at least twice the normal weight. In addition to hypertrophy, all the chambers were dilated. There was a large organizing thrombus in the right atrium, and there were old sclerotic plaques in the right atrium, particularly in the right auricular appendage, indicating that there had been numerous old thrombi which had become organized and incorporated into the endocardium. In the left atrium there was a large organizing thrombus. Just posterior to the apex, involving the posterior wall of the left ventricle and the posterior part of the interventricular septum there was a relatively small transmural myocardial infarct. The coronary arteries, however, showed only slight arteriosclerosis, and this was true also of the aorta and the other major arteries. The outstanding feature in the heart was its enormous size and the firm consistency of the myocardium. On the cut surface of the myocardium grossly there were small, translucent foci, averaging 1 mm. in diameter, which were slightly glassy and sunken. Microscopic study of the old myocardial infarct showed only dense fibrous tissue replacing the myocardial fibers. There was no evidence of a recent myocardial infarct. Throughout the myocardium there were small discrete foci, corresponding to the glassy foci noted grossly, composed of

pale, eosinophilic, hyaline material which surrounded the individual myocardial fibers. Stains for amyloid were positive. High magnification showed the amyloid was deposited as rings, apparently in relation to the basement membrane or the adjacent reticulin framework of the myocardial fibers and the encased myocardial fibers were atrophic or had disappeared entirely (Figs. 4 and 5). There were also deposits of amyloid in the intima of a pulmonary vein and around the epicardial fat cells. There was, however, little amyloid deposited in the other viscera, although minimal deposits of amyloid were identified in the lungs, spleen, esophagus, kidneys, and adrenals.

Cardiac amyloidosis[2, 3] is an uncommon but by no means rare condition. It occurs in perhaps 3 per cent of persons over age 50. It usually appears after the age of 70 and is more frequent in men than in women. It is actually a form of primary amyloidosis. There may be minimal amounts of amyloid in other tissues, particularly in smooth muscle in other parts of the body, but the curious feature of the amyloidosis of senescence is its selective involvement of the heart, almost to the exclusion of other regions, and the heart is the only site of clinically significant involvement. Most of these cases are discovered at necropsy and have not been accompanied by clinical manifestations; that is, the amyloidosis is an incidental autopsy finding. In a few cases, however, the deposit of amyloid in the heart is severe, as in the present case, and leads to progressive heart failure. The clinical features suggestive of cardiac amyloidosis of senescence are the occurrence in a very aged person (usually a male) of rather slowly progressive

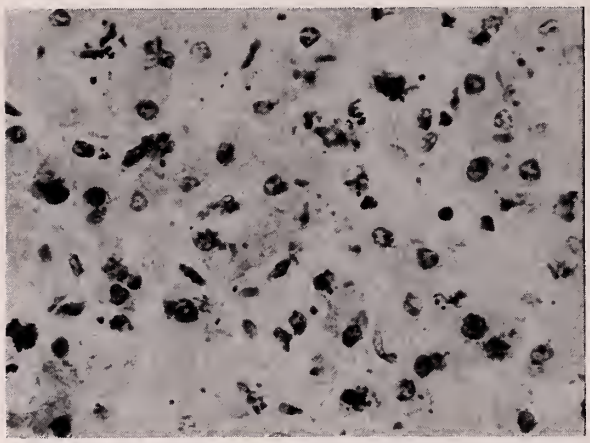

Fig. 2. Liver, high magnification. Liver cells in the center of a liver lobule are shown. Dark plugs of bile are present in bile canaliculi and in Kupffer's cells.

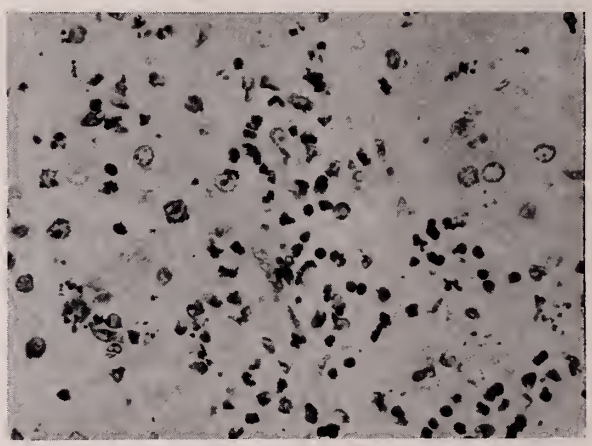

Fig. 3. Liver, high magnification. A portal tract is seen. Neutrophilic leukocytes are present within the connective tissue and in the lumen of a small bile duct.

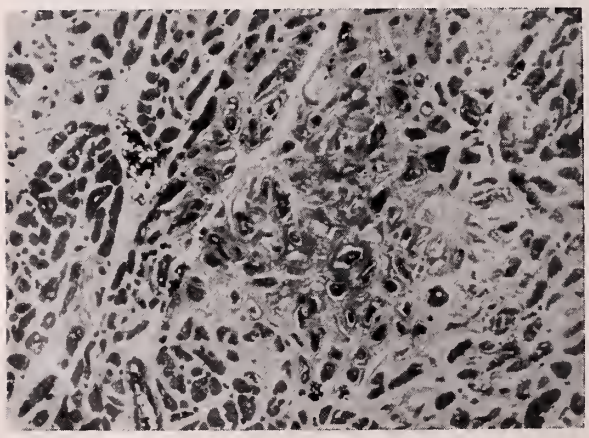

Fig. 4. Heart, low magnification, crystal-violet stain. A pale focus of amyloid is present in the center of the field. In the original stain, this material was red in contrast with the dark blue myocardial fibers.

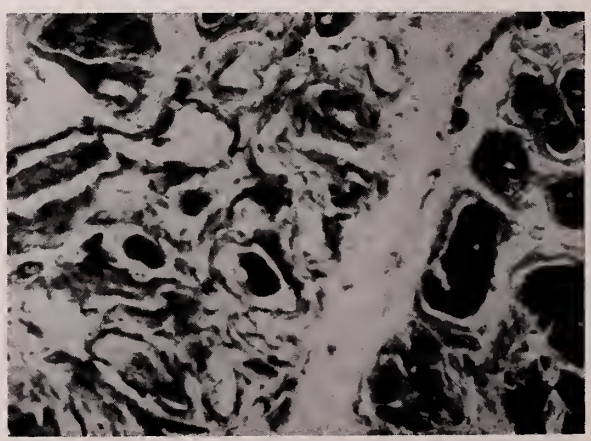

Fig. 5. Myocardium, high magnification. Rings of amyloid material surround myocardial fibers. The amount of amyloid is greater on the left, where the myocardial fibers are noticeably atrophic.

heart failure, the absence of any other demonstrable cause for heart failure, and certain ECG changes, especially a very low voltage ECG and often arrhythmias. Paresthesias caused by peripheral nerve involvement by amyloidosis occasionally accompany the heart failure.

The lungs of this patient weighed 1,700 gm., the result of severe pulmonary edema, which presumably occurred terminally. The main pulmonary artery on the right contained a large thrombotic pulmonary embolus, which was rather firmly adherent to the wall because of early organization (Fig. 6). The embolus had been present for at least three or four days. On the opposite side in the main left pulmonary artery there was an embolus of comparable size, also present for several days and beginning to become organized. There was a single healed infarct in the lungs, but there were no recent pulmonary infarcts. The source of large emboli was doubtless the leg veins. Microscopic study revealed some small thrombotic emboli, many of which probably represent fragments of the mural thrombus in the right atrium. Many of the small emboli were in varying stages of organization. There were, however, not a great number of these emboli, and the patient does not have the findings of thrombo-embolic pulmonary hypertension. On the right, there was a fibrinous pleurisy, secondary to a severe necrotizing bronchopneumonia. The pneumonia was extensive and bilateral with foci of hemorrhage and alveolar septal necrosis, giving the characteristic appearance of a *Pseudomonas* type of necrotizing pneumonia. In addition to this there were some deposits of lipid in the lungs. Large foamy macrophages and fat-laden giant cells indicated the type of lipid pneumonia that occurs as the result of aspiration of mineral oil. Many old persons, particularly those with gastric or intestinal complaints, take mineral oil quite often, and many such persons aspirate varying amounts of this mineral oil, producing sizable areas of lipoid pneumonia. The amount in this case was very small.

Changes in the other viscera were slight and included an acute and several old infarcts in the spleen (doubtless caused by emboli from the mural thrombi in the left atrium), acute infarcts and cloudy swelling in the kidneys, thrombotic emboli in the branches of the renal artery on one side, hemorrhagic cystitis as the result of catheterization on the ward, and ascending bilateral pyelitis and pyelonephritis of slight degree (Figs. 7 and 8).

The autopsy then, in summary, revealed primary amyloidosis involving the heart to a severe degree and to a slight degree other viscera. Severe cardiac hypertrophy and dilatation were accompanied by the usual changes of severe heart failure—acute pulmonary congestion and edema, acute and chronic passive congestion of the liver, and subcutaneous edema. In addition, the patient had serious biliary tract disease with cholelithiasis, choledocholithiasis, and obstructive jaundice. Furthermore he had thrombi in both the right and the left atria, emboli and infarcts involving the spleen and kidney, and a large number of emboli in the lungs, including two large organizing thrombotic pulmonary emboli within the major right and left pulmonary arteries themselves. The

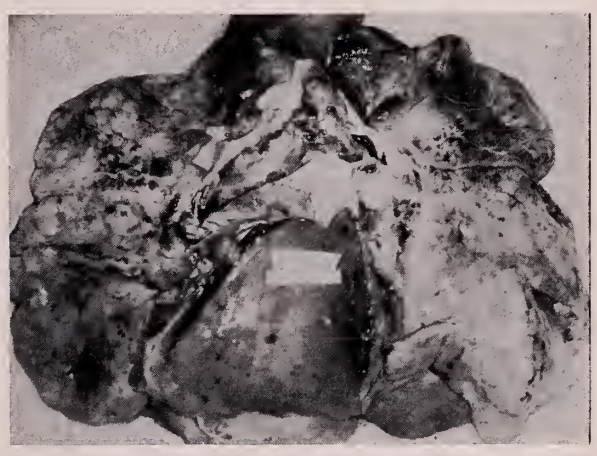

Fig. 6. Right lung. Note the large thrombotic embolus in the right pulmonary artery. The middle lobe, under the label, has a dull, opaque, granular pleura resulting from fibrinous pleurisy.

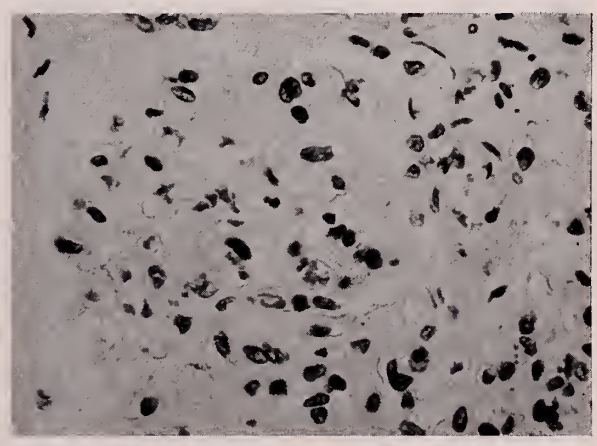

Fig. 7. Kidney, high magnification. One glomerulus is shown. The walls of the capillaries are thickened by an acellular hyaline material representing amyloid.

final event was the development of a severe necrotizing bronchopneumonia, with pleurisy and slight fibrinous pericarditis complicating it. An incidental finding was a minor urinary tract infection. To pick one of these as a specific terminal event is, I think, implausible. Our final conclusions were that he died of a combination of biliary tract disease and cardiac amyloidosis, complicated terminally by thrombo-embolic complications and severe bronchopneumonia. Probably the fever that was noted clinically was secondary to his severe bronchopneumonia, this being the most significant infection that he had. Dr. Ashworth has brought some electron micrographs and will discuss in a little further detail the basic nature of the amyloid material itself.

DR. C. T. ASHWORTH: While electron microscopy of autopsy material has very great limitations because of autolysis, we have found it possible to obtain from formalin-fixed autopsy specimens some information about the fine structure of certain resistant substances such as amyloid. Amyloid has been studied by electron microscopy recently, and its fine structure is well known.[1] The basic unit of amyloid is a fine filament about 100 Å in diameter. These filaments are nonperiodic and are in haphazard and tangled array. Fig. 9 reveals the appearance (electron microscope) of the amyloid deposits in this case around the heart muscle fibers. The amyloid forms a ring-like encasement of the myocardial fibers. As we see the amyloid in higher magnification (Fig. 10) its fine filamentous nature becomes more apparent. The amyloid appears to replace the normal basement membrane of the myocardial fibers. This might be taken as an indication that the formation of amyloid is, in some instances, related to the production of altered cell basement membranes.

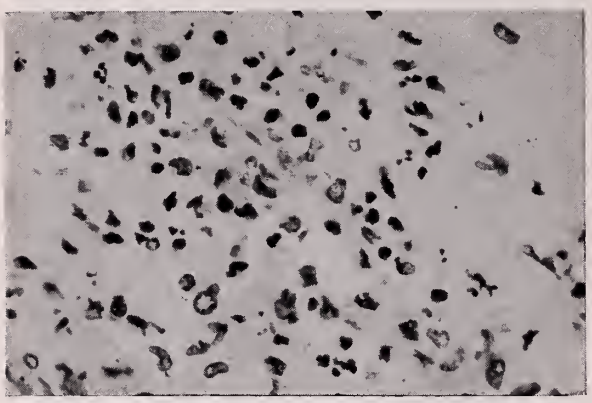

Fig. 8. Kidney, high magnification. The section is taken from the renal medulla and shows a collecting tubule with an infiltrate of neutrophilic leukocytes in the adjacent connective tissue.

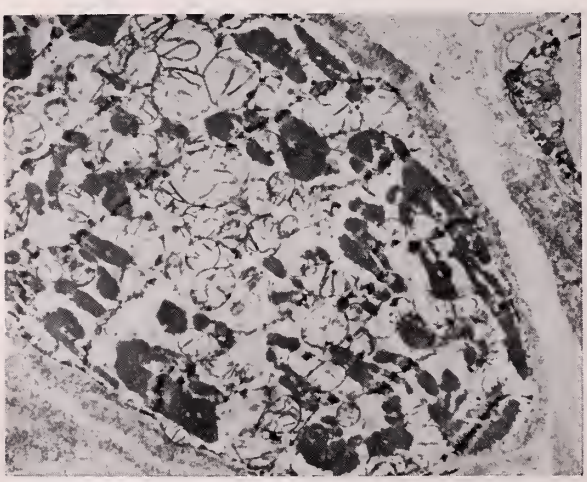

Fig. 9. Electron micrograph of amyloid deposit around myocardial fiber. Cytoplasmic degradation of the muscle cells is from the postmortem autolysis and formalin fixation.

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Drs. Fallis and Ashworth, Department of Pathology.

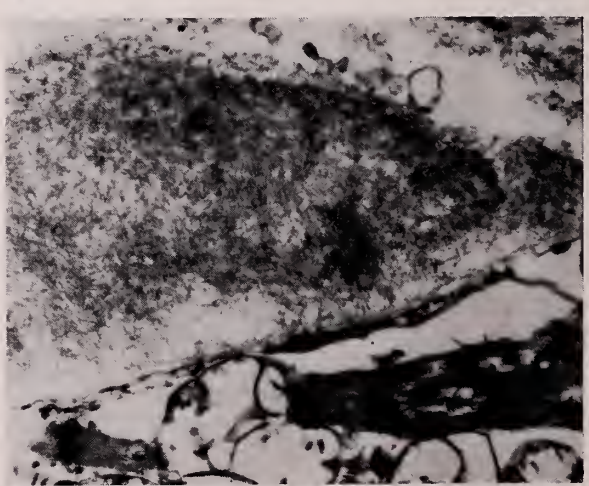


Fig. 10. Higher magnification of amyloid filaments in cardiac amyloidosis. The filaments are devoid of periodicity and average about 100 Å in diameter.

NEWS

AMA Delegates Reelect Dr. Rouse; Texas Resolutions Not Approved

DR. MILFORD O. ROUSE, Dallas, was reelected Speaker of the House of Delegates at the 113th annual meeting of the American Medical Association at San Francisco on June 21-25.

Dr. Rouse was commended for his continued efforts to further enhance the effectiveness of the 228-member House of Delegates, the AMA's top policy-making body.

On policy issues, however, the Texas delegation to the AMA was not fully successful in its efforts to alter existing positions of the American Medical Association in regard to health hazards of cigarette smoking, "bricks and mortar" federal aid, and area-wide planning of hospitals.

Texas was one of ten states which introduced resolutions pertaining to cigarette smoking. The Texas resolution pointed out that the Advisory Committee to the Surgeon General of the United States Public Health Service, after an exhaustive review of all available evidence, has concluded that cigarette smoking is a health hazard. The resolution urged the AMA to officially recognize cigarette smoking as a health hazard and to encourage physicians to cooperate with other groups and organizations in stepping up educational programs to reduce this hazard. This expression previously had been adopted as policy of the Texas Medical Association at the 1964 Annual Session in Houston.

After considering the Texas resolution and those from nine other states, the AMA adopted a substitute statement which included these highlights:

1. The AMA is on record as recognizing that a significant relationship exists between cigarette smoking and the incidence of lung cancer and certain other diseases, and that cigarette smoking is a serious health hazard.

2. The AMA recognizes the contribution of the Surgeon General's Committee in its comprehensive report. In keeping with one of the recommendations of that report, the AMA and the National Education Association have adopted a resolution urging elementary and secondary schools to include programs on smoking and health in their health education curriculums.

3. Recognizing the deleterious effects of tobacco on human health, the AMA House of Delegates previously indicated that extensive research was necessary to clarify its exact relationship to health. The House of Delegates expressed pleasure with the establishment of a research program by the American Medical Association Education and Research Foundation.

The House of Delegates reiterated the AMA's policy of continued support of "bricks and mortar" federal aid in hospital and medical school construction, and turned down resolutions submitted by Texas, Nebraska, Iowa, and Louisiana.

The Texas resolution had asked the AMA to reevaluate its statements related to federal aid, and to formulate uniform and consistent policies which (1) fully support private initiative and endeavor, (2) oppose proposals that may lead to the nationalization of medicine regardless of the route such proposals may take, and (3) work to reduce existing federal aid programs including "bricks and mortar" and thus assist the federal government in balancing the budget and reducing the national debt.

Texas delegates and others appeared before the AMA reference committee to express concern over the continued expansion of federal assistance programs in the area of interest to the medical profession. They pointed out that the explosion of federal financing has tended to discourage private and local funding, and that the growth of federal support could lead to the nationalization of medicine. These arguments were lost, however, as the House of Delegates reiterated the AMA's policy of support of "bricks and mortar." The AMA House further stated that the advantages and desirability of multiple source financing should be kept clearly in mind to prevent federal domination, and that the programs continue to be operated on a voluntary basis with local control.

The AMA House of Delegates took no action on a third Texas resolution on area-wide planning for hospitals. The resolution asked the AMA to urge physicians to continue to take an active role at the local level in planning medical and hospital services for their communities. At the same time, the resolution expressed opposition to compulsory area-wide planning for hospital facilities and requested that all constituent state societies and component county medical societies be informed so that they, in turn, could alert the governing boards of the hospitals in their jurisdictions to the dangers inherent in compulsory planning. Somewhat similar resolutions were in-

troduced by the Nebraska and Louisiana delegations.

After hearing testimony, the AMA reference committee stated that it agreed with the resolutions, encouraging physician participation and leadership in voluntary local community planning. The reference committee also agreed that the AMA should be committed to oppose vigorously the principle of compulsory area-wide hospital planning. The reference committee pointed out, however, that the resolutions reaffirmed existing policies of the AMA and therefore recommended that no action be taken on them. The AMA House of Delegates approved the reference committee's report as well as a recommendation that a national conference on area-wide planning of hospitals and related health facilities be sponsored under the auspices of the AMA. Dual purposes of the conference will be cross communication of current experiences of medical societies and the development of guidelines for physician participation in area-wide planning.

9,830 Join Texas 65 In Second Enrollment

An additional 9,830 Texans, 65 years of age and past, acquired broad health insurance coverage under the Texas 65 health insurance program during the second enrollment period which closed June 15. H. Lewis Rietz, president of the Texas 65 Health Insurance Association, announced that additional enrollment periods will be conducted from time-to-time. However, he said, no date for the third enrollment has been set.

Over 50,000 were enrolled in Texas 65 during the initial enrollment period held last October.

Texans who turn 65 between open enrollment periods have 31 days following their birthday in which to enroll in the program. Those who were 65 and over during the enrollment period just ended must now wait until the next open enrollment period to purchase Texas 65, Rietz said.

Sixty-four insurance companies are participating to make the voluntary health insurance program possible, in accordance with special legislation enacted unanimously by the 1963 Texas Legislature and signed by Governor John Connally.

UT Medical Branch Students Receive Clinical Training Abroad

IN MEXICO, when a patient goes to the hospital he expects to die, reports University of Texas Medical Branch student Mike Schlev who adds that even if the patient is cured, he is still convinced that he will stay there until he dies.

Mike, with Charles Houser, was reporting on his experiences following six weeks of observation at the Tropical Disease Institute in Mexico City.

Mike and Charles are two of 11 students from the Medical Branch who have studied abroad. One of the problems of medical progress is that diseases, when conquered, can no longer be seen firsthand by medical students. To combat this deficiency in clinical training, a foreign training elective program was established in September, 1960, which enabled students to study in other countries and fulfill medical school elective requirements at the same time. Four of the students who are still at Galveston, Mike, Charles, Dr. Dan Johnson, and Milton Husbands, recently discussed their most outstanding memories.

The practice of medicine in Mexico impressed Mike and Charles even more than the diseases they saw. "Doctors there treat five times as many people as U. S. doctors," Mike said.

"The government runs all the hospitals, and the government has multiple holidays—some as long as two weeks. During these holidays, the hospitals close. Only the critically ill remain, with one doctor and one nurse for each floor. Patients come back in after the holidays."

To Mike and Charles, the most valuable experience of their seven-week trip came the last week when they accompanied Dr. Molina-Pasquel and two other members of the hospital team to the southern jungle of Mexico within 30 miles of Guatemala. The purpose of their expedition was to study and treat onchocercosis.

"Virtually everyone in that area contracts onchocercosis," Mike said. The disease could be conquered if the fly that carries it were wiped out, but the students discovered that this was an insurmountable problem. The flies breed in jungle streams, and the density of the jungle and hundreds of streams in that part of Mexico make eradication of the fly virtually impossible.

The students, along with the medical team, rode mules from Huixtla up to remote mountain villages to give the Indian inhabitants medical care. In each village, from 25 to 30 patients were seen, and usually seven operations were required in each village. From necessity, some of the surgery was performed under adverse conditions.

One man had a nodule removed from his face while he was lying on a wooden bench on the front porch of an adobe house. A chickenyard surrounded the porch.

Dr. Dan Johnson, who studied at the lung and chest hospital of Centre Chirurgical Marie—Lannelongue, discovered that he was a medical rarity in France because he was 26 years old and his tuberculin patch test was negative. France has the highest tuberculosis rate of any western European country, he reported, and in addition, vaccination for tuberculosis, which is advocated by the French government, results in positive tuberculin patch tests.

His most interesting experience, he said, came when a Russian cardiovascular specialist demonstrated Russian instruments for suturing tissue with staples. Dan assisted him with demonstrations and translation, and learned while talking to him after the lecture that Russia's successes and failures with cardiovascular surgery correspond closely with ours.

Milton Husbands, who studied at the American University in Beirut from May to September, discovered that the 250-bed hospital inside the UN compound was one of the best-equipped in the Middle East. As part of his work, Milton worked in the nutrition laboratory under Dr. Donald McLaren. They studied the effects of trying to raise the level of vitamin A in colostrum by injections at the time of delivery. Their research showed that the level was not boosted by overwhelming injections.

Even though the research as-

pects of obstetrics interested Milton and he hopes one day to do research, the experiences he cherishes the most came in clinical practice. He delivered his first baby in Beirut. "It was a beautiful little girl, without the usual wrinkles and fatty material."

Any red-blooded American doctor might recall such pride in his first delivery wherever it was performed, but to the students who gave helping hands while learning themselves in unfamiliar circumstances, there was a special added satisfaction.

TMA Group Insurance Programs Improved

Dr. A. R. Hazzard, Austin, chairman of the Committee on Association Insurance Programs, has announced that private flying is now covered under the disability program effective July 1, 1964, and that benefits for nervous or mental disease under the major medical program will be increased in the near future.

Under the new arrangement, the Lumbermens Mutual Casualty Co. has agreed to pay full benefits for a period of one year for disabilities due to accident resulting from private flying. The accidental death and dismemberment benefits will also apply.

The company also has agreed to increase benefits from $1,500 to $3,000 on nervous or mental disease under the major medical program.

In making the announcement, Dr. Hazzard pointed out that these additional benefits, which were granted at no increase in premium, are a continuation of the Committee's efforts to provide the broadest coverage possible under the group plans sponsored by the Texas Medical Association.

MSEA Ex-President Receives Plaque

C. Lincoln Williston, Executive Secretary of Texas Medical Association, last month received a plaque denoting appreciation from the Medical Society Executives Association at their meeting in San Francisco. Mr. Williston has just completed a year as president of that group.

Summing up some of the accomplishments of MSEA during the past year, Mr. Williston discussed the enlarged educational program of the organization and the surveys of four new study committees.

The 350-member association has stepped up activities this year, almost doubling the number of committees in its organization. This comes at a much needed time, Mr. Williston said; "Demands upon the medical society executive are greater today than ever before."

The MSEA has now expanded its educational program to a total of four annual conferences: the first in August in Chicago in conjunction with the AMA Institute; the second as a part of the AMA winter meeting; the third, two separate seminars in management; and the fourth, the annual conference which is a part of the AMA annual meeting. A further development is the completion of arrangements to publish a manual on medical society administration.

Four new committees of the MSEA have been circulating questionnaires to gather information on certain medical society problems. Two groups are studying attendance at annual meetings: one is concentrating on the factors that influence attendance and the other on types of programs that attract better than average attendance. A third survey being made is about retirement programs for medical society executives and their staffs, and a fourth is identifying characteristics of successful and unsuccessful committees.

Personals

Dr. James W. Kemper of Houston is president of the Texas Rheumatism Association. Vice president is *Dr. C. Collom Smith* of Waco, and secretary is *Dr. Homer R. Goehrs* of Austin.

Dr. Jack Pritchard of Dallas is treasurer of the American Gynecological Society.

Officers of the Texas Diabetes Association are *Dr. John Chriss*, Corpus Christi, president; *Dr. Leonard Flohr*, Dallas, first vice president; *Dr. Leonard Robbins*, Houston, secretary-treasurer; *Dr. Alfred Leiser*, Houston, second vice president; and *Dr. William Daeschner*, Galveston, president-elect.

New officers of the Texas Physical Medicine and Rehabilitation Society are *Dr. David Hoehn*, Fort Worth, president; *Dr. J. S. Doughety*, Dallas, vice president; and *Dr. Charles Peterson*, Houston, secretary.

Daughters of *Dr. and Mrs. S. C. Walker* of Hempstead recently received degrees in Galveston. Miss Sara Walker received the M.D. degree from the University of Texas Medical Branch and Miss Gale Walker the B.S. degree from the School of Nursing.

Dr. Ray K. Daily of Houston has been designated as the official AMA representative to the Second Congress of the European Society of Ophthalmology held in Vienna June 7-13.

Dr. Tom W. Duke of Amarillo recently has been elected to membership on the Counseling Committee on Church and Society of the United Presbyterian Church.

Dr. M. O. Rouse of Dallas has been named an honorary member of the Texas Hospital Association.

Dr. George E. Strickland of Henrietta has been named outstanding citizen of that community by the Junior Chamber of Commerce there.

Dr. Moody Bettis of Houston has been named the new psychiatrist-coordinator of the Office of Mental Health Planning replacing *Dr. Spencer Bayles* who is returning to private practice.

Dr. Denton Kerr of Houston and *Pauline Duffield*, librarian of the Texas Medical Association Memorial Library, have been named honorary members of the Texas Medical Assistants Association. New officers of TMAA are president, Mrs. Helen Kirk Lauve, Galveston; president-elect, Josephine Estrada, San Antonio; vice president, Mrs. Jan Mustonen, Garland; recording secretary, Viola Davis, Baytown; treasurer, Mrs. Gwen Martin, Amarillo; immediate past president, Mrs. Barbara Dickerson, Fort Worth; corresponding secretary, Gladys Farris, Galveston; historian, Mrs. Anita McCarthy, Baytown; chaplain, Mrs. Jolyn Weiss, Brenham; and parliamentarian, Mrs. Barbara Wray, Dallas.

Private Clinics and Hospitals Association of Texas is headed this year by *Dr. F. Stanley Ewing* of Sinton. President-elect is *Dr. J. F. Peters* of Alvarado.

Dr. William A. Spencer, director of the Texas Institute for Rehabilitation and professor and chairman of the Department of Rehabilitation, Baylor University College of Medicine, Houston, will deliver the keynote address at the

41st annual conference of the American Physical Therapy Association in Denver July 6-10.

Dr. Frederick R. Guilford, Houston otolaryngologist, has been appointed Texas state chairman of the Deafness Research Foundation, a national voluntary nonprofit organization that promotes scientific research into all types of ear disorders, fosters greater public awareness of the seriousness of deafness, and is currently conducting a nationwide public education campaign urging people with ear disorders to bequeath their inner ear structures to medical research.

Pathology Board Certifies 14 Texans

Fourteen Texas physicians have been named diplomates in pathology, the American Board of Pathology has announced.

They are:

A. Q. Da Silva and Harold L. Harle, Austin; William L. Bendel, Jr., Eufronzyna J. Kozlowski, Gene H. Moore, and Harold H. Varon, Dallas; Vincent C. Cirone and Feliks Gwozdz, Fort Worth; John D. Waggener, Galveston (neuropathology); Robert S. Donner, Edwin W. Godfrey, Philip J. Migliore, Minoru Suzuki, and John A. Webb, Houston; Hector M. Perches and Ruben C. Santos, San Antonio (forensic pathology); and Richard D. Schultz, El Paso.

14 Texans Certified By Ob&Gyn Board

The following Texas physicians were examined and certified by the American Board of Obstetrics and Gynecology, Inc., on May 1:

James Alan Alexander and Richard S. Mann, Houston; John W. Boldt, Robert L. P. Edwards, William E. Locke, and John P. Pfeiffer, Jr., San Antonio; David L. Bond, Garland; Edmund M. Brophy, Dallas; Capt. John F. Fulton, Sheppard Air Force Base; H. Ross Garza, Corpus Christi; Luis Marroquin, Port Arthur; Thomas Lee Martin, Victoria; Capt. Harold R. Misenhimer, Lackland Air Force Base; and Andrew B. Pumphrey, Jr., Fort Worth.

New Books In TMA Library

American Academy of Pediatrics: **Report of the Committee on the Control of Infectious Diseases**, 4th ed., Evanston, Ill., American Academy of Pediatrics, 1964.

Baer, R. L., and Kope, A. W., eds.: **Year Book of Dermatology**, Chicago, Year Book Medical Publishers, 1964.

Bergler, E.: **One Thousand Homosexuals**, Paterson, N. J., Pageant Books, Inc., 1959.

Boies, L. R. et al.: **Fundamentals of Otolaryngology**, 4th ed., Philadelphia, W. B. Saunders Co., 1964.

California Tumor Registry: **Cancer Registration and Survival in California**, Berkeley, State of California Department of Public Health, 1963.

Castleman, B., and Burke, J. F., eds.: **Surgical Clinicopathological Conferences of the Massachusetts General Hospital**, 1st ed., Boston, Little, Brown and Co., 1964.

Cecil, R. L., and Conn, H. F, eds.: **Specialties in General Practice**, 3rd ed., Philadelphia, W. B. Saunders Co., 1964.

Chicago Medical Society: **Conference Papers**, Chicago, Chicago Medical Society, 1960.

Coates, J. B., Jr.: **Activities of Surgical Consultants, vol. 2, U.S. Army Medical Department Surgery in World War II**, Washington, D. C., Government Printing Office, 1964.

Cox, B. S., and Huizinga, J., eds.: **Heredity and Variability of Micro-Organisms, Proceedings of the Conference at the N. F. Gamaleya Institute of Epidemiology and Microbiology of the Academy of Medical Sciences of the U.S.S.R., International Congress Series No. 59**, Amsterdam, Excerpta Medica Foundation, 1962.

DeGraff, A. C., and Creger, W. P., eds.: **Annual Review of Medicine**, vol. 15, Palo Alto, Calif., Annual Reviews, Inc., 1964.

DeWeese, D. D.: **Text Book of Otolaryngology**, 2nd ed., St. Louis, C. V. Mosby Co., 1964.

Lyman, F. L.: **Phenylketonuria**, Springfield, Ill., Charles C. Thomas, 1963.

MacKay, R. P., et al. eds: **Year Book of Neurology, Psychiatry and Neurosurgery**, Chicago, Year Book Medical Publishers, 1964.

Marquis-Who's Who: **Who's Who in America**, vol. 33, Chicago, A. N. Marquis Co., 1964.

Nash, E. M.: **Marriage Counseling in Medical Practice**, Chapel Hill, N. C., University of North Carolina Press, 1964.

Ruffner, F. G., Jr., et al., eds.: **Encyclopedia of Associations, vol. 1, National Organizations of the United States**, 4th ed., Detroit, Mich., Gale Research Co., 1964.

Wasserburger, R. H.: **Normal and Abnormal Unipolar Electrocardiogram in Infants and Children, Baltimore**, The Williams and Wilkins Co., 1963.

White, P. D., and Mitchell, C.: **Fitness for the Whole Family**, Garden City, N. Y., Doubleday & Co., Inc., 1964.

Book Notes

Books reviewed in this column are available from the TMA library.

Handbook of Psychiatric Treatment in Medical Practice. Nathan S. Kline, M.D., and Heinz Lehmann, M.D. 124 pp. Philadelphia, W. B. Saunders Co., 1962. $3.50.

THIS BOOK is a very readable handbook, giving practical common sense suggestions about treatment of psychiatric patients. It cuts through much of the vagueness that seems to enshroud psychiatry and attempts to establish practical rules of diagnosis, treatment, and management that can be applied by the general practitioner. It encourages the physician to get more involved with psychiatric problems and points out that what he does may be a great deal of help to the patient either on a short or long term basis. It is designed for ready and quick reference with adequate indexing and succinct statements as to the type of patients to treat, prognosis, what type of patient not to treat, and how to make referrals. All in all, this is a worthwhile handbook and it is succinct, pertinent, easy to read, well indexed, and not too bulky.—Richard J. Alexander, M.D., Austin.

Recent Deaths

DR. T. S. EDWARDS

Dr. Thomas Sydney Edwards, 87, Knox City physician for 50 years, died there April 24 after a lengthy illness.

Former president of the Baylor-Knox-Haskell Counties Medical Society, he was elected to honorary membership in Texas Medical Association in 1961.

Born in Navasota on May 2, 1875, Dr. Edwards attended the schools in Blanco where he and his family moved when he was a small boy. He was graduated in 1910 from the University of Texas Medical Branch and went to Knox City in September of that year.

He established the first hospital in Knox County in 1912. He supervised the building of Knox County Hospital in 1925, a wing of which was dedicated in his honor in 1949. His life-size portrait hangs in the hospital. He served as Santa Fe Railroad physician for 40 years.

Dr. and Mrs. Edwards celebrated their Golden Wedding anniversary two years ago. She was formerly Alma Rogers, and the couple were married Nov. 14, 1912, in Knox City.

DR. T. S. EDWARDS

He was a member of the Methodist church and had received his 50-year Masonic pin only two weeks before his death.

During his career, Dr. Edwards was estimated to have delivered more than 2,700 babies and performed more than 2,800 appendectomies and other operations.

Survivors include his wife; two sons, Kenneth T. Edwards of Brownwood and Capt. Tom R. Edwards of Fort Sheridan, Ill.; three daughters, Mrs. Tom Hampton of Dallas, Mrs. Charles Turner of Lexington, Va., and Mrs. Fred Dallas of Covington, La.; three brothers, Dr. R. C. Edwards of Knox City, Dr. Hemphill Edwards of Blanco and Sam Edwards of Orange, Calif.; one sister, Mrs. Beatrice Capp of Arizona; nine grandchildren, and two great-grandchildren.

DR. W. W. CARTER

Dr. William W. Carter of Corsicana, 85, died at his home April 13. He had been elected to honorary membership in Texas Medical Association in 1956 through the Navarro County Medical Society of which he was the oldest member and a former president.

He had practiced at Bazette and Powell before moving to Corsicana 41 years ago. A native of Mississippi, he was a graduate of the University of Tennessee College of Medicine. In 1956 he made a trip back to his alma mater to receive a 50-year merit award.

A former Navarro county health officer, he was former chairman of the official board of First Methodist Church, and he was a Mason, Shriner, member of the Knights of Pythias and Rotary Club, and an organizer and former director and vice president of Powell State Bank.

Survivors are his wife; three daughters, Mrs. R. L. Burnet, Vandenberg Air Force Base, Calif., Mrs. Will S. Price, Kerens, and Mrs. W. L. Holman, Corsicana; four grandchildren; and seven great-grandchildren.

DR. J. H. CURBY

Dr. John H. Curby of Maypearl died on March 30 at a Waxahachie hospital at the age of 86. He had practiced medicine for 63 years.

He was elected to honorary membership in Texas Medical Association in 1954 through the Ellis County Medical Society.

He was born in Kentucky on Nov. 20, 1877, the son of John Henry and Kathryn Curby. He attended schools in Grandview, Hillsboro, and Italy, Tex., and was graduated from Dallas Medical College in 1904. He interned at Johns Hopkins.

He practiced first in Bosque County for two years, then in Auburn for a year, and after that moved to Maypearl where he spent the rest of his life. He was mayor of Maypearl for three terms, a school trustee for two terms, director of Maypearl State Bank, elder of the Church of Christ, and a member of the Woodmen of the World.

He married Ida Mathews on Jan. 15, 1899, at Pluto, Tex.

Survivors include his wife and seven children, Lottie Doyle, Fort Worth; Ruth Kizziar, Pomona, Calif.; Loyd Curby, Waxahachie; Edwin Curby, Buena Park, Calif.; Marcus Curby, Kathryn Lowery, and Jack Curby, all of Houston.

DR. J. D. O'BRIEN

Dr. Justin Dennis O'Brien of Dallas died May 6. Born Oct. 5, 1896, in Dallas, he had spent his lifetime in that city.

He received his B.A. degree from the University of Dallas in 1917 and his M.A. degree there in 1918. He attended Baylor University College of Medicine and received his M.D. degree from Northwestern University, Evanston, Ill. He interned at St. Bernard's Hotel Dieu in Chicago and served his residency at St. Paul's Hospital in Dallas.

He was in general practice in Dallas from 1924 until his death.

Dr. O'Brien belonged to Dallas County Medical Society, Texas Medical Association, and American Medical Association. He was past president of the Dallas chapter of Northwestern University Alumni Association. He was a member of the Knights of Columbus and of Holy Trinity Catholic

DR. J. D. O'BRIEN

Church. He was team physician for the Jesuit High School for the last 15 years.

He married the former Alice Hickey of Boston in a Dallas ceremony on Jan. 23, 1930.

He is survived by his wife and two children, Neil J. O'Brien and Mrs. Robert M. Nicoud; two brothers, Judge Frank C. O'Brien and R. J. O'Brien; three sisters, Mrs. Homer T. Wilson, Mrs. Carl Vogel, and Mrs. Albert A. Faber, and six grandchildren; all of Dallas.

DR. L. E. HALE

Dr. Lee Edwyn Hale of Lubbock died April 20 of a rare collagen disease.

A native of Lorenzo, he was a lifelong resident of the South Plains area. His parents are Claude Lee and Clatie Frances (Morrison) Hale of Lubbock. Dr. Hale was born June 18, 1919, educated in the Lubbock public schools, and received a bachelor of arts degree from Texas Technological College in 1940. His medical training was done at Tulane University School of Medicine and he interned at Southern Pacific General Hospital in San Francisco.

During World War II he served as a captain in the Army.

He had practiced medicine in Lubbock since 1946.

DR. L. E. HALE

DR. J. W. FLEMING, JR.

Medical organizations to which he belonged were the American Medical Association, Texas Medical Association, and Lubbock-Crosby Counties Medical Society. He was a member of Phi Chi fraternity.

A Methodist, his hobbies were books and music.

Dr. Hale was married in 1944 to the former Helen Marie Johnson of Ogden, Utah. She survives as do two daughters, Claudia Lee Hale and Cara Lyn Hale, a son, Chris Johnson Hale, and his parents.

DR. J. W. FLEMING, JR.

Dr. John Welborne Fleming, 79, of Kilgore died March 18 from a heart attack. He was elected to inactive membership in Texas Medical Association in 1961.

He had practiced medicine in Gladewater during the 1930's. In World War II he practiced obstetrics in Mobile, Ala., because of a shortage of physicians there. Following this, he and Mrs. Fleming moved to Kilgore where they have lived for the past 20 years.

He was born at Grove Hill, Ala., June 1, 1884, and attended Jackson Agricultural College at Jackson, Ala. He received his M.D. degree from the University of Alabama Medical Department, Mobile, Ala., in June, 1908. He interned at St. Margaret's Hospital in Montgomery, Ala.

Before coming to Texas he had practiced in Arkansas and Alabama.

He belonged to the Gregg County Medical Society and had served as president in 1933.

Survivors include his wife, Iva; two daughters, Mrs. William Sedberry, Marshall, and Mrs. B. D. Beasley, Houston; one sister, Mrs. B. L. Page, Ala.; and four grandchildren.

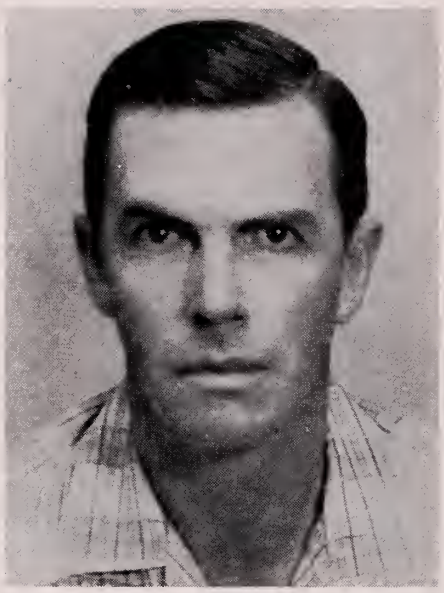
DR. L. P. NOELL, JR.

Dr. Livingston Pope Noell, Jr., of Harlingen was killed in a light plane crash near the Mexican town of Coatzacoalcos on March 29.

Captured by the Japanese on Bataan during World War II, he survived the Bataan Death March and remained a prisoner of war for three years.

After the war, he became a member of the Air Force reserves and entered private practice in Kingsville. He returned to active duty at the outbreak of the Korean conflict and later retired as lieutenant colonel in 1955. At the time he retired he was flight surgeon at the Harlingen Air Force Base. He remained in Harlingen and entered private practice.

Dr. Noell was born April 25, 1916, in Bowling Green, Ky., the son of L. P. and Carolyn G. (Smith) Noell. He attended Nashville, Tenn., schools and received a bachelor of arts degree from Vanderbilt University. In 1939 he received his medical



DR. L. P. NOELL, JR.

degree from Vanderbilt University School of Medicine. He interned at the U. S. Marine Hospital in New Orleans and entered the U. S. Army Medical Corps in 1940.

He married Clara Mae Bickford of Tivoli, Tex., on April 21, 1945, in Chicago. They had met on Bataan where she was an Army nurse.

He was a member of the American Medical Association, Texas Medical Association, Cameron-Willacy Counties Medical Society, American Academy of General Practice, and the staffs of Valley Baptist Hospital, Harlingen, and Dolly Vinsant Memorial Hospital, San Benito. While in Kingsville, he was a member of the Kleberg-Kenedy Counties Medical Society and served as president in 1949.

His hobbies were fishing, hunting, and golf.

Dr. Noell was buried with military honors at Fort Sam Houston National Cemetery.

He is survived by his wife; three sons, Pope III, Steve, and Peter; a daughter, Cindy, all of Harlingen; his mother of Newport, Ark.; a sister, Mrs. J. P. Wyant of Harlingen, and a brother, Graham Noell of Lexington, Ky.

DR. L. E. HARTMAN

Dr. Lee Edward Hartman, a psychiatrist, died unexpectedly at his home in Houston on Feb. 10. In private practice, he was also consulting psychiatrist for the Veterans Administration Hospital in Houston and formerly was consulting psychiatrist to the Texas Department of Corrections and an instructor in psychiatry at the Baylor University College of Medicine.

Forty-seven years old at the time of his death, Dr. Hartman was a native of Greenville, Miss., born on Nov. 30, 1916, to Marcellus and Marie (Feiss) Hartman. He was educated in Louisiana primarily, where after some early schooling in Greenville he entered the public schools in New Orleans. He earned a bachelor of science degree at the Louisiana State University in Baton Rouge, La., in 1938 and returned to New Orleans to get his medical degree in 1941 from the Louisiana State University School of Medicine.

He interned at the North Louisiana Sanitarium in Shreveport, La. Following this, he served with the Louisiana State Department of Public Health in Tallulah until he joined the U. S. Army Air Force in November, 1942. A flight surgeon, he held the rank of captain. After the war, he entered general practice in Buras, La., Newton, Tex., and Vidor, Tex., before moving to Beaumont in 1948. He practiced general medicine in Beaumont from 1948 until 1957 at which time he entered a residency in psychiatry at Baylor University College of Medicine.

He was a member of the Harris County Medical Society, Texas Medical Association,

DR. L. E. HARTMAN

American Psychiatric Association, and American Academy of General Practice.

He is survived by his wife, the former Nona Cook of Jamestown, La., and three children, Sarah Hartman, Lee Edward Hartman, Jr., and Marie Hartman. A brother, Dr. Sam F. Hartman of Beaumont, also survives.

DR. J. M. RAPE

Dr. James Marvin Rape, a past president of the Tom Green Eight Counties Medical Society, died of a heart attack on March 17. The San Angelo physician and surgeon was 73 years old.

Born in 1891 in Pittsburg, Tex., his parents were David Noah and Elizabeth Lester Rape. He received his preliminary education in Sulphur Springs. After attending East Texas Normal College (now East Texas State College) in Commerce and Baylor University, he taught school, and from 1918 to 1919 he served as a private in the U. S. Army infantry.

He married Mary Ethel Halbert in Greenville in 1918.

He attended the University of Arkansas and received his M.D. degree from the University of Tennessee College of Medicine in Memphis in 1927 and served his internship at St. Paul Hospital in Dallas. During his career, he attended postgraduate courses in New Orleans and in New York.

After practicing in Rankin and Iraan, Tex., in 1935 he moved to San Angelo where he was medical director of Western Reserve Life Insurance Company and a member of Shannon Hospital Medical Board.

A 32nd degree Scottish-Rite Mason, Dr. Rape was also a member of the Businessmen's Bible Class and the First Methodist Church. He had served as president to the Tom Green Eight Counties Medical Society in 1944, the year before he was secretary. He was also a member of the Texas Medical Association and the American Medical Association.

Survivors include his wife; a sister, Mrs. Maggie Miller of Fort Worth; and three nephews, Dr. Marvin Greer Rape of Houston, Dr. Joe Glenn Rape of Arlington, and Morris Day of Fort Worth.

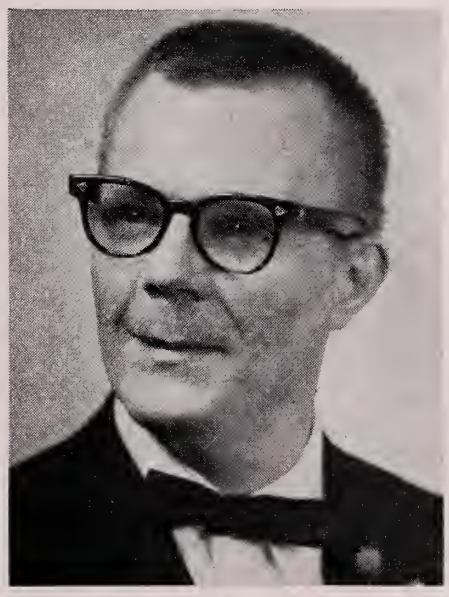
DR. T. W. BISLAND, JR.

Dr. Theodore Wensel Bisland, Jr., of Dallas died there March 1 of a gunshot wound. He was 39.

An ophthalmologist, he was born in Memphis, Tenn., Oct. 25, 1924, the son of Theodore and Frances Bisland. He held a bachelor of science degree in engineering from Yale, an M.D. degree from Harvard Medical School, and a bachelor of law degree from Southern Methodist University.

Able to speak both Spanish and Portuguese, he interned in Sao Paulo, Brazil, at Hospital das Clinicas. His residency was

DR. J. M. RAPE



DR. T. W. BISLAND, JR.

served in ophthalmology at District of Columbia General Hospital.

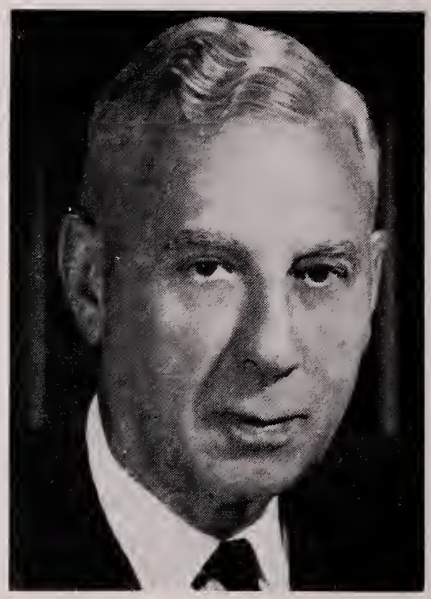
He served in the U. S. Air Force from 1952 to 1955 and was chief of ophthalmology at Carswell Air Force Base, Fort Worth. He practiced ophthalmology in Dallas from 1955 until the time of his death.

He married the former Diane Smith in Dallas on Dec. 30, 1960. They have two children, Kurt and Mel.

He was a member of the American Medical Association, Texas Medical Association, and Dallas County Medical Society.

Survivors are his wife, two children, and his parents who live in Memphis, Tenn.

DR. A. C. BRODERS, SR.



DR. A. C. BRODERS, SR.

Dr. Albert Compton Broders, Sr., of Temple died in a hospital there March 26 after a long illness. Seventy-eight years old, he had been in failing health since a stroke last August.

A world renowned surgical pathologist who served for many years on the staff of Mayo Clinic, Dr. Broders in 1924 originated the numerical system for the grading of cancer which is still used by hospital laboratories all over the world.

He came to Texas in 1951 after retiring as chairman of the department of surgical pathology of the Mayo Clinic, a position held since 1936. He then became senior consultant in surgical pathology at the Scott and White Clinic, Temple, remaining in this position until 1961. A memorial fund for the training of young physicians has now been established at Scott and White in memory of Dr. Broders.

Born Aug. 8, 1885, in Fairfax County, Va., the son of John and Virginia (Woodyard) Broders, he was graduated from the Potomac Academy, Alexandria, Va. He entered the Medical College of Virginia and received his medical degree in 1910. After two years of graduate study in Richmond he entered the Mayo Clinic in 1912 and was associated with the department of surgical pathology. He was certified by the American Board of Pathology in 1937.

He wrote extensively in the field of cancer research.

In addition to the master of science degree in surgical pathology from the University of Minnesota, Dr. Broders was awarded the honorary doctor of science degree from the Medical College of Virginia in 1929 and the honorary doctor of science degree from Washington and Lee University in 1949. He was a member of the Minnesota, Virginia, and Texas Medical Associations, Bell County Medical Society, the Southern Medical Association, the American Medical Association, the American Society of Clinical Pathologists, Sigma Xi, Alpha Omega Alpha, the American College of Physicians, as well as holding honorary membership in medical societies in Chile and Argentina.

He was elected to honorary membership in TMA in 1961.

In 1957 at the request of the Governor of Virginia, he returned to that state's 350th anniversary celebration where he was honored as a distinguished native son.

Dr. Broders was married Sept. 8, 1915, to Miss Adlene Zimmerman of Buena Vista, Va.

His hobbies were hunting, fishing, and gardening.

In addition to his wife, Dr. Broders is survived by two sons, Dr. A. C. Broders, Jr., and Dr. C. W. Broders, both of Temple; one daughter, Mrs. Armour T. Beckstrand of Rockford, Ill.; and eight grandchildren.

DR. R. T. TRAVIS

Dr. Roland Tony Travis, 73, died April 17 in the Jacksonville hospital, named Nan Travis Memorial in memory of his mother, where he had worked for many years. A victim of emphysema, he had been ill for a long time.

DR. R. T. TRAVIS

DR. A PRONIN

Dr. Travis came to Jacksonville in 1920 to join his brother, Dr. J. M. Travis, in practice. The pair formed the Travis Clinic.

A native of Van Zandt County, Dr. Travis was a graduate of Baylor University College of Medicine. He was an Episcopalian, a Mason, and a veteran of World War I.

He had served for many years as a member of the board of the Texas Bank and Trust Company.

He belonged to the American Medical Association, Texas Medical Association, and Cherokee County Medical Society of which he was a former president. He was a fellow of the American College of Surgeons.

Since his retirement two years ago, he had worked for the establishment of a pulmonary research and treatment center within the Nan Travis hospital. He had also spent three winters in Arizona devoting time to the treatment of emphysema.

He is survived by his wife, Mrs. Gladys Travis; a daughter, Mrs. Frank Brinkman of Tyler; three brothers, Dr. Travis of Jacksonville, Albert Travis of Lake Jackson, and Charles Travis of Wills Point.

DR. A. PRONIN

Dr. Arthur Pronin, 53, a general practitioner in Houston for the past 12 years, died April 14 in a Galveston hospital.

He was born in New York, attended school and college there, and studied medicine at the University of Zurich, Switzerland, graduating in 1937. He spent a two-year rotating internship at Perth Amboy, New Jersey General Hospital. Before coming to Houston he practiced for 13 years in Plentywood, Mont.

He was a member of the American Medical Association, Southern Medical Association, Texas Medical Association, and the Harris County Medical Society.

Dr. Pronin was a member of the Temple Emanu El and the B'nai B'rith Lodge.

Survivors are his wife, Mrs. Evelyn Pronin; a son Leslie Pronin, also of Houston; and a sister, Mrs. Maury Golob, Jamaica, N. Y.

DR. P. R. STALNAKER

Dr. Paul R. Stalnaker, the son of a pioneer Texas physician, died April 22 in Houston. A native of Austin, Dr. Stalnaker went to Houston in 1931 and was active in the practice of urology until his hip was broken in a fall a year and a half ago. He had been hospitalized ever since. The 84-year-old physician was elected to honorary membership in Texas Medical Association in 1953.

Dr. Stalnaker was a member of the Sons of the Republic of Texas and was past president of the San Jacinto chapter. Last year he was made a Knight of San Jacinto. He was also a former president of the Paul Carrington chapter of the Sons of the American Revolution.

He was active in the preservation of material of historical value. His father's auto-

graphed copy of the official Confederate book of instruction on surgery and first aid that the elder doctor assisted in compiling, "A Manual of Military Surgery," published in 1863, was presented to the Texas Medical Association Memorial Library on the Association's centennial anniversary in 1953. In honor of the centennial anniversary of his father's graduation from the Medical Department of the University of Pennsylvania, Dr. Stalnaker assembled an exhibit on autopsies for display at the TMA Annual Session in 1955. This subsequently was exhibited at the AMA convention. His father, Dr. John William Stalnaker, served as assistant surgeon in the Confederate Army from Fort Sumter to Appomattox and moved to Austin after the war. He was the first physician, his son believed, to request an autopsy performed on his body.

The younger Dr. Stalnaker attended schools in Austin and received his medical degree from the University of Texas Medical Branch in 1904. Following his junior year, he was allowed to take the state board examination for his license to practice medicine. He had made the highest grade and had won a prize—a chance to be doctor on a ship which carried cattle to British possessions in Africa. While on this trip, he witnessed what he believed to be the first mass inoculation against typhoid fever—soldiers being immunized as they returned from the Boer War.

While in medical school, Dr. Stalnaker was asked to organize Zeta chapter of Phi Chi medical fraternity in Galveston which he did in 1903 with seven charter members. He was a trustee of the fraternity for more than 20 years.

He was commissioned in the Navy in 1905 after having previously served in the Spanish-American war, and at one time served as head of the Naval hospital on Guam. He also was in the Veracruz expedition in 1914 and served in both World Wars.

Dr. Stalnaker was a former urology teacher at the University of Pennsylvania and the University of California, and professor emeritus in urology at Baylor University College of Medicine. He was also a member of the Salvation Army advisory board.

Survivors include his wife, Mrs. Florence Stalnaker; two daughters, Mrs. Mary S. Bock and Mrs. Emily S. Doggett; eight grandchildren; and seven great-grandchildren.

DR. E. P. WALLER

Dr. Edward Pendleton Waller died May 20 in San Antonio of carcinoma of the lung. He had practiced medicine since 1943, first in Lampasas, then in Luling, and since 1952 in San Antonio.

A plastic surgeon, he was a native of Fentress, Tex., born May 21, 1915, to Maurice and Betty (Pittman) Waller. He attended public schools at Fentress and Prairie Lea and received a B.A. degree from Southwest Texas State College. In 1941 he was graduated from Baylor University College of Medicine. The following year he interned at Robert B. Green Memorial Hospital in San Antonio. He spent a second year there in residency and left for Rochester, Minn., for a two-year residency at the Mayo Clinic. He returned to the University of Texas Medical Branch in 1951-1952 for postgraduate work in plastic surgery.

Dr. Waller was a member of Bexar County Medical Society, Texas Medical Association, American Medical Association, the Texas Society of Plastic Surgeons, and the International Medical Assembly of Southwest Texas.

He served as a steward and trustee on the official board of St. Andrew's Methodist Church in San Antonio.

He married Robbie Robinson of Prairie Lea on Aug. 1, 1941.

Survivors include his wife and three children, E. P. Waller, Betty Waller, and Patsy Waller of San Antonio, and his parents of Fentress.

DR. E. P. WALLER

COMING MEETINGS

American Medical Association, Clinical Meeting, Miami Beach, Fla., Nov. 29-Dec. 2, 1964. Annual Meeting, New York City, June 20-24, 1965. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

Current Meetings

JULY

Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1964. Dr. Sanford Glanz, 2425 Morgan, Corpus Christi, Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 16-18, 1964. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

AUGUST

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, San Francisco, April 12-15, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, New York, Oct. 24-29, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, New York City, June 17-21, 1965. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, Los Angeles, Oct. 8-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Oct. 2-3, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Dermatological Association. Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Sec.

American Gastrocnterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society, New York, May, 1965. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Medical Association Congress on Occupational Health, Houston, Sept. 26-27, 1964. Dr. Henry F. Howe, 535 N. Dearborn St., Chicago, Sec.

American Medical Women's Association, New York, Oct. 16-20, 1964. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Neurological Association. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., New Orleans, La., Oct. 8-10, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Chicago, Nov. 6, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons, Mexico City, Oct. 6-11, 1966. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico, Phoenix, Ariz., Dec. 9-12, 1964. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwest Regional Cancer Conference, Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Waco, Nov. 8, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. Leonard R. Robbins, 3618 Yoakum, Houston, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, San Antonio, September, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. Charles Peterson, 1333 Moursund, Houston, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Meltou, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

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Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section, San Antonio, April 29-May 2, 1965. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc., Houston. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Surgical Society, San Antonio, Oct. 4-6, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauliue K. Mathis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, Sec.

United States-Mexico Border Public Health Association, Los Angeles, April 26-29, 1965. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jenseu, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulaue Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

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Nursing Homes, Not Patients, Should Be Classified

DURING the past year, many facets of nursing home business came to the attention of the Committee on Nursing Home Accreditation. One inequality is the manner by which the welfare program bases the amount paid to the nursing home, regardless of the skill of the nursing care supplied to the individual patient.

Under present regulations, the nursing home receives more to care for a bedfast patient than for an ambulatory or semi-ambulatory patient. Thus, many elderly patients could be, and are, confined to their beds when, with proper nursing care, physiotherapy, and occupational therapy, they might become semi-ambulatory or even ambulatory. However, the nursing home with the facilities to improve its patients in this manner then must take a reduced amount for the care of those patients. Excellence of care in this situation is penalized rather than rewarded.

Mr. C. B. Francis, Austin, president of the Texas Nursing Home Association, believes that proper care cannot be accomplished under the present system of charges. A more equitable solution would be to classify nursing homes as to the quality of their care rather than to classify the patient as custodial, ambulatory, semi-ambulatory, or totally bedfast, and to increase the reimbursement to those homes which give excellent care, including all ancillary services. The homes which give less specialized care in the fields of occupational therapy, physiotherapy, recreational facilities, and so forth, should receive less per patient in welfare payments. The home would then be paid the same amount for the welfare patient's care, regardless of his physical status, making it an inducement for the nursing home to improve the health of the patient as much as possible.

The House of Delegates of the Texas Medical Association, at the 1964 meeting, passed a resolution imploring the Texas Legislature to increase

funds available to the welfare department so that the existing rules for accreditation could be enforced. With an increase in funds, additional investigators could be placed in the field to actually assess the quality of care given in each nursing home while the home was being checked for accreditation.

Further, all committees dealing with the aged should study this problem thoroughly, so that a final acceptable program could be designed as speedily as possible. This is not to be misunderstood as a reflection on our welfare agency or any state department, for they must function under their present regulations. It is only a suggestion that nursing homes which use all means to improve the physical and emotional status of their patients should be paid accordingly. Excellent patient care should be the purpose of all nursing home facilities.

—CHARLES B. DRYDEN, JR., M.D.,
Wichita Falls, Chairman,
Committee on Nursing Home
Accreditation.

Preseason Conditioning For Young Athletes

IN THE NOT too distant past, high school and college athletes suited up for practice at the beginning of the school term and started "butting heads" in their first athletic drills. This procedure has given way, after intensive studies in various parts of the country, to preseason physical conditioning programs as well as noncontact conditioning at the time that school sessions begin, before actual participation in athletic contests involving bodily contact.

Basically, preseason conditioning can be broken down into four major components: nutrition, muscle training, reduction of fatigue onset, and heat tolerance.

During the summer months, parents are apt to be too inattentive to the nutritional requirements of their teenagers and young adults. A high protein diet with moderate amounts of fat and starch should be utilized, as well as a high vitamin intake. At any rate, a good all-around diet should be tantamount to any kind of strenuous physical exercise. In addition, a physical checkup should be done, particularly if the youngster has not had one prior to beginning strenuous physical exertion. It does no harm to have this repeated at the time he enters school.

Probably the three main factors involved in preseason conditioning are muscle training, which includes both muscle building and coordination, fatigue tolerance increase, and in this part of the country, tolerance to heat. In the athletic department of the local university with which I am associated, we initiated several years ago a program of wind sprints, weight lifting, and isometric contractions, working gradually up to maximum output. Getting youngsters to participate in handball is one of the best ways to increase coordination through eye and bodily movements and it also enhances their ability and increases their tolerance to withstand fatigue. I've also suggested to high school coaches that when school is out in the spring they should anticipate which boys will continue or enter the athletic program and give them some basic instruction in the wind sprints and in muscle building exercises. The old "Randolph shuffle" is still one of the best exercises to build coordination and at the same time increase muscular endurance and work performance. Usually none of these preseason physical conditioning programs can be supervised, so it is advisable to explain to the youngster the importance of being in good physical condition when he reports to school to start training for the coming season.

In the past several years there have been some unfortunate instances of heat exhaustion and even death occurring at the first of the season when heat is intense. This is particularly noticeable when the humidity is high. Heat exhaustion and heat stroke can be brought on rapidly. It has been our experience that a period of rest every 45 minutes lasting 10 to 15 minutes, particularly during the first two weeks of football training, has worked out well. We have had no unusual occurrence of heat exhaustion. At the rest period, each individual is *required* to drink a certain amount of water in which electrolytes are added.

There are always a few boys who will carry out a program such as this without supervision because of their intense desire to excel at their particular sport. However, the ones on the opposite end of the line think that they can go directly from a leisurely existence into strenuous physical exercise without deleterious effects. Fortunately, the

physical resiliency of some young people allows them to get by doing this. However, it is taxing to their cardiopulmonary systems to suddenly be thrown into violent exercises without some form of conditioning beforehand.

As suggested, particularly in the high school levels, it is important for physicians to instruct coaches in their localities to furnish some brief written instructions regarding preseason physical conditioning in muscle training, pulmonary-ventilatory increase, and heat tolerance.

There is nothing so gratifying as to see a youngster develop into a truly fine athlete, but at the same time, a program for his nutrition, body physiology, and protection from potential injury should be carried out to the best ability of the coaching staffs and their medical supervisors.

—WILLIAM S. HORN, JR., M.D., Fort Worth.

Are You Guilty?

"DOCTOR, if you put me in the hospital to do those tests my insurance will pay for it." How often each day do you, a practicing physician, hear this question? What is your ethical responsibility in handling this request? In what ways do you as an individual contribute to rising insurance premiums for all your patients, and to the overutilization of beds in your community hospital by acquiescing to the demands for unnecessary diagnostic hospitalization as an apparent economic convenience? Each practicing physician has the moral obligation to reevaluate his position on these and related questions.

If government financing of medical care for any age group, except on the basis of need, is to be prevented, then we physicians must strengthen the position of voluntary health insurance within the existing framework of our system of medicine. It is up to us to educate our own ranks in regard to the place and use of hospital insurance. Then, we may be in a position to help the patient understand the principles which mold a strong voluntary health insurance industry, and to explain why the soundness of this industry directly benefits him.

When faced with a patient intent on using —or rather misusing—his hospitalization insurance, the doctor's position is a delicate one. In most cases, the patient is either misinformed or uninformed on the principles of the health insurance philosophy. This may be the fault of the insurance salesman, of the language of the policy itself, or the inability of the physician to face the problem and meet the issue in a frank discussion with the patient.

It is surprisingly easy to convince the patient that his hospitalization insurance is an investment against catastrophe just as is true of his automobile liability insurance. It is a protection against the high cost of the treatment of an illness requiring hospitalization. It is not meant to pay the cost of preventive medicine any more than an automobile accident policy should pay for a routine motor tune-up with adjustments.

When the time for adequate discussion is taken, few patients fail to understand that benefits arise from the money they and others pay into the company periodically in the form of premiums, and that each time a policy is used unnecessarily the price of the premium must be raised because the benefits are not paid from moneys generated by the company, but from the accrued assets of the combined policyholders. This is a simple economic fact. Most people are aware of the apropos analogy of fire and accident insurance rates which are fixed on the basis of the incidence of fires and accidents in the locale.

Unnecessary hospitalization for the purpose of reaping the insurance policy benefits is contributing, in no small fashion, to the increasing shortage of hospital beds. And so, the vicious cycle begins. The citizen is called upon by public subscription or by local taxation to provide more beds at an ever-increasing cost. These increased numbers of beds cost him money in several ways. The increased utilization of those beds for diagnostic studies which legitimately should be done outside the hospital increases the premiums which he must pay for his hospital insurance.

When the private health insurance plans are forced to price themselves out of the reach of the average citizen, and when local communities can no longer afford to provide sufficient hospital beds to handle their needs, the consequence is obvious. The government will rush in and the vacuum will be filled by

a national compulsory health insurance program, owned and operated under the direct supervision of the federal government.

You, the physician, are the key figure in the maintenance of a healthy voluntary health insurance program. Other important facets of the problem are improvement in the coverage of the policies themselves, improvement of insurance plans designed to meet the local needs, and increased integrity in health insurance salesmanship. But in the final analysis, it is up to you, the physician, to say, when it is applicable, "I'm sorry, but I do not believe that I can honestly warrant hospitalization for these studies. Let me explain to you why this is the case."

—Tom W. Duke, M.D., Amarillo.

Problems of Management And Health Insurance

After WORLD WAR II, the fiscal policy of the federal government, the rise in the graduated federal income tax, and the social security tax resulted in inflation of the American dollar and a diminution in the pay, in effect, of the labor force. At approximately the same time, the growth of health insurance supplied labor a method of increasing pay, in effect, and of meeting expenses for health care without further loss of pay through income tax. As labor demanded more fringe benefits and more pay, management also faced the same taxes and inflation. For the sake of industrial peace, however, so many of labor's demands in health insurance were granted that it has become a difficult expense to meet.

At the same time the increased cost of health care, with the exception of physicians' fees, added to this problem. In the last several years, physicians' fees began to catch up with inflation. The result has been that management and the health insurance industry have begun a search for a method to reduce the cost of health care to their respective industries.

As with every new industry, many mistakes have been made. Health insurance policies have been written in such a manner that they cover many expenses other than those incurred for relatively serious illnesses.

The patient, faced with expensive diagnostic tests, has demanded and received hospitalization because his insurance would not pay benefits for tests performed elsewhere. In addition, much minor surgery is now done in the hospital because the doctor knows it is safer and because the patient demands it to collect his insurance. Outpatient procedures done inside a hospital are at least 50 percent more expensive; in addition, accession to such demands promotes the institutional practice of medicine.

Since the charges for hospitals, nursing care, drugs, and allied services are relatively fixed, the financial troubles of insurance companies and of management are often blamed on practicing physicians. Familiar by now are the claims that insured patients are hospitalized unnecessarily and are charged higher fees. This may be true in a few cases, but such abuses are being stopped by the medical profession itself.

Management, insurance, and the medical profession must realize that policyholders must be educated as to the purposes and uses of health insurance. Most medical organizations already are encouraging their members to work toward this goal.

It is not feasible to reduce benefits already given; however, future policies might be written so that the portion of costs paid by the beneficiary would discourage his remaining in the hospital longer than necessary. Another approach might be to allow appropriate insurance benefits for procedures that can be done as well (and far less expensively) in the doctor's office as in the hospital.

Although many committees, both in management and the insurance industry, seek to solve their problems by proposing further regulations in regard to physicians' services to insured patients, such actions imposed on all would not correct the abuses of a few. In fact, control by any outside group would result only in deterioration of the quality of medical care. Incidentally, claims to the effect that "if you don't let us do this, or that, the federal government will take you over" seem baseless and irrational.

The medical profession is willing to help solve this problem, for it is well aware that vast benefits to patients and doctors alike have come about through the public's acceptance and use of health insurance. But let us not accept all of the blame. Medical care has evolved for centuries to reach its

present degree of quality. Likewise, it will take the infant health insurance industry a reasonable length of time to develop its services to the satisfaction of the majority of those concerned. Our profession realizes the desirability of attaining this goal and will cooperate to its fullest capacity, but it is not willing to sacrifice its freedom.

—WILLIAM M. SHERRILL, M.D., Houston.

Local Help for Future M. D.s Aids Student, Medicine, Public

SHOULD LOCAL medical societies finance medical scholarships for young people of their own communities? Many doctors, looking back on the hardships and sacrifices they made to get a medical education, are prone to say, "Let them get it the hard way, as I did. It will do them good. If a man wants a medical degree he should be willing to work for it."

Quite right, for us. We probably are better doctors for having had to make the grade the hard way.

That was yesterday. If we may indulge in a masterpiece of understatement, things are different now.

Medicine formerly attracted its full share of the best scientifically oriented minds. Medicine formerly was one of relatively few outlets for such minds. That is no longer so. There are many new sciences, and many aspects of the venerated sciences—so many outlets for the brainy young man or woman! They are shopping around, and they find that science in industry, research, government, and other areas bids high for their talents. Industry, for example, is only too eager to snatch the top-ranking student from high school and carry him all the way through college—to work in that particular industry's field. The young man or woman who may lean toward, or even prefer, medicine but who is without funds for the expensive trek toward the M.D. degree, will be lured to another science. So, another good doctor is lost, and with the growing complexity of medicine, the profession simply cannot afford any but the best.

AMA-ERF, of course, came to the rescue at a time when the seesaw was tilting against us, and is helping to reverse the trend.

Is it enough? We in Nueces County think not. Nueces County Medical Society is presently sponsoring two students from our county to the extent of $1,000 a year for their studies in Texas medical schools. Annually, each member is assessed $5 per student to support this program. An additional scholarship fund of $5,400 was established from surplus funds from our successful county-wide Sabin polio immunization drive in 1962. Two awards of $500 each, and one of $400, have been made this year.

In at least one instance, and perhaps in all, these scholarships are being used by young men who could not have afforded medical school without assistance. Nor is it likely that they would have received AMA-ERF funds, since the competition and demands are so great.

The local society sponsoring medical education in its own community not only encourages local talent, it achieves a tremendous impact of community good will. It raises the prestige of local physicians, and helps to counteract the myth that doctors are remote and uncaring.

In a society of 200 members, $5 a year from each member will keep a student in medical school. That is the equivalent of two cartons of cigarettes. Is it too much to invest in the future of good medicine in your own home town?

—MEYER KURZNER, M.D., Corpus Christi, President, Nueces County Medical Society.

Preventing Head and Neck Injuries in Football

BETTER PROTECTIVE equipment for the football player has brought a decline in the number of serious head and neck injuries. The simple addition of the face guard has caused a 70 per cent decrease in face and mouth injuries. Since the use of a mouth or tooth guard is now mandatory, there should be a similar decrease in tooth and jaw injuries.

Recent studies, however, have uncovered information on several practices dangerous to the football player. Alley,[1] in a review and study of football injuries in California, found in patients with head injuries that

only 4.7 per cent had helmets considered properly fitted. This is astonishing, as is the report that the practice of "handing down" equipment is common. Equally astonishing is the finding that over 60 per cent of these injured had been taught to "spear," that is, to use the helmet as a ram, and that 72 per cent of the injured players had received less than nine days preseason conditioning.

It seems then that much beyond good equipment is needed to reduce injuries further.

Recommendations to those who coach and train football players are listed in a Report on the National Conference on Head Protection for Athletes:[2]

1. The technique of "spearing" is dangerous and should be outlawed.
2. Proper conditioning of the athlete, including specific exercises for the neck muscles, is a valuable protection.
3. The face guard should be fitted as close to the face as possible to afford maximum protection and to eliminate its use as a "lever."
4. A properly fitted, reliably built helmet can provide maximum protection.
5. Mouth guards will add additional protection against tooth and jaw injuries.

Equipment doubtless will continue to improve, but we must be concerned as much or more that coaching and training are proper and that the athletes are provided the best (and best-fitted) equipment available.

—DANIEL B. POWELL, JR., M.D., Austin.

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Air-Medics Take Lead In Education, Investigation

WITH the tremendous increase in air travel has come a vast responsibility for the physicians who are examiners for the Federal Aviation Agency. They must certify that pilots, control tower operators, dispatchers, and others who serve these flights are physically, mentally, and emotionally fit to carry out their duties.

Literally millions of people each year depend on the abilities of airlines personnel to provide safe travel, but other aspects of aviation have expanded, too, and are of vital concern to physicians. Problems arising from aerial crop-dusting, private flying, and space technology also fall within the realm of medicine.

Encompassing these areas, and ever broadening its interests, the Texas Air-Medics Association has become an educational group with much to offer. As a related organization, it meets in conjunction with the Texas Medical Association's Annual Session, and is composed of some 400 members from several states and from South America.

Its members, and visitors to its scientific meetings, can learn of the exceedingly high relationship between use of alcohol and fatal aviation accidents. (Of 158 such accidents in 1963, 56 were determined to be caused by pilots' use of alcohol as shown by positive blood and tissue alcohol findings in routine toxicological examinations.)

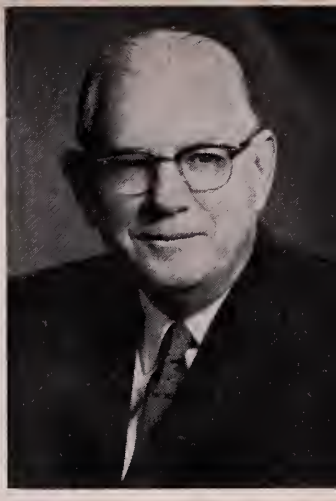
Physicians who have private pilots among their patients are interested in learning what physical standards these pilots should meet, and what other advice should be given regarding the physical effects of flying.

Nearly all doctors have reasons to be concerned about insecticides, especially when these chemicals are sprayed from planes over wide areas. Pilots sometimes become seriously ill after accidental poisoning; also, the question arises as to whether certain chemicals can contaminate fruits and vegetables. The effects of such insecticides on the human and animal life sometimes must be evaluated; and physicians involved in these studies also must be aware that drainage from sprayed areas can find its way into rivers and lakes to contaminate sea foods.

Still another area of interest to the Texas Air-Medics Association is the new and exciting field of space medicine, which provides growing numbers of scientific reports.

For the past 18 years, this organization has served Texas physicians who wish to inform themselves of the scientific aspects of aviation. Its officers, now planning the 1965 program, give assurance that as air science and medicine advance, so will the contribution of this group of physicians to their colleagues.

—WALTER B. ADAMS, M.D., Wichita Falls, President, Texas Air-Medics Association.



Cost of Medical Care

The cost of medical care has increased in 25 years in about the same proportion as the cost of all other services. This is the conclusion, as I interpret it, reached after a three-year study by the AMA Commission on the Cost of Medical Care and released at the annual meeting in June.

One major cause of increased costs in overall medical services is that hospital charges have risen 480 percent, for labor is higher, more people use hospitals now, and as compared with 25 years ago there are proportionately more medical than surgical cases treated in hospitals.

Another major cause of increased cost is the universal demand by doctor and patient alike for more laboratory tests, use of x-ray, and drugs. However, aside from hospital charges, medical costs have risen only 91 percent as compared to 123 percent for all other services.

The improved health, longer life expectancy, and virtual elimination of some diseases--all the result of medical advances and improved care--have greatly influenced the national economy. For example, Sabin oral vaccine cost $653,000,000 to develop, but it already has resulted in a saving of $6,700,000,000 in life, income, and medical care, plus the inestimable savings in warped lives and bodies. Also, fewer lives are claimed by tuberculosis, influenza, pneumonia, intestinal infections, and osteomyelitis--to name a few--and infant and maternal mortality rates have been greatly reduced.

Although treatment today is more adequate and effective, time required for hospitalization and convalescence is less, and the doctor's fee has not risen in proportion to other medical charges, the doctor still must share in the responsibility for the overall increase in medical care costs. He alone determines to what extent x-ray, laboratory work, therapy, and drugs are used, and these have been a large factor in the rising cost of medical care.

Mat E. Johnson

President
Texas Medical Association

Hemoptysis of Undetermined Etiology

RESEARCH & INVESTIGATION

THE CAUSE of hemoptysis is usually undetermined when the patient first consults the physician. To identify idiopathic bronchopulmonary bleeding, the method of investigation outlined in Table 1 must be pursued. Any source of blood other than from the lower respiratory tract must be excluded first. Hemoptysis of undetermined etiology is better understood and its prognosis more readily assessed through the experience gained by thorough follow-up of all cases.

This study has been conducted from the viewpoint of the endoscopist and his role in making the differential diagnosis. Including all the patients bronchoscoped by this writer in the past ten years, some 265 consecutive private cases have been investigated and evaluated. Endoscopy reports, office charts, and hospital charts have been studied. Referring physicians have been contacted and their records reviewed and finally, when necessary, the patients themselves have been written for any undisclosed information.

Method of Investigation

History.—A history of "spitting up blood" does not have to be accompanied by other symptoms. One apparently healthy young male without preceding symptoms suddenly had a profuse hemoptysis which subsequently was diagnosed as a moderately advanced tuberculosis. Cough is the most commonly related symptom, but others often encountered are dyspnea, expectoration, weight loss, fever, malaise, and chest pain. Each of these symptoms when correlated with the hemoptysis gives a clue to the cause. The smoking habit in carcinoma patients is an undeniable factor. Some errors are recognized readily in reviewing old records for symptoms after final diagnosis and follow-up are known. Historians too frequently record their impressions rather than the true facts in the patient's story. One young man's complaint of chest pain was recorded as functional by several clinicians. He eventually died of carcinomatosis with metastasis in the pleura shown at postmortem to be derived from a primary carcinoma in the pancreas. The amount of blood produced does not give a clue as to its cause.

TABLE 1.—*Method of Investigation.*

1. History
2. Physical examination
3. Chest x-ray
4. Sputum examinations
5. Bronchoscopy
6. Bronchial lavage for
   a. Acid-fast smear and culture
   b. Predominant organism
   c. Papanicolaou smear
7. Bronchography
8. Scalene node biopsy
9. Thoracotomy

Physical Examination.—Nasopharyngoscopy and mirror laryngoscopy, procedures peculiar to the otolaryngologist's examination, give him an advantage over other physicians in differentiating hemoptysis from epistaxis, hematemesis, and bleeding from the mouth. For this reason, many cases are referred to rule out bleeding from sources other than the bronchopulmonary tree. It is remarkable that patients and some physicians[3] can confuse hemoptysis and epistaxis, thinking blood from the nose can be aspirated and subsequently coughed. This is most unlikely and when it rarely occurs it is evident and easy for the otolaryngologist to

To determine the significance of hemoptysis 265 cases are studied.

CAM STIERNBERG, M.D.

observe. When there is no blood being produced at the time of examination it becomes difficult to ascertain its source. Having the patient cough before and while examining the larynx with a mirror will sometimes produce a little blood that can definitely be seen coming from the tracheobronchial tree. Esophageal bleeding is characterized by bright red blood, little cough, regurgitation, and no frothy blood-mixed sputum.

The case in which the patient gives a history of having a small quantity of blood appear with little or no cough or a little blood brought up with just a throat clearing offers a challenge to determine the source of bleeding. A number of individuals bleed from the gingiva around second and third molar teeth and particularly from around third molars that are unopposed by the corresponding lower or upper third molar. These areas are far back in the mouth and easy to neglect with the toothbrush, so consequential gingivitis with bleeding results. This blood is not detected by the patient until it reaches the larynx and stimulates throat clearing. The patient erroneously thinks it comes from the chest and sometimes misleads his physician. Inspection and massage of these third molar areas will sometimes disclose the presence of blood exuding from around the tooth. Adequate use of the toothbrush, massage, and mouth washes will usually clear these gum infections and stop intermittent production of blood that has been mistaken for hemoptysis.

X-ray.—A chest x-ray is always indicated. The various pulmonary radiological findings and their interpretation are not within the scope of this paper. The limitations of x-ray are known and easily demonstrated when films are subsequently compared with the more exact pathology determined at biopsy, thoracotomy, and postmortem. Roentgenograms are usually positive for some pathology by the time a carcinoma or tubercular patient expels blood. Bronchograms are necessitated by findings suggestive of bronchiectasis.

Sputum.—Sputum examinations for acid-fast bacilli are usually negative; however, this examination is an integral part of any complete work-up. Papanicolaou smears of the sputum and bronchial aspirations which are being done more frequently offer another adjunct to diagnosis. The physical aspects of the sputum give some clues because copious purulent sputum with some blood is suggestive of bronchiectasis.

Bronchoscopy.—Bronchoscopy is usually indicated in all cases of definite hemoptysis even though the chest x-ray is negative. One exception might be cases with proven tuberculosis. It is noted in older reported series of cases of hemoptysis and in series in which tuberculosis was a predominant cause that many patients were not bronchoscoped.[1] Included with the malignancy cases was one that was bronchoscoped for hemoptysis of undisclosed cause with accompanying symptoms of weight loss and cough plus x-ray findings of pleural thickening and/or fluid. Seven months later this patient was diagnosed through thoracotomy as having carcinoma. This illustrates the necessity of pursuing the cause of hemoptysis, especially when there are other findings. Another patient who evidenced no hemoptysis was dismissed with a diagnosis of bronchitis but subsequently evidenced malignancy 14 months later. The difficulty of pulmonary diagnosis suggests that all possible means of investigation must be understood and employed.

With such an undeniably vital role the endoscopist must look at the picture as a whole and not just through the 7 mm. lumen of the bronchoscope. The limitations of bronchoscopy are probably not as well understood by the average general practitioner or internist as they are by the otolaryngologist, yet endoscopy is an indispensable procedure. The expense and the discomfort are the most objectionable factors, but by careful preparations the latter can be minimized. No known complications arose from these 265 bronchoscopies other than one toxic Pontocaine reaction which was resolved with barbiturates in five to ten minutes and without further sequelae.

Bronchial Lavage.—Testing of bronchial washings for predominant organisms is done frequently, however this seldom offers any real help in diagnosis or treatment. The acid-fast smear and culture plus the Papanicolaou stain are more rewarding. Following bronchoscopy, scalene node biopsy and thoracotomy are often necessitated in cases where carcinoma is suspected, these latter procedures being done by surgeons.

Differential Diagnosis.—In order to understand the hemoptysis of undetermined etiology the cases where the etiology can be determined must be properly understood, thus a differential diagnosis must be sought and discussed. Besides such common causes as bronchogenic carcinoma, bronchiectasis, pneumonia, bronchitis, abscess, and tuberculosis, Pratt[7] reports some 40 additional unusual causes of hemoptysis. In Table 2, 223 of 265 cases in the present review are broken down into the various pulmonary diagnoses. Each disease is further separated into the number with hemoptysis, the number without hemoptysis, and the total cases of each different pulmonary diagnosis. The 42 cases not included in Table 2 were bronchoscoped for a variety of reasons, such as asthma, postoperative atelectasis, foreign body, cancerophobia, unexplained cough, and others.

It can readily be seen that carcinoma occupies the dominant role in considering hemoptysis and lung disease. Carcinoma is the feared disease that both patient and physician think of first. If carcinoma is the causative factor in hemoptysis, the earlier the diagnosis the better for some slight chance of cure. Only two patients of 52 were known to be alive five years after diagnosis was made; another four were lost to follow-up. If carcinoma is not present, the patient's anxiety may be alleviated by hearing this news or by a diagnosis of less serious nature. There were 52 cases of proven bronchogenic carcinoma of which 29 or 56 per cent did have hemoptysis. This percentage is about the same as is reported by several authors in the literature.[5] Hemoptysis is probably the most important symptom in carcinoma, but unfortunately it occurs late. The amount of blood expectorated was usually small. The frequency of chest pain in lung cancer is noticeable.

TABLE 2.—*Causes of Hemoptysis.*

Disease	No. with Hemoptysis	No. without Hemoptysis	Total
1. Carcinoma	29	23	52
2. Bronchitis	21	37	58
3. Bronchiectasis	12	20	32
4. Tuberculosis	8	8	16
5. Pneumonia, pneumonitis, and right middle lobe syndrome	8	11	19
6. Lung abscess	3	1	4
7. Empyema and pleuritis	3	5	8
8. Pulmonary infarction	3	0	3
9. Lung cyst	1	1	2
10. Histoplasmosis	1	0	1
11. Hemoptysis of undetermined origin	28	0	28
	117	106	223

Proven bronchiectasis was found in 32 of the patients, a larger number than expected. Twelve or 37 per cent spit up blood. Moersch[6] noted that the older bronchiectatics more often had hemoptysis, or in other words, the longer the disease is present the more likely it is to produce bleeding. The disease occurs more in the younger age groups in contrast to bronchogenic carcinoma which occurs more often in the older male who has smoked for many years. Boucot[2] found hemoptysis from all causes twice as common in smokers as in nonsmokers. Only 16 of the 265 patients bronchoscoped proved to be tuberculous, and of these, eight had hemoptysis.

It can be observed in Table 2 that 21 patients with hemoptysis were given a bronchoscopic diagnosis of bronchitis. Another 37 did not have hemoptysis, making a total of 58 cases of bronchitis. On endoscopy bronchitis was concluded if there were observable objective changes in the mucous membranes such as swelling and inflamma-

tion with or without excess secretions. The hemoptysis cases diagnosed as bronchitis and in which no other pulmonary pathology was diagnosed may be considered in much the same relatively benign category as the cases with unexplained hemoptysis. Nineteen patients of this group are still living two to ten years after their initial hemorrhage, although several still have symptoms of chronic bronchitis. Two were not traceable. One bronchial asthmatic has had two or three recurrences of hemoptysis, but is otherwise in satisfactory condition and able to work every day.

The opinion has been expressed that undiagnosed hemoptysis cases have a serious underlying pathology. However, if the investigation is thorough and the patient does not persist in coughing blood, material found in this study indicates that the physician should have reasonable confidence that the prognosis will be relatively good.

There were 28 cases of hemoptysis in which no bronchopulmonary disease was found. In Table 3 the age, the sex, and the number of years each patient of this group survived without developing major chest pathology is recorded plus a column for comments. These people had relatively normal x-rays, and except for those (15) with a cough, they had no other positive symptoms. It is noted that three of the group were reported as having emphysema, two with calcified hilar nodes, and one with enlarged hilar adenopathy. If the radiologist reported anything other than these, the case was given a different classification or diagnosis, that is, those with pulmonary infiltrates on x-ray were included in the group of pneumonias in Table 2.

It is observed that there were two cases lost from observation, five with follow-ups of less than one year, and another five with just a year's follow-up. Eventually some disease could develop in this group of 12. One 23-year-old male (No. 17) died within one year of acute glomerular nephritis. What significance this might have is unknown. Attention has already been called to one case of unexplained bleeding which dropped from observation and was diagnosed as carcinoma seven months later. This man has been listed with the carcinomas although to be completely impartial it might be more honest to list him with the "hemoptysis of an undetermined etiology," that is, one in which the prognosis was ultimately poor.

TABLE 3.—*Cases of Hemoptysis of Undetermined Etiology.*

Patient No.	Age	Sex	Years Follow-up Without Developing Major Chest Disease	Comments
1	45	M	9	Cough
2	50	M	8	Cough
3	62	M	8	Deceased, disease of prostate, emphysema
4	52	M	8	
5	30	F	5	
6	43	M	5	Bleeding point seen, cough
7	33	M	8	
8	59	M	4	Emphysema, cough
9	61	F	4	Calcified hilar node
10	46	M	5	Cough
11	38	M	3½	
12	50	M	3	
13	64	F	3	Deceased, coronary
14		F	2	Cough
15	56	M	2	Hypertension, cough
16	24	F	1	
17	23	M	1	Deceased, nephritis
18	51	M	2	Cough
19	54	F	1	Enlarged hilar node, cough
20	39	M	1	
21	45	M	1	Cough
22	55	M	1	
23	69	M	1	Cough
24	48	M	1	Cough
25	37	M	1	
26	55	M	1	Cough
27	48	F	*	Calcified hilar node
28	70	M	*	Emphysema

*No follow-up.

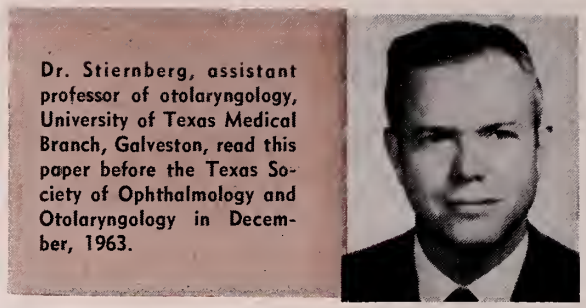
There must be a cause for this bleeding. It may be reasonable to conclude that bleeding could result from rupture of emphysematous blebs. Some suggest tuberculosis and in the past with a few exceptions all cases with chest hemorrhage were considered tubercular. An exhaustive follow-up shows that none of these cases in the group of idiopathic hemoptysis subsequently developed active tuberculosis nor did any of those with adequate follow-up develop any disease of major proportions. Undiagnosed bronchiectasis may be more likely as small bronchial dilations are less easily disclosed although adequate bronchography is done. Limited dry bronchiectasis is consistent with ex-

tended periods of good health and no symptoms.[8] Lung abscess, empyema, and pneumonia produce definite if not characteristic x-ray findings.

Some have considered trauma as a cause. There was one classical case in this series of a healthy 39-year-old male (No. 20) who developed a profuse hemoptysis the day after firing a shotgun. Right angle telescopic examination of the right upper lobe bronchial orifice definitely identified the origin of the blood as the apical segment of the right upper lobe. This would be in the closest proximity to the applied trauma of the gun's kick. There has been no recurrence and chest x-rays are normal after one year. However, trauma has certainly not been a consistent finding in this series.

Douglass[4] could not demonstrate an increased incidence of hypertension in his idiopathic hemoptysis cases. Vinson[8] suggests vicarious menstruation and bronchial erosion from old calcified lymph nodes as causes of idiopathic bleeding. One interesting hemoptysis case that was ultimately classified among the tuberculous group had a large hilar mass on x-ray that was presumed to be malignant. Surgical removal and pathologic study demonstrated broncholithiasis with erosion, obstruction, peripheral bronchiectasis, and atelectasis plus evidence of acid-fast infection. There were two more (Nos. 9 and 27) in Table 3 with calcified nodes that were strongly suspected of erosion as the cause of the hemoptysis.

The writer thought he had an original clinical explanation for this etiologically obscure bleeding; however, review of the literature reveals another author has suggested the same idea. All otolaryngologists see epistaxis with relatively profuse bleeding from a single minute point on the septum where the surrounding nasal mucosa is normal in appearance and there is no history of trauma. The bronchial mucosa is the same pseudostratified columnar epithelium common to the upper and lower respiratory system. Similarly the bronchial mucosa is tightly bound to cartilage rings as the septal mucosa is the underlying cartilage. This binding of the mucosa prevents blood vessel retraction so that bleeding is prolonged. Why cannot the bronchial mucosa bleed in the same manner as the nasal mucosa? Another common relationship is that nosebleeds are frequently seen during or following upper respiratory



Dr. Stiernberg, assistant professor of otolaryngology, University of Texas Medical Branch, Galveston, read this paper before the Texas Society of Ophthalmology and Otolaryngology in December, 1963.

infections as hemoptysis may occur with or following bronchitis. In case No. 6 the bleeding point in the tracheal mucosa was seen through the bronchoscope. This directly observable bleeding point was almost identical to those commonly seen in Kesselbach's triangle on the nasal septum. In conclusion it is felt that there is no one cause for these unexplained cases of hemoptysis. External trauma and particularly the trauma of coughing must play a causative role and especially when applied to the inflamed mucosa of bronchitis or the weakened alveoli of emphysema.

The cases of carcinoma, tuberculosis, bronchiectasis, empyema, and lung abscess have almost always been diagnosed initially. This does not imply that cases of hemoptysis in which a diagnosis is not made at the beginning should be considered safe and free of disease so as to require no follow-up. It does suggest that the prognosis is relatively favorable in such situations after thorough work-up and especially in those in which there is no recurrence of the bleeding. It is seen in Table 2 that there were 28 cases of hemoptysis of undetermined etiology plus another 21 bronchitis cases that had chest hemorrhage, making a total of 49 (42 per cent) of the 117 hemoptysis cases investigated. In other words 42 per cent of this series had a benign bleeding and ultimately presented a good prognosis.

Summary

Two hundred sixty-five consecutive bronchoscopy cases and their comprehensive follow-ups have been studied in an effort to determine the significance of hemoptysis. One hundred seventeen of these cases had hemoptysis of which 42 per cent were benign and of which 28 cases were of undetermined etiology. To evaluate these 28 cases of hemoptysis without etiology, a definite diagnosis of pulmonary hemorrhage must first be

made and other sources of blood such as the nose, mouth, and stomach must be excluded. A method of investigating these patients is presented along with a differential diagnosis of those cases in which the cause is determined. Finally an explanation for hemoptysis of idiopathic etiology is suggested and discussed, plus a comment on its prognosis.

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Fucose Deficiency May Cause Mucus in Lungs of CF Victims

A deficiency of fucose—one of the chemical constituents in lung tissue—may be related to the formation of the deadly mucus that clogs the lungs of victims of cystic fibrosis.

A report of this was presented to doctors and medical researchers concerned with cystic fibrosis at their annual meeting in Seattle, Wash.

Dr. R. E. Knauff, who prepared the paper with J. A. Ragone, both of the Cystic Fibrosis Research Institute in Philadelphia, said their conclusions are based on analytical profiles of cellular fractions from normal and cystic fibrosis human lung tissue and rat lung tissues. The results cited the possibilities of an overall excess of sialic acid and a deficiency of fucose in the lung tissue from CF victims. There was a particular deficiency of fucose in the nuclear and supernatant fractions.

Cystic fibrosis, which afflicts about 3,000 babies born in the United States each year, may be inherited when the parents of a child each carry one CF gene. The disease causes the mucus-forming exocrine glands in the victim to create a thick mucus which collects in the lungs and other parts of the body.

About 90 percent of the deaths from CF are due to lung obstruction and consequent infection. About 10 percent of those afflicted with full cases of CF live to be adults. But early diagnosis and intensive care are now improving prospects for those born with this dread disease.

Angioid Streaks

Important Clue to Obscure Cause Of Gastrointestinal Hemorrhage

CASE REPORTS

OCCURRENCE of gastrointestinal hemorrhage as a complication of angioid streaks and pseudoxanthoma elasticum has been recognized with increasing frequency since Revell and Carey's report[16] in 1948. The purpose of this writing is to review the manifestations of this generalized elastic tissue disease and to emphasize the retinal findings that may provide the clue to correct diagnosis of an obscure cause of gastrointestinal bleeding. A case report illustrates these points.

Case Report

A 28-year-old white male laborer was admitted to the John Sealy Hospital in Galveston because of gastrointestinal bleeding and anemia. Three days prior to admission he had become nauseated and vomited dark brown material and had several black, tarry stools. Hematemesis and melena continued to the time of admission. The patient denied having had abdominal pain in the past or present illness.

Over the past ten years, the patient had irregular episodes of hematemesis and melena approximately once or twice a year, but never needed blood transfusions. He was hospitalized at the age of 18 and a diagnostic work-up was performed, including an upper gastrointestinal series, the results of which are not known. During this ten-year period he had been treated intermittently with milk, cream, and antacids. He had been a large beer consumer and at times his dietary intake was poor. He denied ever having had jaundice or ascites.

The patient was an only child. His mother was said to have died at age 47 of "stomach trouble." A maternal grandmother and maternal cousin have excessive nosebleeds.

On physical examination, the patient appeared in slight distress. Blood pressure was 136/70; pulse rate, 114; and respiratory rate, 18. The nail beds and conjunctiva were pale. The bowel sounds were moderately hyperactive, the liver and spleen were not enlarged, and there was only slight epigastric tenderness. On initial examination, retinal and skin changes were not noted.

Blood hemoglobin was 6.7 gm. and the hematocrit, 22 per cent. White blood count was 20,000 with 78 per cent polymorphonuclear leukocytes, 13 per cent lymphocytes, and 9 per cent monocytes. Urinalysis, cephalin flocculation, thymol turbidity, acid phosphatase, and bilirubin were within normal limits. Blood urea nitrogen was 45 mg. per 100 cc. initially, and later fell to 17. Electrocardiogram was normal. Upper gastrointestinal series revealed the esophagus and stomach to be unremarkable. The gastric mucosal pattern and peristaltic activity appeared normal. The duodenal cap filled well, showing no evidence of active ulceration or deformity.

The patient was given a Sippy No. 1 diet, anticholinergic drugs, phenobarbital, and blood transfusions. On the second day of hospitalization, despite administration of seven units of whole blood, the hemoglobin was down to 4.5 gm., and the hematocrit to 15 per cent. Because of continued bleeding, an exploratory laparotomy was performed on the second day. At surgery a duodenostomy and multiple enterostomies for irrigation were performed. There was blood in the bowel, and the entire gastrointestinal tract from stomach to ileum was inspected with no source of bleeding found. No definite procedure was done. The patient tolerated the procedure well, but continued to have black stools for ten days postoperatively. On the first postoperative day, the hemoglobin was 5.4 gm., and by the third postoperative day it was up to 8.5 gm. He received a total of 11 units of blood.

The patient's hemoglobin stabilized at about 9 gm. No further hematemesis occurred, but stools remained slightly dark and guaiac positive. He was discharged on the 14th postoperative day on a soft bland diet and was asked to return to the outpatient clinic in two weeks. He failed to keep this appointment, and all subsequent attempts to contact him have failed.

Ophthalmologic consultation was obtained on the second postoperative day. Vision was 20/25 in both eyes. Positive findings were limited to the fundus examination which showed typical angioid streaks in both eyes (Figs. 1 and 2). The macular areas in both eyes showed a brownish discoloration suggesting old reabsorbing hemorrhages. The entire retina exhibited a fine granular, mottled appearance. Because of the retina findings, dermatologic consultation was obtained and the skin was noted to show a yellow discoloration and wrinkling in the lateral aspect of the neck and in both inguinal areas. A biopsy was performed in the axillary and inguinal areas and tissue submitted for stains. Histologic study revealed the characteristic features of pseudoxanthoma elasticum (Figs. 3 and 4). The overlying epithelium was not remarkable except for

A case report is presented which illustrates why the ocular fundus should be examined in all cases of unexplained bleeding.

ROBERT L. ROCK, M.D.

focal areas of broadening of the rete pegs with slight elongation. The underlying corium showed deposits of calcium in the middle third of the corium with fragmentation of elastic fibers.

Angioid streaks of the ocular fundus were first described by Doyne[4] in 1889. Darier[3] in 1896 described the skin lesions of pseudoxanthoma elasticum. However, these two findings were not associated until 1929 when Grönblad[7] made his correlation. Since then, the combination of skin and ocular lesions has been known as the Grönblad-Strandberg syndrome. Numerous reports then occurred in the ophthalmologic and dermatologic literature, but only since about 1938 has the generalized nature of the elastic tissue degeneration been recognized with the attendant involvement of elastic components of the blood vessels. In 1948 gastrointestinal bleeding associated with the Grönblad-Strandberg syndrome was first reported; since then many more cases, as well as hemorrhagic tendencies in other organs, have been reported.

Ophthalmologic Manifestations

Eye lesions are almost always bilateral. In one large series of pseudoxanthoma elasticum, 87 per cent of these patients were noted to have angioid streaks.

The appearance of the ocular fundus is one of a bizzare network of delicate pigmented streaks, extending out into the fundus, varying in color from dark red to brown or even black, and having slightly jagged, serrated edges. They lie beneath the retinal vessels and above the choroidal vessels. They usually extend toward the equator in a radial distribution like spokes in a wheel and give the appearance of cracks in dry mud. These streaks may appear joined together in a mosaic pattern like branches of a tree in winter. They vary in width from fine lines to wider than retinal veins. The fundus may appear mottled by minute yellow spots and dustlike pigment stippling. The macular area is usually involved with fresh or old hemorrhages, scars, and dark pigment mottling. Vision varies from normal in early cases to poor as a result of hemorrhagic tendencies in the macular areas. In one large series studied, a significant loss of visual acuity was noted in 63 per cent.

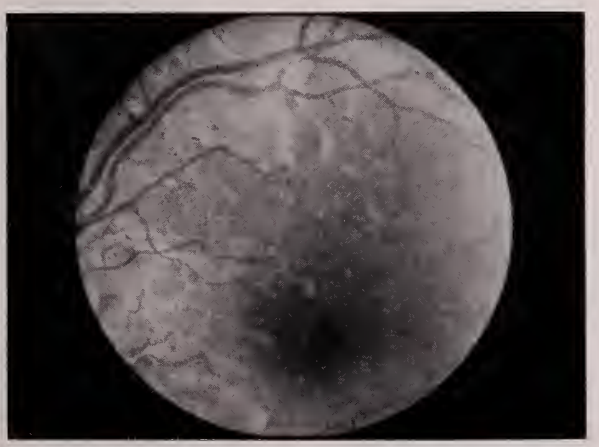


Fig. 2. Fundus photograph of the left eye, showing same changes as in the right eye.

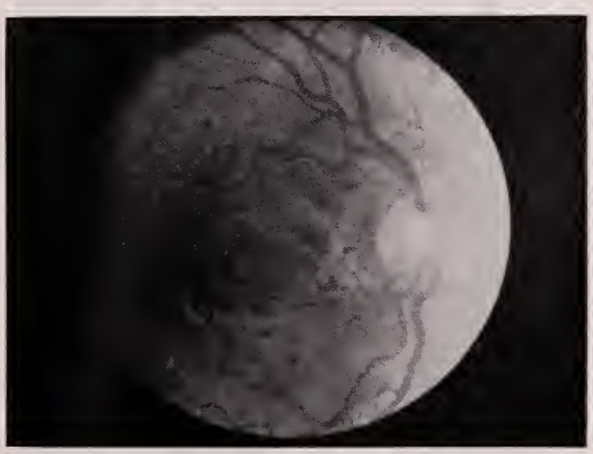


Fig. 1. Fundus photograph of the right eye, showing angioid streaks radiating out from the optic disk.

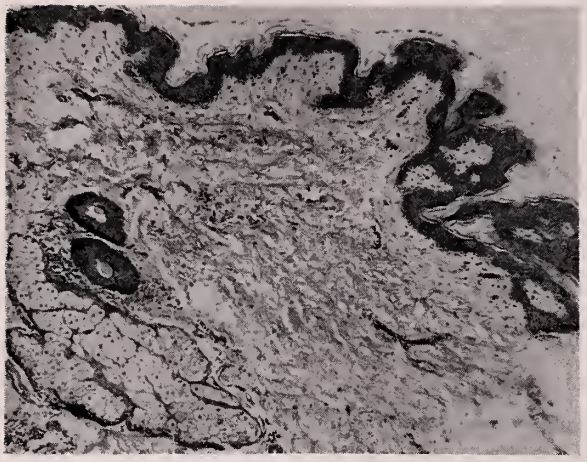


Fig. 3. Biopsy of the skin showing disruption of the elastic fibers. (Hematoxylin-eosin stain.)

Pathologic studies (Böck; Hagedoorn; Klien) have shown that the histologic lesion responsible for angioid streaks involves Bruch's membrane which is interrupted at the sites of the streaks. [Bruch's membrane is a thin membrane also known as the lamina vitrea, which lines the entire inner choroid. It consists of an extremely thin external elastic lamina derived from the network of delicate fibril which invests the chorio-capillaries and an inner cuticular layer produced by the retinal pigment epithelium.] The lamina vitrea takes a basophilic stain, and shows a variation in thickness and staining properties. The defects and ruptures in this membrane are irregular, with jagged edges and varying breadth. Fibrovascular tissue from the choroid can be seen to fill the defects and even extend through them beneath the adjacent pigment epithelium. The pigment epithelium over the breaks may be normal, elevated by scar, or partially degenerated.[9]

The pathologic process underlying the diffuse opaque yellowish or grayish areas consists of extensive new formation of cuticular substance similar to the cuticular portion of Bruch's membrane but is many times thicker.

In the late stages of the disease the macular area is invariably occupied by a lesion that has the typical clinical and histologic appearance of disciform degeneration.

Dermatologic Manifestations

The earliest changes usually are observed in the skin. The appearance is one of soft, inelastic, wrinkled folds of granular yellow skin (Fig. 5). The descriptive terms of "picked chicken" skin or "morocco leather" have been applied. Lesions vary from pinhead to pea-sized flat papules that may fuse to form plaques. The lesions are usually symmetrical and best seen in the flexural folds of the axilla, posterolateral neck, antecubital fossae, and inguinal regions.

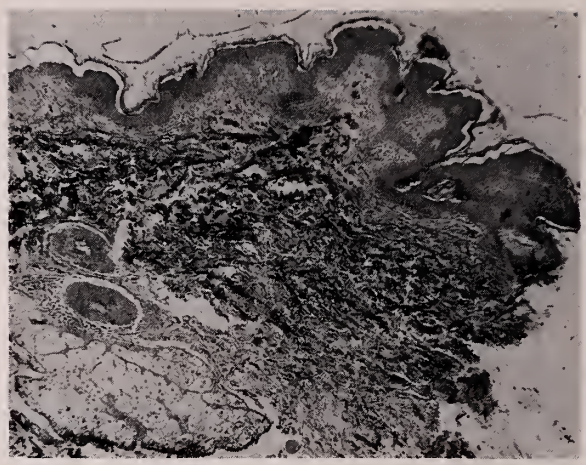


Fig. 4. Biopsy specimen of the skin, Verhoeff's stain for elastic tissue, showing fragmentation, disintegration, and disorganization of the elastic tissue. Elastic fibers are stained black.

Histologically, these changes are the result of alterations in the elastic tissue in the mid and deep corium. Proliferation and nodular degeneration of elastic fibers is noted. Swelling, granularity, fragmentation, and loss of elastic fibers are seen in these foci. Calcification is common in the areas of elastic tissue degeneration. The tendency to selectively involve the elastic tissue and spare the collagen is a characteristic feature.

Visceral Manifestations

Kaplan and Hartman,[10] in 1954, reported the case of a 23-year-old female with two episodes of gastrointestinal bleeding which required a partial gastric resection. Superficial mucosal hemorrhages and erosions as well as capillary and venous dilatation of the mucosa and submucosa were noted. Histologic study of the resected stomach showed elastic tissue degeneration of the small and medium-sized gastric arteries with microaneurysms and intimal obliterative sclerosis with calcified plaque formation.

Bleeding may be massive, and numerous episodes may occur. The occurrence of gastrointestinal bleeding and angioid streaks during pregnancy has been stressed. Other changes in elastic tissue do occur during pregnancy. Biopsy of abdominal striation during pregnancy show frank disintegration of elastic fibers.

Incidence of gastrointestinal bleeding seems higher in females. Although gastrointestinal bleeding is one of the more serious forms of bleeding, other organs known to be

Fig. 5. Typical skin lesion of pseudoxanthoma elasticum in flexural folds of neck, as described. (Photograph courtesy of Dr. Leonard C. Alexander, Detroit, Mich.)

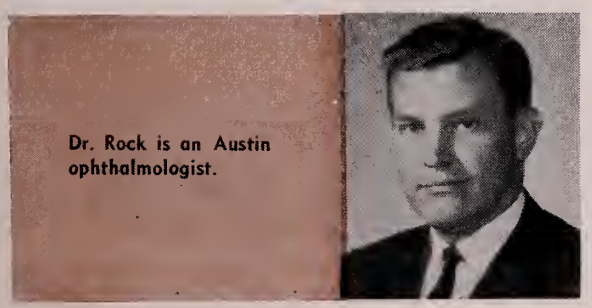
affected with arterial damage include the kidneys, bladder, lungs, pancreas, uterus, brain, thyroid, heart, and mesentery. Arterial changes consist chiefly of fragmentation of the elastic tissue of the internal elastic laminae and medial calcification. Clinically, hemorrhagic tendencies in the aforementioned organs have been reported in the form of hematuria, epistaxis, uterine bleeding, hemoptysis, bleeding from the gums, subarachnoid, and intra-articular hemorrhage.

Several cases of osteitis deformans (Paget's disease) associated with angioid streaks have been documented.[22]

In view of the diffuse involvement of the elastic tissue degenerative process, the terms "systemic elastic disease," "hereditary elastodystrophy," and "elastorhexis," have been suggested to better indicate the true nature of this process.

Familial predisposition is indicated; but because of the relatively small number of cases studied, no precise genetic picture can be formulated. However, an autosomal recessive trait is suspected.

The ocular fundus should be examined in all cases of unexplained bleeding, especially gastrointestinal.



Summary

The significance of the various ocular, dermatologic, and vascular manifestations of the Grönblad-Strandberg syndrome (pseudoxanthoma elasticum) as part of the more generalized process of elastic tissue degeneration is cited. A case is presented in which a patient with unexplained gastrointestinal bleeding and without typical symptoms of peptic ulceration was noted to have angioid streaks; further confirmatory evidence was obtained.

The ocular fundus should be examined in all cases of unexplained bleeding, especially gastrointestinal. The characteristic changes of angioid streaks should be appreciated by all physicians.

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Nasal Glioma

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Reported is a case of one of the rarest congenital nasal tumors.

CASE REPORTS

CONGENITAL LESIONS of the nose are rare and may be serious. They have been classified by Macomber and Wang[4] according to their primordial origin. Those of neurogenic origin comprise possibly the most serious congenital nasal tumors and include encephalocele, neurofibroma, meningocele, and glioma. The neurogenic tumors must be differentiated from those of mesodermal origin, including hemangioma and lipoma, and those of ectodermal origin, including dermoid cysts, sebaceous cysts, epidermoid cysts, abscesses, nasal polyps, ethmoid cysts, and lacrimal duct apparatus cysts.

The nasal glioma is one of the rarest congenital nasal tumors. The first reported case was by Schmidt[6] in 1900. Since that time there have been some 90 cases reported. Of these, 60 per cent have been extranasal, 30 per cent intranasal, and 10 per cent have been a combination of the two. Developmental errors are believed to be responsible for these tumors. The glioma is thought to arise either from primitive neural ectoderm that was isolated in the nose during early fetal development,[2, 3] or from trapped congenital herniations of the cerebrum[5] similar to the formation of an incarcerated encephalocele. Differentiation of nasal tumors is made chiefly by histologic appearance. The growth generally is well circumscribed, but nonencapsulated, and is covered either by skin or nasal mucosa. Astrocytes are fairly well distributed throughout cerebral matrix and glial fibers (Fig. 1). There also are irregularly scattered capillaries and minimal pericapillary fibrous tissue.[8]

Clinically, the nasal glioma is a congenital lesion with either intranasal or extranasal presentation or a combination of the two. This presentation may be associated with broadening of the nasal dorsum or nasal obstruction. Intranasal tumors are attached by a stalk high in the nasal vault. Extranasal tumors appear lateral to the midline of the root of the nose. The tumor is firm, pink, incompressible, and does not change size when the patient cries or sneezes. Growth of the tumor is proportional to the growth of surrounding structures. Other congenital defects are not associated, and there is no familial incidence.

There is uniform agreement that surgical excision is the treatment of choice. Preliminary planning may serve to avoid the complications of intracranial infection and to prevent recurrences due to limited excisions. The surgical approach varies from transnasal to lateral rhinotomy. A preliminary biopsy or needle aspiration may tend to complicate the case if there is an intracranial connection. Smith[7] has urged an intracranial exploration in those with intranasal lesions,

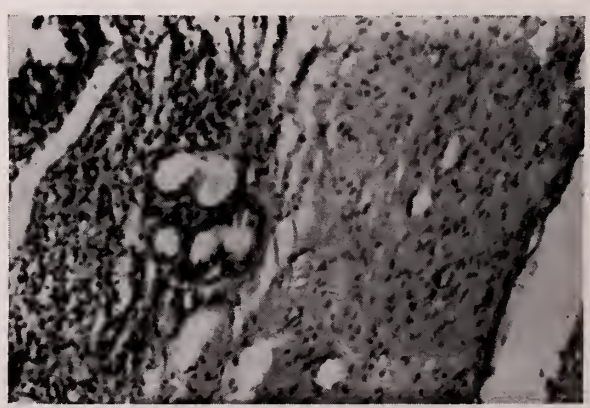

Fig. 1. Low power photomicrograph showing nasal mucosa, subcutaneous tissue, and tumor.

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those with cerebrospinal fluid rhinorrhea, or those who have had meningitis. A craniotomy should also be performed when intracranial connections are found. It is most important to be prepared to proceed immediately with a craniotomy when excision is followed by a cerebrospinal fluid leak. Radiosensitivity has been suggested in only one report.[1]

Case Report

This patient, age one, was first seen on Feb. 2, 1963. Otherwise normally developed, he had had complete obstruction of the left nasal passage since the age of six months. A nasal tumor had been diagnosed when he was nine months. The physical examination showed a well developed, well nourished white male. There was broadening of the root of the nose and flattening of the left alarfacial groove. The left nasal passage was completely blocked with a firm, smooth, pink lesion that extended to the anterior end of the inferior turbinate. The lesion did not expand when the child cried. It appeared to be attached high in the nasal vault by a broad pedicle. Laminograms failed to reveal a defect in the cribriform area. The preoperative diagnosis was nasal glioma or dermoid cyst.

Surgical excision on Feb. 12, 1963, was by a lateral rhinotomy approach. Excision was performed by elevation of a mucoperichondrial flap of the entire left septal mucosa which was carried across the vault of the nose to include the broad pedicle of the tumor. There was no cerebrospinal fluid leak. The septum was grafted with a split thickness skin graft.

The histologic examination was consistent with nasal glioma. The neurosurgical consultant was of the opinion that a combined approach should be reserved for any recurrence.

Postoperatively, there was a moderate febrile course of three days' duration which responded to antibiotics.

It is now 17 months since the operation. The patient has had some mucous drainage and crusting; the cosmetic results, however, have been good, and the airway is excellent. There is no clinical evidence of recurrence.

Summary

With this brief review of nasal glioma and case report of an intranasal lesion, caution is urged in dealing with congenital nasal lesions, and the possibility of intracranial connections is stressed.

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The Quiet Company

What do the following have or once have in common: actress Nanette Fabray, astronaut Alan B. Shepard, Jr., a traffic cop in New York, elder statesman Bernard Baruch, Thomas A. Edison, a business executive in Wyandotte, Mich., Ludwig von Beethoven, and from 15 to 18 million living Americans?

The answer is impaired hearing, the No. 1 physical handicap in the United States today, according to the June, 1964, issue of *Medicine at Work*, published by Pharmaceutical Manufacturers Association.

Cytogenetic Studies

Diagnostic Tool in Clinical Medicine

REVIEW ARTICLES

CYTOGENETIC STUDIES may be helpful to the physician in diagnosing certain clinical conditions. As yet laboratory facilities are not available to evaluate every patient who deserves it, but clinical findings may enable one to select patients in whom studies are apt to be most helpful.

A brief discussion of the carrier of genetic information, deoxyribonucleic acid (DNA), is in order. The function of DNA is to replicate (duplicate) and to control the chemical processes that give an organism specificity.[11] The DNA molecule is constructed of three building blocks: phosphate, deoxyribose, and four bases (two purines, adenine and guanine, and two pyrimidines, cytosine and thymine). Each single group consisting of a base, sugar, and phosphate is called a nucleotide. Hence, DNA is spoken of as a polynucleotide chain. A gene then may be considered as a region of DNA material with a particular sequence of nucleotides that indirectly controls the synthesis of one nucleotide chain, one specific enzyme, or one protein; hence, a gene is a functioning unit of the chromosome.

The nucleus of a cell contains the genetic material (DNA) in the form of chromosomes and varies in appearance depending on the stage of development of the cell. During mitosis the chromosomes appear as thread-like structures composed of two chromatids each containing exactly the same genetic material. In metaphase the chromosomes are sufficiently separated so that they can be examined by microscopy for identification and counting.

Tjio and Levan in 1956[22] first reported the normal number of human chromosomes to be 46 (44 autosomes or somatic chromosomes and 2 sex chromosomes). The chromosomes were classified into numerical groups by common consent of geneticists meeting in Denver in 1960. This classification, referred to as the Denver classification,[12] is the one most widely used in this country in identifying chromosomes by their length and the position of the centromere (Fig. 1). The *centromere* is the point on the chromosome at which it is attached to the spindle during cell division. The three types of chromosomes found in man are described as *metacentric* (centromere in center with arms of the chromosomes of equal lengths), *acrocentric* (centromere near end) and *submetacentric* (centromere between these two positions).

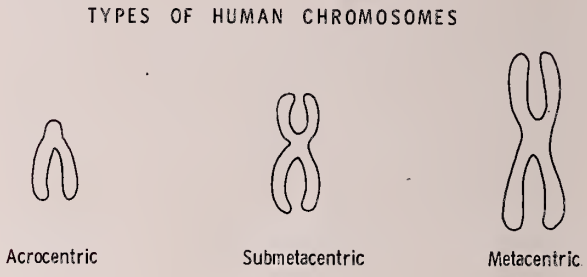

Fig. 1.

Other terms commonly used in discussing chromosomes will be mentioned briefly. *Diploid,* the number of chromosomes in somatic cells, is constant for any species and is 46 in man. *Haploid* refers to the number of chromosomes found in the germ cells and is 23 in man. *Euploidy* refers to any multiple of the haploid number, and *aneuploidy* refers to a deviation from a multiple of the haploid number. Examples of aneuploidy might be a *trisomy* which indicates that three instead of two chromosomes of a given type are present, and *monosomy* which suggests the presence of one instead of two of a particular chromosome type. A *mosaicism*

From the present state of human cytogenetics, an interesting review of some of the main conclusions of clinical interest, illustrated with cases from the authors' own experience.

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is defined as a species which contains more than one cell line of chromosomes.

Many body tissues can be utilized for chromosomal analysis, but the method described by Moorhead and others[16] in 1960 for studying peripheral blood or bone marrow is presently the most practiced. Leukocyte-rich plasma is taken from 10 cc. of sterile heparinized blood and placed in a special culture medium containing phytohemagglutinin used for stimulation of mitosis (Nowell, 1960).[18] The culture is incubated for 72 hours at 98.6 F. (bone marrow—24 to 48 hours). Colchicine is then added to stop mitosis (hopefully in metaphase), and to help shorten and spread the arms of the chromosomes. Next the cells are treated with a hypotonic citrate solution to swell them, and then are fixed with glacial acetic acid and alcohol. Feulgen or aceto-orcein, two stains with an affinity for DNA, can be used to stain the chromosomes. The cells are squashed on a slide under a cover slip, or prepared by the air-dry method, and are then ready for study by phase or light microscopy. The yield of mitoses spread well enough for counting is somewhere from none to many per slide, therefore many slides must be prepared, for if the chromosomes are not well spread, they cannot be counted or photographed. At least 30 cells must be counted, and if essentially no difference exists in the number of chromosomes per cell, one can feel fairly certain of the diploid number of the patient. If some few cells differ in chromosomal number, it may be necessary to count chromosomes in as many as 100 cells. Representative cells are photographed and enlargements made. The chromosomes are then individually cut out, paired, and classified in order to prepare a *karyotype*. The karyotype of a normal male (Fig. 2) demonstrates how the chromosomes can be grouped according to the Denver classification, and it also shows why it is impossible to differentiate some of the chromosomes within a given group because of their similarity in morphology. The minimum work hours required to complete such a study on one patient have averaged from 24 to 36. If an abnormality is suggested after counting the first 30 cells, as many as 40 to 50 additional work hours may be involved in one study. It must also be recognized that definite limitations of the test are imposed by the fact that the cells are grown in an environment only simulating the in vivo situation and that other factors, for example, medications being taken by the patient, may also influence cultural results. Furthermore, the finding of a given karyotype in one body tissue does not necessarily indicate that other tissues would reveal similar patterns; thus, the possibility of mosaicism must be considered in evaluating information obtained on chromosomal analysis.

One of the mechanisms of formation of abnormalities of chromosomal number is called *nondisjunction* (Fig. 3). This occurs,

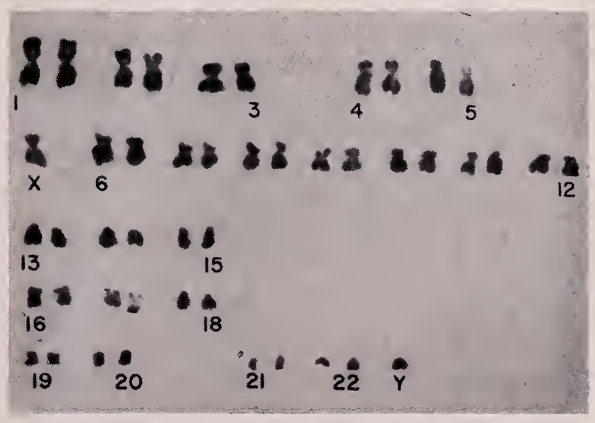

Fig. 2. Karyotype normal male. W. C., Case 5, four years old, ambiguous external genitalia, November, 1962.

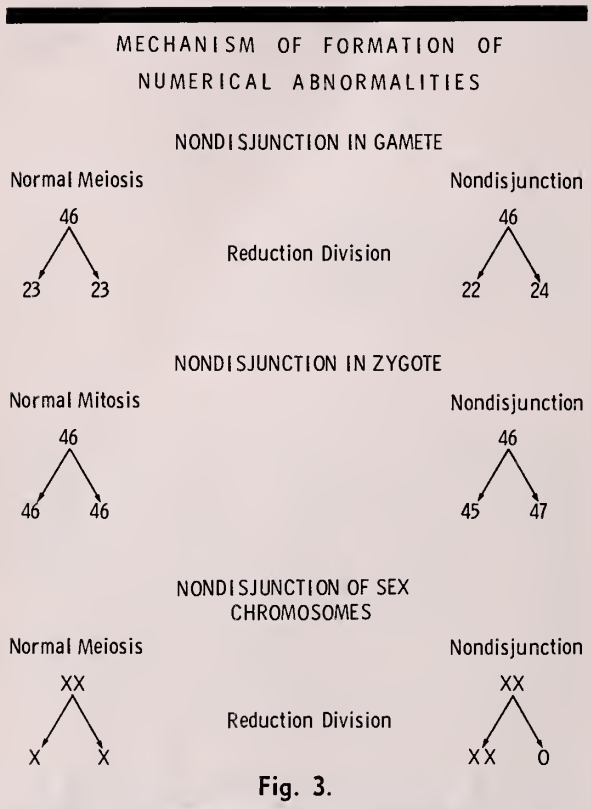

Fig. 3.

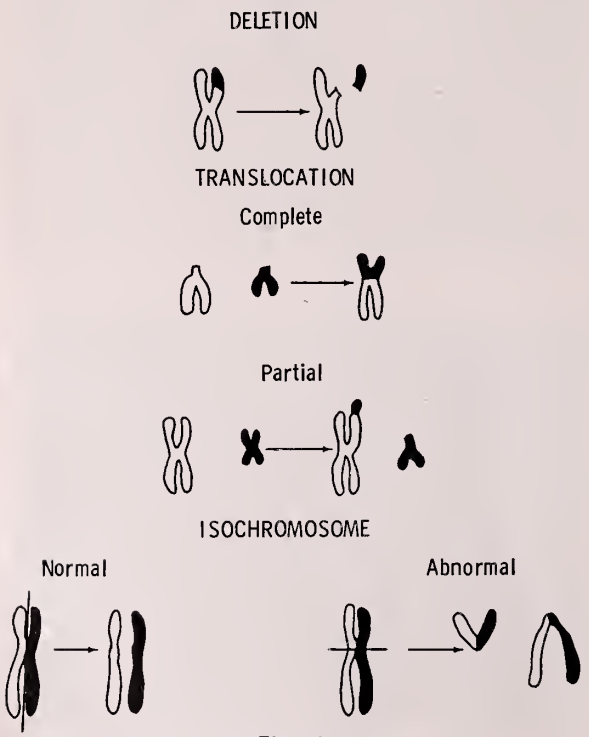

Fig. 4.

during mitosis or meiosis, when a pair of chromosomes fail to separate at anaphase and both go to the same pole. Hence, the daughter cells will not contain equal numbers of chromosomes. This can occur in the germ cells in either of the two meiotic divisions, or it can occur in the early cells of the zygote—that is, in the early embryonic cells—during mitosis. Fig. 3 also demonstrates the mechanism of development of a mosaicism. The abnormalities resulting from nondisjunction of the sex chromosomes will be discussed later.

Structural changes of the chromosomes can also occur (Fig. 4). *Deletion* or omission of a segment of a chromosome is possible. *Translocation* may be complete or partial, complete being transfer and attachment of one chromosome to another, and partial, meaning transfer of a segment of one chromosome to a different site on that chromosome or to a site on another chromosome. A third abnormality known as an *isochromosome* results when splitting of the chromatids occurs transversely rather than longitudinally during anaphase.

Last to be considered are some of the clinical conditions which merit chromosomal studies (Table 1). These will be divided into two groups: conditions associated with abnormalities of the chromosomes, numerically or structurally, and conditions in which genetic sex might be uncertain.

Numerical Abnormalities Of Autosomes

Several syndromes have been described in association with numerical abnormalities of the autosomes, the classic example being mongolism, better termed Down's syndrome after the man who first described it in 1866. Lejeune[14] in 1959 found that some patients with features of mongolism had 47 chromosomes, the extra one matching in morphology those of the 21-22 group. It has been realized since that trisomy 21 is characteristically found in mongols born to older mothers and results from nondisjunction of the gametocytes.

In contrast, another chromosomal abnormality is observed more often in mongols born to younger mothers. More correctly classified with the structural abnormalities, it consists of a translocation between two of the small acrocentrics of the 21-22 group[7] or

TABLE 1.—*Clinical Conditions that Merit Chromosomal Analysis.*

I.	Clinical conditions associated with chromosomal abnormalities
	1. Autosomal
	a. Trisomy 21-22 (mongolism, Down's syndrome)
	b. Trisomy 16-18
	c. Trisomy 13-15
	2. Sex chromosomes
	a. Ovarian dysgenesis
	b. Klinefelter's syndrome
	c. Unusual patterns (XXX, XXXX, XXXXX)
	B. Structural abnormalities
	1. Translocation mongol
	2. Philadelphia chromosome
	3. Various abnormalities following irradiation or use of certain drugs
II.	Clinical conditions associated with uncertain genetic sex

of a translocation of one of the 21-22 group to a member of the 13-15 group.[3] Translocation mongolism appears more often to be familial, and carrier states in the male as well as the female have been described. In these mongols the total count is 46 but karyotyping reveals an abnormal chromosome which probably contains the genetic material of the two chromosomes involved in translocation.

An example of a patient with Down's syndrome is represented by the following case:

Case 1.—(Fig. 5) R. F., a three-year-old male was born Aug. 15, 1960, weighing 6 lbs. 15 oz., following a 38-week gestation complicated by maternal anemia. The amniotic fluid was stained yellow, and the infant was questionably icteric and somewhat hypotonic. His head circumference was 33.5 cm. and chest circumference 32.5 cm. He had hepatomegaly, mild hypospadias, descended testes, and bilateral syndactyly of the second, third, and fourth toes and third, fourth, and fifth fingers. Hyperbilirubinemia due to hemolytic disease necessitated three exchange transfusions. Brain damage was suspected and the question of mongolism was raised. Family history revealed that the mother was 39 years old, and the father, 45, had syndactyly as did seven of the 11 siblings. At age ten months the child was seen following an episode of cyanosis thought to be due to congenital heart disease; however, the physical examination of the heart, chest x-ray, and electrocardiogram were all within normal limits. His developmental level at that time was estimated to be at two to three months. He was not seen again until January, 1963, when he developed a right lower lobe pneumonia which cleared slowly with antibiotic therapy. Pertinent physical findings included small stature, typical mongolian facies, fissured tongue, flattened occiput, stubby fingers with syndactyly as noted previously, transverse palmar and plantar creases, hypotonic muscles, hyperflexible joints, and mild hypospadias. Chromosomal studies of peripheral blood leukocytes

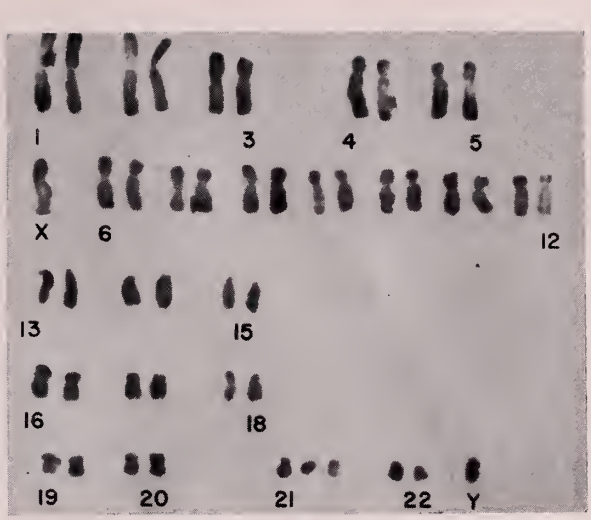

Fig. 5. R. F., Case 1, three-year-old, Down's syndrome.

revealed a modal number of 47 with an extra chromosome (trisomy) in the 21-22 group, thus confirming the diagnosis of Down's syndrome.

Two other numerical abnormalities of the autosomes have now been well delineated: the trisomy 13-15 first described by Patau and his colleagues[21] in 1960, and the trisomy 16-18 described by Edwards and associates[4] in 1960. Although there is some variability in the more than 30 cases of trisomy 16-18 now reported, the pattern of clinical findings is consistent enough for recognition in a given patient. A patient with this syndrome usually has an oddly shaped head, low-set and malformed ears, micrognathia, and various malformations of the heart, intestines, and extremities. Patients with trisomy of one of the 13-15 group also usually have numerous anomalies, some of which include microphthalmia, cleft palate and lip, hemangiomas, malformations of the heart, polydactyly, and flexion deformities of the fingers and hands. Patients with either of these syndromes have 47 chromosomes, are usually mentally retarded, and have short life expectancy.

Chromosomal analysis in individuals with these findings is indicated to confirm the diagnosis, so that one may counsel the parents as to prognosis, and help determine what elective surgical repairs of anomalies are indicated. For these reasons the following case was studied:

Case 2.—(Fig. 6) G. B., a ten-week-old white female was the product of a term gestation complicated at six weeks by maternal bleeding and treated with hormones of unknown type. Labor and delivery were normal, but the baby was noted to have bilateral cleft palate and lip, microcephaly, and multiple hemangiomas at birth. She required tube

Fig. 6. G. B., Case 2, ten weeks old, trisomy 13-15.

feeding for six to eight weeks and was noted to have episodes of cyanosis presumably due to aspiration of feedings. She remained weak and inactive and failed to gain weight.

Family history was negative except that the mother, age 22, had had three previous miscarriages and one normal living child, now two years old.

Physical examination revealed a small white female, weight 6 lbs., 3 oz., (tenth percentile for birth), length 51.5 cm. (75th percentile for birth), and head circumference 33.5 cm. (tenth percentile for birth). She was inactive, had bilateral cleft palate and lip, narrow tapered forehead, bilateral microphthalmia, and hemangiomas over her head and back. Her chest measured 30.5 cm., was symmetrical, and lungs and heart were within normal limits. There was a small umbilical defect, but no palpable abdominal masses, and the genitalia were normal. The general muscle tone was poor, Moro reflex was fair, and her hands were fisted with pronounced flexion of the middle phalanges bilaterally. She had syndactyly of the left second and third toes.

Laboratory studies revealed a hemoglobin of 10.5 gm. per 100 cc. and hematocrit of 32 per cent with normal total and differential leukocyte count. Urinalysis and blood urea nitrogen were normal.

An electrocardiogram was suggestive of biventricular hypertrophy; however, a barium swallow and chest x-rays were normal. Skull x-rays revealed decreased ossification, shallow supra-orbital ridges, and no intracranial calcifications. Roentgenograms of the feet showed a soft tissue deformity of the left foot with wide separation of the first and second toes. An intravenous pyelogram was normal. Chromosomal studies of peripheral blood leukocytes revealed a modal number of 47 with an extra chromosome in the 13-15 group, thus she had a trisomy 13-15.

Numerical Abnormalities Of Sex Chromosomes

Several syndromes are presently known to result from abnormalities in number of sex chromosomes, some of which can be recognized on the basis of clinical appearance.

While the phenotypic sex is readily established, the nuclear sex must be determined by studying cells from a buccal smear preparation and counting the number of nuclei containing a Barr body[5] or sex chromatin mass. This body is thought to be the heterochromatic, inactive X in the female, as was suggested by Ohno[20] in 1962, and it is found in close proximity to the nuclear membrane. There may be a small percentage of cells (1-2 percent) with nuclei containing bodies resembling the sex chromatin mass in the normal male; however, a larger percentage of positive nuclei are consistently found in the normal female, usually greater than 30 percent. The number of sex chromatin masses found in the cell nuclei is always one less than the number of X chromosomes found on karyotype. Thus the absence of a chromatin mass would suggest a normal XY male or an XO sex chromatin negative female.

The syndrome known as ovarian dysgenesis (Bonnevie-Ullrich, Turner's syndrome) presents the external appearance of a female (phenotype) most commonly, but buccal smear preparations are negative for sex chromatin mass, and the cells have a chromosome number of 45 with an XO configuration of the sex chromosomes as first described by Fraccaro.[6] The clinical findings of sexual infantilism, webbed neck, mental retardation, and coarctation are well known and need not be discussed here. These patients are usually sterile.

Case 3.—(Fig. 7) L. F., an eight-year-old white female, product of an eight-month gestation, weighed 6 lbs. at birth. She was noted to have swelling of

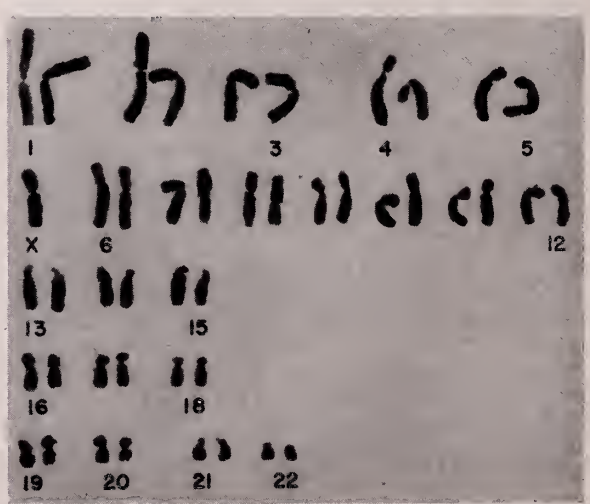

Fig. 7. L. F., Case 3, eight years old, gonadal dysgenesis.

the lower extremities and webbing of the neck, but otherwise seemed normal until four weeks of age when she had an episode of cyanosis. This cleared with oxygen and no cardiac abnormality was found. Other than the usual childhood diseases and one episode of pneumonia, she remained in fairly good health and her developmental pattern was within normal limits.

In August, 1963, she began having occipital headaches and was found to be hypertensive with blood pressures in right arm 180/150, left arm 176/140, and right leg 180/120.

She measured 116.5 cm. in height (tenth percentile for 7½ years) and weighed 56.5 lbs. (50th to 75th percentile for 7½ years). She had a low hairline with bilateral webbing of the neck, a moderate pectus excavatum and a bulging left parasternum. Her lungs were clear and cardiac findings included a short early ejection murmur at the base, pulmonic, and aortic areas, a short systolic murmur at the left scapulohumeral area, and a split second sound with no evidence of cardiac enlargement. The remainder of her physical examination was normal.

Intra-aortic pressure studies revealed no evidence of coarctation of the aorta and further studies are in progress to determine the etiology of her hypertension. The hemogram and urinalysis were normal. No sex chromatin mass was seen in the 300 cells that were examined and culture of her peripheral blood leukocytes revealed a modal count of 45 which confirmed the XO configuration of the sex chromosomes.

A second numerical abnormality was described by Bergman[1] in Klinefelter's syndrome which presents as a phenotypic male with clinical findings of small soft testes, absent spermatogenesis, and some decrease in mental activity. The majority of these patients have buccal smears with positive sex chromatin and most characteristically have 47 chromosomes with an XXY configuration. Other variants XXXY,[5] XXYY,[17] and XXXXY[8, 9] have been reported but in general do not differ from the more common XXY type with the exception of the XXXXY which usually has severe mental retardation, hypogonadism, and skeletal abnormalities.

One other well-documented but rarer abnormality, the Triple X female ("Super Female"), who has amenorrhea and subfertility and on buccal smear preparations two sex chromatin masses in the nuclei, shows on karotyping a total of 47 chromosomes with an XXX configuration.[10] In addition there have been reports of patients with XXXX[2] and XXXXX[13] which have three and four sex chromatin masses in the cell nuclei. The child with the XXXXX had multiple anomalies and retarded growth and development, however many of the other variants have only mental retardation.

All of the before mentioned abnormalities might result from nondisjunction during gametogenesis and the XO and other abnormalities might also result from nondisjunction after fertilization in the embryonic cells.

Structural Abnormalities Of Chromosomes

The translocation mongol, one of the structural abnormalities, has been previously mentioned.

Another structural anomaly is that found in chronic myelogenous leukemia. It is the only consistent chromosomal abnormality reported in malignant disease. A variety of other chromosomal aberrations have been noted in association with neoplasms and also following irradiation or the use of radiomimetic drugs. In chronic myelogenous leukemia, a deletion of part of the long arms of one of the chromosomes in group 21-22 is found in some of the cells of the blood and bone marrow.[19] The significance of this abnormal chromosome, the Philadelphia chromosome, in relation to chronic myelogenous leukemia is not known, but this association, along with the higher incidence of leukemia in Down's syndrome (trisomy 21), suggests that leukopoiesis is governed in some respects by genetic material on chromosome number 21.

The finding of the Philadelphia chromosome in the following case enhanced the clinical diagnosis of chronic myelogenous leukemia.

Case 4.—(Fig. 8) M. H., a 12-year-old Negro female, was first seen on May 29, 1963, with the history of trauma to her abdomen in September of

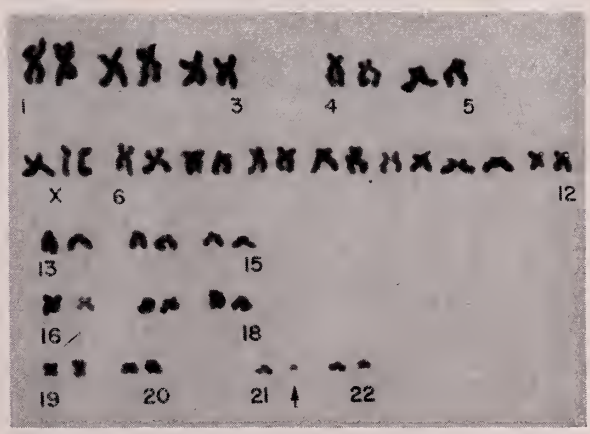

Fig. 8. M. H., Case 4, 12 years old, chronic myelogenous leukemia. Arrow designates Philadelphia chromosome.

1962, following which she had persistent left lower quadrant pain. Subsequently she developed weakness, anorexia, intermittent fever, and weight loss.

On physical examination her temperature was 100.4 F., weight 83 lbs. (25th percentile for 12½ years), and height 60 inches (50th percentile for 12½ years). She had splenomegaly and hepatomegaly.

A hemogram showed a white blood count of 267,000 with 22 per cent polymorphonuclear cells, 25 per cent stabs, 36 per cent metamyelocytes, 9 per cent myelocytes, 4 per cent basophils and 4 per cent eosinophils. Bone marrow aspiration was compatible with the diagnosis of chronic myelogenous leukemia. Cultures of peripheral blood leukocytes demonstrated cells containing a very small chromosome. Further study of photographed cells revealed the morphology of this small chromosome to be compatible with the previously described Philadelphia chromosome.

Conditions Associated With Uncertain Genetic Sex

Examination of the chromosomal pattern and the determination of nuclear sex should be an integral part of the evaluation of any patient with ambiguous external genitalia. While these studies are most important in early infancy in assignment of proper gender role, they may also be useful in certain older children so that parents' fears may be allayed and plans made for any necessary surgical procedures. Intersexuality is a term which has been used to describe these conditions in which there is discrepancy of sex structures with resultant characteristics of both male and female. The term hermaphrodite and pseudohermaphrodite have been used with similar meaning, with the gonadal structure dictating the choice of descriptive term. Thus the individual with both ovarian and testicular structures is called a true hermaphrodite and those with only one type, that is ovarian or testicular, are called female or male pseudohermaphrodites respectively. True hermaphrodites are rare, and the majority are sex chromatin positive with a normal female karyotype, however a few are chromatin negative with a male karyotype.

The following case represents a patient with ambiguity of the external genitalia in which chromosomal analysis was helpful.

Case 5.—(Fig. 2) W. C., a four-year-old white child was first seen in November, 1962. An imperforate anus had been repaired at birth, at which time it was noted that the phallus was quite small. He was considered to be a male with cryptorchidism.

Dr. Rouse, pictured, and Drs. Lockhart and Haggard are with the Department of Pediatrics, Divisions of Child Development and Hematology, at the University of Texas Medical Center, Galveston.

Physical examination revealed a normotensive white child in the 90th percentile in both height and weight for age. The pertinent abnormalities were limited to the external genitalia. The phallus, midline between a bifid scrotal structure, measured 1 by 2 cm. and was covered anteriorly with a prepuce. The urethral opening was at the distal end in normal position. The scrotal sacs were flat and positioned much like labia, but each sac contained a 1 by 1.5 cm. mass which had the consistency of a testis. Above this was a structure which appeared to be a mons pubis.

Sex chromatin was negative in two buccal smear preparations. The bone age was normal as was the intravenous pyelogram. Chromosomal studies revealed a modal count of 46 and a karyotype of a normal male.

Summary

It may be seen that chromosomal analysis, although still in its developing stages both clinically and technically, has many useful applications in medicine. It may be used as an aid in diagnosis of certain disease states (Down's syndrome, trisomy 13-15, trisomy 16-18, chronic myelogenous leukemia). With confirmation of the clinical diagnosis one can then better counsel the family as to the prognosis of the patient and the likelihood of involvement of future offspring. Further, when sexual ambiguity exists, the early determination of genetic sex allows the proper gender role to be assigned, and any necessary corrective surgical procedures to be planned. It must be remembered, however, that chromosomal analysis is a costly and time-consuming laboratory examination, and while it may be regarded as an integral part of the study of many patients, as with any laboratory test, the results obtained must be considered in relation to the clinical findings. Lastly, it is hoped that advancement of knowledge through chromosomal analysis will lead to the eventual localization of specific genes governing the development, growth, and function of the human organism, and thus aid in the prevention and treatment of such abnormalities as have been described here.

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Mothers Help with Nursing in This Children's Hospital

How can hospitalization be made less traumatic for a child?

The Boston Floating Hospital for Infants and Children believes it may have the answer in a unique arrangement which allows the patient's mother to become a "bed-side" temporary member of the hospital care team.

The hospital, which is the pediatric unit of Tufts New England Medical Center, is trying the new concept in a pilot study financed by a federal research grant. Miss Geneva Katz, R.N., administrator of the hospital, describes the study in the current issue of *Hospitals*, Journal of the American Hospital Association.

At Boston Floating Hospital, the child's mother is permitted to live in with the patient in one of four single rooms adjacent to a nursing care area.

Each room contains a crib or bed for the child, a studio couch for the mother, and a snack-stocked refrigerator, hot plate, and toaster for the mother's personal use.

The mother works with the nursing and medical staff in a close relationship, Miss Katz says, providing many of the services for the child that the mother would normally at home.

From 5 p.m. to 8 a.m., the mother assumes full responsibility for the child, calling the nursing supervisor only if necessary. The mother's constant presence lightens the nurses' load, lowering hospital rates $5 per day for the child, Miss Katz explains.

There is no charge for the mother's food or room, Miss Katz says. The father can replace the mother to give her time off.

The special units are apart from the regular wards to provide greater privacy. Children who are patients in the special section seem less restless, have better appetites, and are more content, Miss Katz writes.

Admissions thus far have been limited to medical and surgical, simple treatment, or diagnostic patients, although the hospital hopes to broaden its base of admissions to the units as confidence in the plan grows.

Genetic Counseling

R. L. Tips, M.D., Ph.D., Henry T. Lynch, M.D.,
and C. Wallace McNutt, Ph.D.

CPC

MODERN GENETIC COUNSELING stresses the clinical approach to family problems that arise from genetic implications of disease.[3] The psychologic and sociologic conflicts generated in affected families are seen in a wide spectrum of psychopathology.[2]

Quantitative assessments of reproductive performances among kindred have demonstrated a cessation of child bearing after the birth and diagnosis of affected offspring.[1, 4] Multiple emotional stresses have led to disruption of family harmony and to intrafamilial conflicts.[3]

A family with two children with cystic fibrosis exemplifies the myriad problems found in genetic counseling.

Case Report

The propositi were two siblings, an eight-year-old male and a six-year-old female, both with severe fibrocystic disease. Their family was chosen from among 15 families with cystic fibrosis children who have been included in genetic counseling studies during the past four years.

Both children were the products of uncomplicated pregnancies. Both were considered normal until they were approximately nine months of age when each developed a severe bout of bronchiolitis with subsequent frequent episodes of bronchopneumonia associated with respiratory distress, flatulence, and typical foul smelling, greasy stools. The older child remained undiagnosed until he was three years old when his nine-month-old sister developed her illness pattern. Respiratory disease (frequent bouts of bronchitis and bronchopneumonia) predominated their clinical picture until the older sibling reached five years, when diarrhea, malnutrition, and psychological problems became prominent. Final diagnosis was firmly established at three and five years of age when sweat electrolytes and duodenal aspiration studies were done on both children. The parents were given the report of a poor clinical prognosis for these children and adverse genetic potential for additional children.

The boy was sent to kindergarten at age five, but was advised to remain at home because of emotional immaturity, frequent fecal soiling of clothing, and respiratory distress. He quickly realized that frequent deliberate fecal soiling with the attendant unusual odors was an extremely useful means of controlling his parents and his environment so that subsequent schooling was impossible. Both he and his sister perfected this behavior pattern to the point that the parents were reluctant to take them out of the home or accept visitors. They hesitated even to obtain essential medical aid when respiratory infections became prominent.

The family was referred for genetic counseling by the family physician and at the suggestion of a similarly affected family who had received help in the counseling clinic.

A ten-year-old male sibling was in excellent health except for occasional attacks of asthma until three years prior to the initial interview. During this period the asthma became increasingly severe and was associated with mild diarrhea. His school work progressively deteriorated. His behavior at home became sullen and aggressive. He often refused to respond to his mother, so that deafness was once seriously considered.

An 11-year-old female sibling was in good health except for a mild nasal allergy, until three years prior to the present interview. Her personality had been pleasant, friendly, and outgoing, and her school performance in the first and second grades had been excellent. During the preceding three years her school performance and behavior deteriorated. She became withdrawn. Her nasal allergy intensified.

The mother was a 33-year-old housewife and college graduate, who had always been in excellent health. During the past three years she had complained of excessive fatigue, night sweats, insomnia,

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This clinicopathologic conference, provided by Baylor University College of Medicine, was edited by Dr. S. H. Frazier of the Department of Psychiatry at BUCM. Authors are Dr. R. L. Tips, clinical associate professor of psychiatry, Dr. Henry T. Lynch, resident in internal medicine at the University of Nebraska College of Medicine, and C. Wallace McNutt, Ph.D., associate professor of anatomy of the University of Texas Medical Branch.

The nature of genetic counseling problems is revealed in this conference concerning two patients with cystic fibrosis.

and a chronic hacking cough. Her physical examination was negative.

The father was a 34-year-old college graduate and a successful businessman. He had always been in excellent health, except for recurring complaints of diarrhea each spring, which he attributed to a pollen allergy. During the previous three years he had progressively withdrawn from the family environment, finding it "necessary" to spend more time with business problems.

The family history revealed a previously unsuspected consanguineous relationship between the parents in that the paternal grandmothers were sisters. It was noted also that the siblings of the parents have not produced any children during the past three years, so that kindred reproduction had essentially ceased with the diagnosis of the propositi.

Prior to the diagnosis of the propositi the parents had sought aid from several sources in an attempt to establish definitely the nature of the medical problem facing them. Their attempts had met with a frustrating lack of specific information, as the underlying genetic nature of the problem had not been defined. Upon receiving the diagnosis both were relieved. Neither had been interested in having more children, and therefore did not find the adverse genetic prognosis overtly disturbing.

During the family history interviews, both parents obviously comprehended the genic mechanisms involved in the disease. However, both were extremely disturbed concerning their interpersonal relationships during the past three years. Their sexual relationships ceased completely. Nocturnal arguments and accusations concerning "bad genes" in both families quickly ensued. The father felt that his overwhelming frustrations eventually led to his withdrawal from the family environment. The mother revealed that during the past three years she had become preoccupied with the mechanics of washing and bleaching the soiled clothing of the propositi. The resultant foul odors of this constant soiling did not seem to disturb her, but her preoccupation with cleaning and bleaching became so great that the two older normal children were neglected. Family meals had become irregular and, in general, the normal household routine had collapsed.

The mother became very critical of her husband's family. Her criticism eventually encompassed every aspect of their lives. She attempted to point out that his siblings had produced affected children also, even though the accusations were inaccurate. She believed her husband's mother was at fault for neglecting to have her own children's allergies treated. Eventually this criticism was also directed toward her own parents and siblings. Her misunderstanding of the symptoms of cystic fibrosis process led to friction in the entire family.

During the initial counseling interview of the family, the propositi became resentful of the procedures. They were restless and aggressive and continually created disturbances. The parents did not attempt to control their behavior. When instructed to be seated, the children immediately defecated in their clothing as an act of defiance. However, this was ignored. The parents became increasingly uncomfortable and distracted.

Discussion

DR. LYNCH: Previous investigations into the evolving discipline of genetic counseling have firmly established the need for a clinical approach to these problems. The manifold conflicts uncovered in the present counseling interview serve to substantiate and enhance the validities of these convictions. This family had become severely and progressively disturbed during the previous three years. The family unit (the propositi, the parents, and the siblings) had deteriorated to the point of complete disintegration of harmonious relationships.

Despite the complicated medical management problems of the propositi, the family had adjusted well and were able to function in a coordinated manner during the first five years of illness. Following the definitive diagnosis, these medical problems remained; however, the genetic aspects of the condition were introduced and imposed additional anxieties during a time when the parents were not overtly interested in having more children. The interjection of the genetic aspects served to intensify their emotional burden.

The parents began having nocturnal arguments, and normal sexual relationships ceased at that time. Recall that these disturbances were related to the genetic prognosis rather than to the medical prognosis, although the two cannot strictly be separated. Note both parents developed marked feelings of guilt and self-incrimination. They also made misunderstood accusations toward each other to the point that they were unable to communicate realistically. Their interpersonal hostilities gradually extended to and involved their relationships with their children. The

normal children were neglected, and their social behavior and school work deteriorated. Illnesses were magnified. The parents' attitude toward the affected children became overprotective and permissive.

Thus, the present family demonstrated the sequential disruption in adjustments that originate from the interjection of the genetic aspects of disease. Such implications produced unwarranted concern about reproduction which led to serious adversities in the interpersonal and psychosexual relationships of the parents. These anxieties, in turn, permeated the entire family unit and affected the motivational drives supporting family harmony. These altered drives were manifested as deterrents not only to the medical management of the propositi but also to the behavioral and social guidance of the normal siblings.

The next important problem encountered in counseling also is illustrated by this family. The added emotional burdens imposed by the genetic nature of the disease inhibit the proper medical management of the patients. The present strife-ridden family was practically controlled by the illness of the propositi. The parents had become incapable of maintaining an effective treatment program. Respiratory infections remained essentially untreated. The defiant encopretic behavior served only to magnify the parents' inadequacies. Allergic phenomena in the normal siblings were also intensified and uncontrollable.

Exploration of the psychodynamic undercurrents of the genetic aspects of disease in the family milieu in relation to the medical management will generally lead to a more effective program. This may be accomplished in a counseling atmosphere where the emotional conflicts of the family unit are taken into account. The parents are taught to recognize the real origin of the psychosexual conflicts and to understand how these permeate the family environment and extend into the kindred in the form of unrelated intrafamilial frictions. Solution of these psychological barriers emanates from a realistic understanding of these mechanisms. Once the severe psychological problems can be circumvented the medical problems can be successfully managed.

Another problem frequently encountered in counseling is the discovery of consanguinity in families. Usually all parents are disturbed to discover that they are related, even when only normal children have been produced. Such disturbances can cause feelings of guilt and distastefulness that may lead to disruption in psychosexual adjustments between parents and to unrealistic conclusions about reproduction. This in turn may alter the motivational drives supporting family harmony. These emotional burdens also permeate the entire kindred in the form of intrafamilial hostilities. Thus, consanguinity itself serves to produce emotional problems similar to those observed in genetic disease.

The discovery of consanguineous relationships in families where abnormal children have been produced constitutes an even more complicated problem. Not only do parents have the problem of medical management of the affected children, they also have emotional anxieties produced by the genetic nature of the disease, further burdened with the complicating implication of consanguinity. This additional burden can be a disastrous blow. However, in the proper genetic counseling atmosphere, the complicating influence of consanguinity, as well as the other problems already discussed, can be handled readily.

In summary, it is obvious that this family demonstrates the multifaceted emotional problems generated in families with genetic disease. The clinical geneticist who is investigating such a family must have clear insight into such problems and must undertake the task of communicating the significance and the origin of them to the parents in a manner that they might both understand and accept. This requires good clinical judgment and evaluation. With this in mind, one becomes more impressed with the folly of the cold, traditional, stereotyped approach to genetic counseling. Actually one is dealing with emotional content that is equated with the genetic phenomena, and the mathematical analysis during the initial interview would not only have been inappropriate but, more important, would have been severely rejected.

DR. McNUTT: Cystic fibrosis results from the presence of two pathologic autosomal recessive genes in an individual. In order to produce a recessive homozygote, each normal parent must carry a pathologic CF gene. Each parent must also have a normal gene at the CF locus. Other individuals who do not have the pathologic gene would

possess two dominant normal alleles at the CF locus.

Three types of individuals with respect to the CF gene are thus present in the population; normal, with both dominant alleles; carrier, with one normal allele and one CF gene; and affected, with two pathologic CF genes. Carriers can be detected only through the production of affected children. All affected children are produced by carrier parents since affected persons (homozygotes) with cystic fibrosis rarely live to reproduce.

Carrier parents can produce three types of children: normal, who have received only normal CF alleles; carrier or heterozygote, who have received a normal allele from one parent and a pathologic CF gene from the other parent; and affected individuals who have received a pathologic CF gene from each parent. Carrier parents are capable of producing these three types of offspring in the ratio of 1:2:1. Thus, each child produced by such parents has one chance in four of being either completely normal or of being affected, and one chance in two of being a carrier. These chance phenomena do not change from one pregnancy to the next. No matter what genotype the previous offspring have been, the next child has the same genetic potentialities as all of the siblings.

Because of the rarity of carriers in the population, it is unusual that two carriers will marry and produce affected children. Carriers are much more likely to occur in kindreds of affected cases than in the general population. Because of the familial aggregation of the carrier state, there is a high incidence of consanguinity in recessive disease; that is, a carrier is much more likely to marry another carrier if the person were related than if he were not.

The clinical geneticist must well be aware of the genetic mechanisms in the families that he investigates. He must always remain vigilant for possibilities of consanguinity among the parents. Frequently, as in this family, parents are not aware of these relationships.

Comment

During genetic consultations with this family the parents were led to recognize the myriad emotional problems that had enveloped their family. This was accomplished principally during the family and social history interviews, where the parents were permitted to describe the conflicts. Insight was gained rapidly by them, and they came to accept the existence of their interpersonal hostilities and could then communicate. During counseling sessions the emotional problems were gradually related to their concern about reproduction. When this finally occurred, they were then capable of comprehending the genetic biological phenomena in a more realistic manner. Such insight alleviated many of the feelings of guilt and interpersonal hostilities. The consultations were terminated after several visits when it became obvious that many adjustments were being made in the family.

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Somatic Equivalents of Depression

CLINICAL MEDICINE

SOME PATIENTS express depression by complaining of body malfunctions. This can be misleading. Sometimes, there actually is physiologic or autonomic change in body functions as a result of depression, but many patients complain when there is no serious disturbance in organ function.

Causes of Depression

The causes of depression offer some explanation of the relationship between somatic symptoms and the hopeless, depressed, and suicidal feelings of the typically depressed person. Three psychologic phenomena are relevant to the experience of depression.

First, the patient has suffered some type of *loss.* This loss may be of a person or thing, for example, the loss of a wife or child, money, or business; or the loss may be of a sense of self-esteem or personal invulnerability. A common example of this last is the sense of loss that accompanies the climacteric, the diminution in quality or frequency of erection in males, or coronary or other serious illness that limits activity and brings a man up against his own vulnerability to injury, disease, and death. This sense of loss occurs in a person who is vulnerable to depression because of earlier experiences or genetics or perhaps both.

Second, *anger,* primitive rage, develops in response to the loss.

Third, the anger is turned toward the self because of *guilt feelings* arising from a sense of personal responsibility for the loss because of errors of omission or commission. Anger also is directed at persons the patient believes share responsibilities for the loss. The patient places in this role those close to him and with whom he identifies—those he considers himself akin to because of shared feelings, interests, and personal qualities.

Turning anger on oneself results in feelings of worthlessness, hopelessness, and dejection, and constitutes the principal preoccupation of the typical depressed person. The anger toward self (expressed as self-blame for many real and fantasied events) is acknowledged by most patients, but the anger toward others is denied. That the anger and aggression toward others is in fact present is manifested in the way patients make their families and friends miserable with their complaints and bleatings, by their expression of a wish to die, and by suicide threats. Suicide is best understood in terms of Freud's explanation as the expression of a wish to die, a wish to be killed, and a wish to kill others. A dramatic illustration is provided by the homicide-suicide tragedies reported daily in the news media.

Thus it becomes easier to see how anger and self-destructive wishes may be suitably expressed by believing that some organ or body system is not functioning or is dead, and by manipulating some well-meaning but unwary surgeon into removing it. This maneuver (a partial suicide, according to Dr. Karl Menninger[2]) punishes the self, removes at least part of the body, and provides a new direction for anger—the surgeon. The initial relief is followed quickly by a return of symptoms, sometimes more intense than before, complaints to the physician, and demands for further surgery.

Depression manifests itself in several ways. Instead of saying they feel "blue," some patients come to believe that some organ or body system is not functioning or is dead. Many insist on drastic treatment.

JACK R. EWALT, M.D.

Types of Complaints

The most common area of complaint is the lower abdomen. The patient is preoccupied with constipation, flatulence, burning on urination, urgency, itching anus or perineum, painful intercourse, undependable erection, and an assortment of pains that, via the patient's exposure to medical history-taking and ladies' tea talk, quickly conform to textbook descriptions. The complaints offered by the patient have a persistent and urgent quality. They demand attention. He wants drastic treatment. During his first few reports the patient elicits sympathy and attention, but repetition of his complaints and the rather aggressive manner in which he presents them prove wearing to the family and tax the patience of the physician. The family expresses its anger by urging the doctor to do something, preferably something drastic and immediate. This device allows the family not only to express anger by assaulting the patient (via the surgeon) but also to assuage or conceal guilt about it for they obviously are concerned and trying to help. The physician may also fall in with this maneuver; his efforts to cure the patient and please the family may serve to discharge some of his own anger via manipulations and surgery that his quiet inner judgment tells him are unnecessary. After removal or suspension of function of one organ the symptoms recur, and a series of operations may follow, each more futile than the one before, developing into a cycle of loss and depression.

In another type of case, complaints center about the nose or mouth. A common manifestation is complaint of odors. The patient may believe that he stinks, or he may have olfactory hallucinations. The former is more common in depression, the latter in schizophrenia and sphenoid ridge tumors. A variant type suffers preoccupation with appearance and requests plastic alterations of the face that are not justified by the patient's actual appearance. Nose bobbing, sinus drainage, septal surgery, and similar manipulations afford only temporary relief; the return of the symptoms produces subsequent hostility toward the surgeon because the patient comes to believe (and sometimes correctly) that he is worse off than he was before. Patients may pick at their noses and produce infections and crusts that require local treatment; the physician is faced with the dilemma of giving treatment that the patient will complain about. In a destructive cycle of cause and effect the patient will continue to aggravate the condition, fail to get well, and continue to use the physician as another outlet for his complaints and hostility as well as a source of support and care.

Dentists see similar patients who complain of odd tastes, burning, and other disagreeable oral sensations. The unwary dentist may remove the teeth and find himself in a losing battle over dentures. Each attempt to make the plates fit better (or taste better) aggravates the symptoms. One such case will often suffice to make a dental surgeon suspicious of people who have potential for repair but who seem overanxious for extraction of teeth.

Similar cases involve indigestion and gallbladder diseases. Patients gulp a vast amount of air which swells, causing complaints of pain and near starvation and expressions of wishes for relief or death.

Some alcoholic patients drink to hide depression, and while under the influence of

liquor do not show the typical depressive symptoms. They tend to try to overcome drinking but there appears no way to differentiate them from other addictive drinkers. If the physician succeeds in getting them off alcohol, the depression becomes more manifest. At this point they need emotional support and treatment from the physician. The therapy has not succeeded if the patient, although sober, commits suicide. In other words, if one takes away his medicine (the alcohol) one must provide a substitute such as Librium and liberal doses of personal interest.

Another type of case is the long-suffering wife of a chronic alcoholic who may find she cannot stand her husband as a vigorous, non-dependent, non-drunken male. She then gets depressed and may come for treatment, or she may entice or seduce her husband into a relapse (without being altogether conscious of what she has done).

A mother of a schizophrenic daughter became depressed when the daughter experienced a good remission. The daughter had a relapse, and the mother recovered. The daughter had a second remission which she maintained, and the mother developed a severe chronic depression that resisted the best efforts at psychotherapy and electric convulsive therapy.

In cases of this last type the illness of a family member seems to be an adequate outlet for the patient's anger and guilt feelings. The patient does not feel depressed so long as the relative remains dependent, troublesome, and an object of guilt. The loss of the other person's dependency is experienced as a loss of his own usefulness and self-esteem and he becomes depressed.

Diagnosis

The somatic symptoms of the depressed patient are much like those of neurotics treated in physicians' offices, but treatment is significantly different. Therefore, it is necessary to have some differential diagnostic points.

First, the symptoms are offered with an intensity and persistence that far exceeds that of the ordinary neurotic. The symptoms are used not only to gain sympathy but also as a means of beating on friends and relatives. The patient makes one suffer with him. One also can detect a note of anger in the patient's manner of relating to the physician and to his own family. He usually will withdraw interest from former hobbies and community interests. He is occupied wholly with his symptoms. Sexual function and interests are decreased from the previous norm for the particular patient. He often has a weight loss and some anorexia, in contrast to the neurotic with gastrointestinal complaints. He usually will have disturbance in his sleep pattern and some insomnia. Many neurotics complain of insomnia but observation by nurses or family members fails to confirm the complaint. Few people with depressions, either typical or with equivalent somatic complaints, enjoy their normal sleep rhythms. Early morning awakening occurs frequently, but whatever the type of insomnia the total amount of sleep is less than normal for the particular person.

The patient's attitude toward the physician also may be different. Neurotic patients as well as the depressed ones do not want to get well; they want to maintain their dependence upon the physician. The ordinary neurotic prefers to remain on friendly terms with the doctor; as long as one makes no demands for recovery he remains pleasant and cooperative. The depressed patient also resists recovery because he wants to remain dependent, but for him love is not enough. He wants to suffer and to make the physician suffer with him. He will be difficult. He will request and perhaps demand service at inconvenient times. He can snarl up the physician's schedule with amazingly little effort and can make one feel guilty for complaining.

The physician's attitude toward the patient also is an important diagnostic clue. Neurotics may bore, annoy, or irritate the physician, but they seldom if ever enrage him. The depressed patient will make the physician angry by subtle baiting and hostility, and then the physician will feel guilty about his own anger.

Treatment

The depressed person must be treated; if not, he will be nibbled to death by a series of operations or manipulations, or he may commit suicide. Some of these patients may be referred to psychiatrists, but many gen-

eral practitioners will have to manage them at home.

It is basic to the care of the patient that he believe that he has someone he can trust and cling to and to whom he can complain without being rejected. The physician will become exasperated with the complaints, but remembering that the anger, complaining, and generally miserable state are due to the patient's illness may make them easier to bear. He may wish to see the physician often, or to telephone. This will be helpful to him, and the physician need not spend long periods of time with him. He wants to know that the physician is there, available, and still interested in him. Some patients will call often at first, but as they become convinced that the physician can be reached their calls decrease and may stop altogether. Scheduled interviews may also be short during the acute phase of the illness; 15 minutes is long enough. Interest in him and assurances that his suffering is due to his depression and that he will recover should be the central theme for such sessions. At first he will talk mainly of his complaints, but in time he will speak of his family, his boss, and his friends. In the beginning he will deny any anger toward anyone, but one will be able to pick up signs of it here and there. As he discovers that the physician is not critical of his anger he will express it more freely. (At this stage he will also begin to express anger or rage more overtly toward his family, and they may complain that he is getting worse.) As he develops confidence in the physician and realizes that he can be angry at his losses without disaster to himself or others, and that he can survive in reasonable comfort despite the loss, he will feel relieved, and the symptoms will begin to abate. The first improvement requires more subtle handling than any other part of treatment. The patient's suffering and the physician's feelings of frustration at slow progress along with his damaged sense of therapeutic omnipotence all tend to make him greet the first signs of improvement with joy and enthusiasm. However, if the physician reveals the extent of his pleasure, the patient will promptly get worse. He may conclude that the physician was not interested in him as a person after all, but only in him as a potentially successful case. The proper tone is one of pleasure that the patient is suffering less. He should be reminded that he will eventually get well and warned that recovery from depression is often punctuated by short periods of partial relapse, which are no cause for undue concern.

A third part of the treatment is directed toward symptomatic relief. Older treatments are still useful. Use of barbitals for sleep and one of the amphetamines upon arising and at midday can provide substantial relief for some of the symptoms. This drug regimen is cheap, relatively safe, and therefore old-fashioned. Imipramine, Tofranil (Geigy), and the closely related amitriptyline, Elavil (Merck Sharp & Dohme), may be helpful in some cases. Monoamine oxidase inhibitors seem to work in some climates, but a carefully controlled study in Boston of two such inhibitors, Nardil and Marplan, showed that they were no better than placebos.[1] Drugs of the so-called energizer group must be given for periods of up to three weeks before they can be deemed ineffective. Some investigations of administration of enzymes to speed up and augment the effects of drugs have suggested that there are interesting possibilities, but these studies are still in the experimental stage.

Some current opinion holds that the tranquilizers, especially meprobamate, may make depressed patients worse. This is true in many cases, but an occasional patient with many anxiety symptoms will be relieved with Compazine (Smith, Kline & French), reasonably small doses of chlorpromazine, or meprobamate. Patients who show much agitation and anxiety as part of the depression may obtain considerable symptomatic relief.

Electroconvulsive therapy is the treatment of choice in severe suicidal and in severely agitated depressions. It is drastic, but it works—and sometimes dramatically. However, in patients with somatic equivalents, E. C. T. is less effective. No one knows why this is, but empirical observation indicates that any symptomatic relief is short. The immediate relief followed by a recurrence of symptoms (and often with memory and muscle complaints added) is similar to the relief with subsequent recurrence that follows surgical intervention for the symptoms.

Supportive psychotherapy and judicious use of medications is the treatment of choice. Experience also suggests that the family, especially the spouse, will require some help not only in understanding the patient's problem and how to cope with it but also in gaining some understanding of his or her contribution to the patient's symptom. A social

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worker may be most effective in work with the spouse, or he or she may be referred to a colleague. There may be circumstances in which a physician will be seeing the spouse as well as the patient, but this can be complicated since they may get him in the middle, vying for his support in a marital struggle. Some physicians handle such situations by an occasional joint interview.

One may ask what is the suicidal risk in these patients? As in all depressions, the wish to die is present. In some patients the somatization of the anger and self-destructive tendencies offer some protection against death of the whole man. An organ may be lost instead of the total organism. However, this is not a safe generality. The prediction of suicide is not a precise art. The same warning signs and the same protectors apply as in the more typical case of depression. A history of impulsive acts, excessive use of alcohol, and a lack of interested friends and loving family require particular caution. The presence of a loving and attentive spouse, no more angry or hostile than the average, is a protection. When the patient becomes convinced that his physician is reliable and he can depend on him, this also is a protection. Physicians are prone to assume the patient trusts them before he actually does. The patient has developed confidence in his physician when he can discuss his anger at fate, at the physician, and at members of his family. Another protection against suicide is the physician's instant availability. This can be by telephone. A patient should be encouraged to call if he feels too lonely or too desperate. Knowing he can call is often enough. Use of this method must be sincere. If the patient called and the physician refused to talk to him, a suicide attempt could be precipitated. As a further safeguard, the name of a colleague may be left and arrangements made for him to take the call only if a patient fails to locate his physician. If there is doubt about the suicide risk, one should play it safe and have a dependable special nurse or companion. They must be *with* the patient. To be in the hall when the patient is in the bathroom or to go down the hall for a phone call or medication or coffee offers no protection at all. Having tricked a nurse into leaving for a moment, the patient will seize the chance to go out a window, cut his throat, or set fire to clothing.

With symptomatic improvement achieved, most patients lose interest in continuing therapy. If they want more treatment, they should be referred to a psychiatrist for psychotherapy. The general practitioner can be an effective therapist as long as he stays with the immediate issues and with the management of symptoms. Attempts to dabble with unconscious material or to change the patient's basic orientation to life are for those who are especially trained to do it. Ineptitude in this area may precipitate another psychosis or a suicide attempt.

Most patients will get along well if managed according to the preceding suggestions. Treatment may require a year or more, but if it is nonpunitive and supportive the prognosis is good.

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COMMENT

The author has lucidly presented the dynamics of a depressive reaction. The relationship to anger is well brought out, though perhaps overemphasized since other factors also enter into the production of these reactions. It should be brought out that most depressive reactors are anxious to go along with whatever therapeutic measures are suggested to gain relief. The suggestions on telephone management are particularly good.

—Edgar S. Ezell, M.D., Fort Worth.

A Case of "Psychosis" from Drugs

THEODORE H. GREINER, M.D.

Hallucinosis and distortion of behavior as a result of improper use of drugs is a growing problem. Diagnosis may be difficult, and the patient must be managed as an acute psychiatric emergency.

CASE REPORTS

A 25-year-old college student was brought to the emergency room of the Houston Veterans Administration Hospital by a friend. He had been sent there after being examined at a local general hospital. The friend said that the patient's wife returned from work and found her husband incoherent, stumbling around the apartment, and alternately laughing and crying. The friend also told doctors that the wife had "got lost" while the patient was being examined at the local hospital.

The man was admitted about 7 p.m. with the tentative diagnosis of acute schizophrenic reaction. He was mumbling incoherently, did not respond to questions, and seemed to be hallucinating. Particularly, we suspected visual hallucinations, for he would feel around on the floor as if he were trying to pick up some object. At other times he was resistive, became combative, and rammed his head against the glass of the nurses' station. Muscular incoordination, delirium, and dilated fixed pupils, together with the mysterious disappearance of the patient's wife, suggested that the man might have attempted suicide with drugs. Medical consultation produced no additional findings. The internist, apparently impressed by the patient's resistance to examination, held schizophrenic reaction to be the most likely diagnosis.

Several hours after admission, the patient's wife arrived and said that the patient had never drunk alcoholic beverages or taken drugs, but that they had been involved in a marital battle for two weeks. In fact, the patient had bought some bullets for a pistol, and the wife considered this a threatening gesture. The patient apparently had skipped classes all day while his wife worked, and had written a long letter to his parents which included the statement, "Whatever happens is not my wife's fault."

The wife was asked to search the apartment for signs of medication. She did so, and found in the waste basket a box which had held Sominex tablets. Sominex is a heavily advertised sleeping capsule compounded for over-the-counter sale.

Since the patient's clinical appearance was compatible with acute scopolamine poisoning (scopolamine is a major ingredient of Sominex) he was placed in a quiet room and observed. Eight hours after admission he responded to questions, and reported no more strange visions. After 24 hours he showed no signs of poisoning except the anxious tension and dilated pupils. Later, he estimated that he had taken 35 Sominex tablets about five hours before being examined in the emergency room.

Discussion

In the emergency room, it is not a rare experience to see a patient who is incoherent and hallucinating. Diagnosis and management must, at that point, be on an emergency basis. If no history accompanies the patient, there is a tendency to diagnose an acute schizophrenic break, or delirium tremens if drinking is in evidence. Our emergency room psychiatric consultant settled quickly on a schizophrenic break, although in retrospect, the "feel" of the patient was more of acute delirium.

Later, when the history was available and the toxic agent suspected, a check of standard pharmacology texts yielded a laboratory test to confirm the diagnosis of poisoning with scopolamine—a drop of the patient's urine in a cat's eye should dilate the pupil. This, of course, was not routine, and by the time arrangements could be made to get a cat, the patient was coherent and had told us what he had taken.

There is increasing concern about the uncontrolled use of drugs that produce "kicks" by altering the mental state. Recent editorials emphasize the danger of intense hallucinogenic agents without adequate medical supervision[3] and deplore the publicity these drugs have received.[1] Control measures

which have restricted the availability of LSD and other classical hallucinogens could force the thrill-seeker to turn to other drugs which mimic the LSD effect. Indeed, deliberately self-induced hallucinations have been reported in three young people experimenting with an asthma remedy (as in the present case, a patented drug preparation containing scopolamine and other related compounds). However, the patients were still coherent when first examined, and were able to aid in their diagnoses.[2]

If the acute hallucinated state has been produced unknowingly, diagnosis will depend on more than winning the patient's cooperation. The availability of history from another source, and alertness to details that do not ring true (such as dilated pupils in this case) can be critical points. Since sleeping medicine is always a favorite way of escape, over-the-counter preparations can be expected now and then to be used in suicide attempts. They can result in hallucinations and psychiatric emergencies if the scopolamine group of drugs is chosen. As a further complication, iatrogenic hallucinosis has been reported attributable to some anticholinergic compounds used in ophthalmology.[4] These reactions were thought to be idiosyncrasies, prone to occur in young and/or brain-damaged persons and not likely to produce much diagnostic difficulty.

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Summary

A case report of drug delirium caused by Sominex is presented, and discussed as a diagnostic difficulty (misdiagnosed as acute schizophrenic reaction), as well as a potential problem from readily available drugs which alter the mental state in such a way as might attract thrill-seekers.

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Glue Still Tacky; Sniffers Not Wacky?

The teenage glue sniffing fad may soon be passé—not because teenagers aren't what they used to be, but because the glue may be changed.

The Hobby Industry Association of America, reports *The AMA News* (June 22), has announced development of a new chemical to replace intoxicating solvents now used in model airplane glues.

Expected to be ready within a year, the new ingredient is hailed as a possible solution to the problem of glue sniffing.

The Athletic Toll

A. ROSS DAVIS, M.D.

REVIEW ARTICLES

ATHLETICS and the treatment of injuries resulting from participation in athletic endeavors are subjects which have become popular in recent years. With a renewal of interest in athletic injuries in this country, new organizations must spring up to perpetuate this interest.

The National Athletic Trainers Association was founded in 1950, and held its most recent meeting in Cincinnati in June, 1963. The meeting was well attended and the program included educators, physicians, physical therapists, and athletic trainers.

In May, 1963, the American College of Sports Medicine held its tenth annual meeting. The American Medical Association has set up a special conference on athletic injuries which has held its fifth meeting. In June, 1963, Rhea H. Williams, Ed.D., Interscholastic Athletic Director, Bureau of Public School Service, set up a conference on "Safety in Athletics," which was attended by physicians, high school coaches, trainers, and administrators. It was held in Austin.

In light of this upsurge in interest regarding injuries in competitive sports, we should be able to draw certain conclusions as to exactly "What is the athletic toll?" Are the scales balanced in favor of progressive increase in competition in sports activities, or is the toll so great that only regimented programs of physical fitness should be conducted in the school system?

It has been only within the past few years that we have been keeping score to try to determine precisely how many and how severe are these injuries in competitive sports. Gonzales[2] wrote on fatal injuries in competitive sports in 1951. Thorndike[6, 7] first reported on athletic and related injuries in 1940. He reported that in all Harvard varsity athletics over a 17-year period, 3,453 injuries occurred among 14,375 contestants, or approximately a ratio of one injury to every four contestants. He also points out, however, that a majority of these injuries were not serious, approximately two-thirds being sprains, strains, and contusions. Schneider,[5] in 1961, wrote on "Serious and Fatal Football Injuries Involving the Head and Spinal Cord."

On Jan. 6, 1964, the National Collegiate Athletic Association reported through its 32nd annual survey of football fatalities prepared by Dr. Floyd Eastwood,[1] and later revised to the author by personal communique, that during the 1963 season there were 2 deaths in college football, 1 in professional football, 12 in high school football, and 1 in unorganized sandlot football in the entire United States. This has been estimated to be approximately 1½ deaths per 100,000 high school students, which may seem high initially; however, when this is compared to approximately 21 deaths per 100,000 high school students from automobile accidents, the figure becomes less alarming.

Let us now look at the situation in Texas. During the 1962 season for which the figures are complete, there were 925 schools participating in league football, with a total of 32,088 boys participating. In the reporting schools there were 2,181 boys listed as having received injuries, which made a total of 10 percent of the 21,817 boys participating in football in the reporting schools. Another interesting figure from the Texas High School Coaches Association is that the number of injuries incurred was approximately three to one in games as in contrast to practice, which indicates that competition in-

A complete analysis of the physical and monetary toll of athletic injuries in Houston schools, and general survey of injuries at state and national levels are presented.

creases the element of risk. There were no deaths reported in Texas during the 1962 season, but there were two deaths during the 1963 season.

Now, let us look at a local situation. The Houston Independent School District kept close records for the fiscal year from October, 1962, through August, 1963. Through the cooperation of Dr. Austin E. Hill, medical director, and Mr. Horace Elrod, director of athletics, these figures were made available for this report. Mr. Curtis Rose, supervisor of athletics, has kept these statistics throughout the year, and provided the author with data that are as complete as possible.

There are 45 junior and senior high schools participating in competitive athletics. Table 1 indicates the number of youngsters participating in the various sports in the Houston school district. Football commands the largest number of the 13,115 total participants. Of 1,378 students who reported injuries, 603 (47 per cent) were treated in the two training rooms of the stadium. In all probability, this is a healthy figure as it is indicative of the fact that these youngsters reported in for treatment of all of their minor injuries as well as the serious ones.

There were 775 youngsters sent to physicians, which again speaks very well for the medical care these youngsters received. This indicates that in no instance was there any hesitation about obtaining adequate medical care for the athlete. The junior high school group comprises the age bracket of 13 to 15 years, and the senior high school group comprises the age bracket of 16 through 19 years.

TABLE 1.—*Number of Boys Participating in Athletics in Houston Independent School District.*

Sport	Junior High Schools	Senior High Schools	Total
Football	2182	1446	3628
Basketball	1683	650	2333
Track	2055	855	2910
Spring football	509	1325	1834
Baseball		721	721
Swimming	1034	327	1361
Tennis		137	137
Golf		191	191
Total			13,115

Reporting: junior high schools, 18 white, 9 colored; senior high schools, 13 white, 5 colored.

TABLE 2.—*Injuries, 1962-1963.*

Head		Neck		Shoulder		Arm		Hand		Body	
Teeth (97 boys)	122	Fracture	2	Contusion	38	Contusion	5	Contusion	5	Contusion, chest	15
Eye cut	41	Dislocated vertebra	2	Dislocation	10	Cut	9	Cut	14	Contusion, rib	18
Eye contusion	11	Contusion	14	Fracture	14	Cleated	3	Fracture	6	Cinder burns, stomach	4
Eye operation	1	Bruised trachea	2			Broken	18	Fractured fingers	16	Back strain	17
Nose cut	12					Dislocated elbow	4	Dislocated fingers	4	Contusion, kidney	4
Nose bruise	6					Contusion, elbow	5	Contusion, fingers	10	Bad kidney	1
Nose fracture	5					Chipped elbow	2	Finger cut	4	Fractured ribs	2
Lip cut	35					Cut elbow	2			Contusion, testicle	8
Tongue bite	4					Contusion, wrist	5				
Ear contusion	3					Dislocated wrist	3				
Ear cut	1					Broken wrist	5				
Face cut	10										
Chin cut	8										
Jaw contusion	4										
Jaw fracture	2										
Concussion	36										
Heat exhaustion	27										
Parietal craniotomy (arteriogram)	1										

Hip		Thigh		Knee		Leg		Ankle		Foot	
Contusion	12	Contusion	15	Broken	3	Broken	9	Broken	8	Broken	5
Dislocation	1	Operation	2	Contusion	41	Contusion	16	Dislocated	2	Cleated	6
				Cut	1	Cleated	21	Sprain	16	Contusion	8
				Cartilage removed	27	Cut	8	Severe sprain	10	Spiked	12
						Spiked	4	Cleated	2	Broken toes	5
						Baker's cyst	1			Contusion, heel	1
						Severe leg injury (hematoma)	1			Blisters	5

HOUSTON INDEPENDENT SCHOOL DISTRICT

DEPARTMENT OF ATHLETICS

Houston, Texas

PARENT'S APPROVAL—Participation in Athletics

I hereby certify that_____has my approval to play

at home or away from home on the athletic teams of the_____ High School as follows:

FOOTBALL	TRACK	OTHER SPORTS
BASKETBALL	BASEBALL	

(Parent will draw a line through sports or events not approved.)

With the distinct understanding that the Houston Board of Education and the employees of the Houston Board of Education assume no responsibility for any accident or injury as a result thereof, except the rendering of first aid treatment and the services of an assigned team physician, semi-private hospital care, and diagnostic facilities when recommended by him. These services shall be deemed to completely discharge any and all responsibility resulting from any athletic injury.

Date_____ Signed_____
(Parent or Guardian)

Address_____ I certify that this release was signed in my presence.

Telephone_____ Signed_____
(Principal or Notary)

(This form to be signed in duplicate. This permit must be received by the Coach before the individual will be allowed to practice for any sport. The original to be filed in the office of the high school attended, the duplicate is to be sent to the Extramural Athletic Office, 3800 Cullen Boulevard.)

Fig. 1.

Table 2 breaks down the injuries into the areas of the body that were involved. The two most important facts to consider in this regard are: (1) there were no deaths as a result of athletic injuries, and (2) there was only one injury that could be considered a threat to life. That one injury resulted in a craniotomy being performed as a result of a blow on the head; the case turned out satisfactorily. It is noteworthy that a large number of teeth were injured; this probably could be eliminated by the cooperation of players, parents, and coaches to assure that all players wear proper mouth pieces.

The athletic toll must be considered not only from the standpoint of injuries, but also from that of the cost for the care of these injuries. The Houston Independent School District is unique in the manner in which the costs for care of athletic injuries is underwritten. The parents of the participating youngsters sign an approval slip (Fig. 1) before the student is permitted to participate in a competitive athletic program. They are further asked to sign a statement that they would be willing to use their family health and accident insurance to defray expenses of medical and hospital bills for their son if he is injured while participating in interscholastic athletics and if he should require medical or hospital care. The school system has assumed a moral responsibility for defraying the cost of all medical care over and above the insurance that the parents carry as individuals on their youngsters. The schools do not maintain a group insurance program on the athletes. The cost of the expenses of medical care is maintained through the use of money obtained from gate receipts received during the athletic programs. The cost of maintaining this type of excellent medical care for the fiscal year of October, 1962, through August, 1963, was an overwhelming $49,230.72. Nevertheless, this figure was less than it had been in some previous years, because a more diligent effort was made to request parents to utilize their own insurance insofar as possible. The family's personal insurance accounted for an additional $11,740.52, which the school system did not have to pay. The total cost then, for one year for 45 participating schools, was $60,971.24 to underwrite the care of athletic injuries.

Table 3 shows allocation of the bills paid, and Table 4 illustrates the extent that personal insurance policies of the injured athletes underwrote the entire program.

TABLE 3.—*Bills Paid for Athletic Injuries.*

Ambulances	$ 85.50
Anesthesia	117.50
Brace shops	154.50
Clinics	5,709.85
Dentists	8,877.00
Doctors	11,320.54
Hospitals	10,317.48
Neurosurgeon	1,310.00
Nurses	295.00
Oculists	149.00
Orthopedic	10,333.76
Pharmacies	374.59
Plastic surgeon	75.00
X-ray	111.00
Total	$49,230.72

We must consider the other side of the scale as it is the author's belief that the advantages of participating in this program outweigh the toll, both in regard to injuries and in regard to the cost of these injuries. Many cliches can be quoted such as "Healthy bodies and healthy minds are inseparable," and "Everyone must learn the value of team play." Certainly there can be no serious doubt that having youngsters participate in an organized sport and allocate their afternoons to practice in preparation for this sport, rather than spending their afternoons in leisure and in the corner drug stores, far outweighs the monetary costs of a program such as this. The building of character, of respect for authority, of strong bodies, and of a desire to excel certainly contribute to molding the individual participants into better citizens. Aside from all of this, O'Donoghue[4] has pointed out that there are certain material benefits for the athlete in our present educational system that have much importance. He has stated that "many a young man goes to school on his athletic ability." I would like to illustrate this point even more emphatically. During the fiscal school year discussed herein, there were 78 athletes who received scholarships in all sports from the Houston Independent School District. This number may not seem very large until it is multiplied by the six grades that participate in this program. Roughly, 450 boys participating in this program, or 3.4 percent, can be expected to receive athletic scholarships, and it is a proven fact that many of them will maintain a high scholastic average. Many of these youngsters are also qualifying themselves for a permanent career in related fields.

Novich[3] has pointed out that "emotional needs are satisfied, physical skills are developed, and valuable lessons in social living are learned" in contact sports. Even more important, however, is the basic fact that youngsters are going to compete with each other and must be supervised. This is graphically pointed out by the survey of football fatalities[5] which shows that over the past 32 years, 22.8 per cent of all the fatalities directly resulting from football have occurred in totally unsupervised (sandlot) situations.

TABLE 4.—*Amount Paid by Personal Insurance Policies of Injured Athletes.*

Ambulances	$ 9.50
Anesthesia	57.75
Doctors	1,116.90
Hospitals	6,393.94
Oculists	127.00
Orthopedic	3,960.43
Plastic surgeon	75.00
Total	$11,740.52

In summary, the statistical analysis of the participants in competitive athletics in the Houston Independent School District points out the merits of its program. Although we can anticipate that there will be certain injuries which will require treatment, these injuries taken overall are not so severe as to negate the obvious advantages of participating in such a program.

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An East Texas Pioneer

W. D. THAMES, JR.

HISTORICAL ARTICLES

DR. JAMES HARPER STARR arrived in Nacogdoches, Texas, on Jan. 17, 1837. Under unusual circumstances, he had come a long way to reach this place and start a remarkable career as a Texan.

James Starr was born in New Hartford, Conn., Dec. 18, 1809. He grew up in various parts of the North, was graduated from an academy in Worthington, Ohio, and was a member of the first class of the Reformed Medical Society of the United States of America. He quickly decided to move to Georgia with some friends from school. He began his first practice there in McDonough and Pleasant Grove, met and married Harriet Johnson, and continued to practice for about three years. During this time he developed a lasting interest in politics, probably influenced greatly by a younger brother, Franklin Starr, who had a law practice in a nearby town. James served as postmaster for awhile. He also became the executor of his father-in-law's estate—no mean financial chore—and learned to be an expert in ways of finance.

Franklin Starr became dissatisfied, moved to Texas, and helped Sam Houston in the Texas Revolution. He wrote encouraging letters to James, who by this time had become involved in some business operations which had not turned out well and partly for that reason was interested in migrating to Texas.

In April, 1836, Dr. Starr, his wife, and faithful slave, Tempie, left for Texas in a covered wagon pulled by two horses. They had about a two-year supply of clothing and $1,300 in money. It was a long and devious trip—overland to New Orleans, thence up the Mississippi and Red Rivers to Natchitoches, La., by boat, and then again overland to Nacogdoches. There they were reunited with Franklin and his wife and family. Dr. Starr established a thriving medical practice, and his brother built a good law practice. They became pillars of the community and were soon assuming many duties as political servants. Late in 1837, Franklin became ill and died with fever, therefore, Dr. Starr assumed the responsibility of the estate of his brother.

After Franklin's death, Dr. Starr felt that he might want to leave Nacogdoches. He looked around but found no more promising area, so he came back, built himself a log house, and became chairman of the board of aldermen. He also took time for jury service in the various courts of the area, and in 1838 he became president of the local board of commissioners for Nacogdoches County in the General Land Office. He had wide experience on dealing with people and running down fraudulent claims during his ten years as land commissioner.

In August of 1838, Vicente Cordova instigated his Mexican revolution and a mild Mexican rebellion in East Texas. Brig. Gen. Thomas J. Rusk, commander of the Texas Army, appointed Dr. Starr as acting surgeon general. Cordova anticipated the aid of the Indians in his rebellion, but this never materialized and the rebellion ended at the battle of Kickapoo.

Dr. Starr became an ally and great friend of Mirabeau B. Lamar and General Rusk, and was active in Lamar's race for the presi-

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Dr. James H. Starr served Texas in many ways. He was active in politics, banking and finance, and land ventures, but he never forsook the rigid integrity of his first love—the practice of medicine.

dency of Texas in 1838. Lamar appointed him to be Texas' Secretary of Treasury in 1839 as a reward for his efforts. He took a very active interest in the government and became convinced that Lamar and his group were the ones who were most honest and sincere and had the welfare of the Republic of Texas at heart. Starr's zealousness and honesty had endeared him to Lamar, and later he introduced a bill that created a South Texas county named in honor of James Harper Starr.

In 1840, Starr resigned his post as Secretary of the Treasury and returned again to practice medicine in Nacogdoches. He gave his full time to that cause because he believed that a diversity of interest impaired a man's professional fitness. He acted as family physician to the old elite families of the Nacogdoches area, but never refused treatment to anyone, whether slave or free. His practice ranged from delivering babies to fixing teeth and setting limbs. He rather disapproved the extravagant use of some of the medicines peddled in his day. The principal drugs that he prescribed were camphor, Epsom salt, calomel, ipecac, prepared charcoal, rhubarb, quinine, snakeroot, cream of tartar, sweet oil, flaxseed, and senna. Later he began to use cod liver oil and morphine or opium. He made his charge according to the distance he had to go to see his patients and the cost of the medicine he administered. Many patients paid him well, but much of his service brought no financial returns; some people simply ignored their bills. For this reason he was forced to carry on a real estate business on the side, despite his desire to devote all of his time to medicine. He became increasingly annoyed by patients' refusal to assume the responsibility of paying him for his work. Finally, in the 1840's he reluctantly abandoned the practice of medicine. He decided that his land business, which gave him more money, took so much time that he could not devote enough time to his practice.

About this time he also became engaged in banking, and was the first real banker in Nacogdoches. By astute business management, he developed a thriving bank and real estate business, and became quite wealthy. He remained actively interested in the affairs of the Republic, and advised different officials in various ways and manners. He became deeply involved in the presidential election of 1841, supporting David G. Burnet because he thought Sam Houston had abused Nacogdoches citizens. After Houston's election, he and Starr became bitter enemies, and Houston even tried to sue him and take away his Nacogdoches homestead. Starr had able support from Thomas J. Rusk, James P. Henderson, and other important men, and thus Houston's suit was never successful.

Actively interested in education in East Texas, Starr was instrumental in establishing and maintaining Nacogdoches University, one of the first universities to be established in Texas. He raised the money by public subscription and caused the building to be erected about 1845. He was active and influential in urging the Republic of Texas to accept annexation to the United States. After annexation, he centered his interest in his land agency, and also was instrumental in bringing railroads into Nacogdoches before the Civil War.

In 1860, a true Southerner by now, he was still in politics and was against the Abolitionists. He had slaves of his own and, although he did not know Abraham Lincoln personally, he was afraid that the candidate was too radical, and voted against him. Although he was unhappy with the results of the election and the events that followed, he fought against the secessionists; when it became obvious that he could not win this battle, he became involved in preparation for common defense. His words in this situation were, "We act now upon the principle that one's house is on fire. We must unite in efforts to extinguish the flames without waiting to inquire where or why the fire originated." Disabled by a crippled knee and too old to serve in combat during the war, he filled many civil positions and assisted in securing supplies for needy soldiers. Depression and drought caused hard times in 1861, and Dr. Starr was deeply in debt. He turned his home in Nacogdoches into a workshop during the War, where women spun thread and made cloth.

He was appointed postal agent for the West by the Confederacy and had to move to Marshall, Texas, to assume this office. Deposed from this position and completely penniless at the end of the Civil War he then returned to Nacogdoches.

In the postwar period he continued to make his living in the land business. Even

Dr. W. D. Thames, Jr., is in general practice and surgery in Lufkin.

in his advanced years, he was often named as executor of the wills of his friends, and he supervised the cases of Lamar, Albert Sidney Johnson, and J. P. Henderson, among others. He still had charge of all the Nacogdoches University land, also.

A staunch defender of the rights of the Negro following liberation, he fought the practice of assessing them severe penalties for minor crimes. In 1870 he was thoroughly dissatisfied with his land business in Nacogdoches, and moved back to Marshall where both railroads and telegraph lines were available. He and his young son, Frank, established the first bank in Marshall. Dr. Starr's ardor for education had not ceased, and soon after arriving in Marshall he became a trustee for the University of Eastern Texas. He was active and instrumental in the establishment of the University of Texas at Austin and was appointed to its first Board of Regents. Because his health was poor, he declined the appointment; he died on July 25, 1890, in Marshall.

Dr. Starr was dedicated to Texas, and spent 50 years of tireless effort in its development. He was a true pioneer in many fields and none can say that his efforts were entirely in vain.

SUGGESTED READING

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Dr. Thames, 818 Frank Avenue, Lufkin.

Shoulder Pads Blamed for Gridiron Neck Injuries

Low, professional-type shoulder pads are partly responsible for lateral flexion neck injuries among college football players. Increase in this type injury has been reported in members of the Amherst College and University of Massachusetts football teams.

Drs. Donald Chrisman and George A. Snook of Northampton, Mass., say (*JAMA*, June 29, 1964) that injury increase was noted after the conventional high-type shoulder pads were discarded for the low pads which do not limit the range of lateral flexion of the neck of the player.

"The injury is a sprain, with associated stretching of the lower cervical nerve roots on the side of the sprain," Chrisman said. "Clear evidence was obtained [in half the 22 injured players studied] that lateral flexion was the major element of the impact leading to injury, and that the symptoms were produced on the side on which the force was applied."

The clinical picture is the same in all cases seen. Neck and shoulder ache was usual, and neurologic changes in the arm were common.

"Diminution of the biceps or triceps reflexes and patches of radial or ulnar numbness lasted for several months in some injured players. Muscle weakness in either the extensor-supinator or the flexor-pronator groups was noted in over half the patients," he said.

Re-injury is common unless the player uses a protective collar.

Clinicopathologic Conference

Thomas F. Dutcher, M.D.; John D. Nelson, M.D.;
Joan Windmiller, M.D.; and John Eberts, M.D.

This is the fifth in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School; Dr. C. T. Ashworth, professor and chairman of the Department of Pathology, serves as guest editor for the series.

Dr. Dutcher is with the Department of Pathology; Dr. Nelson and Dr. Windmiller are with the Department of Pediatrics, and Dr. Eberts is with the Department of Radiology. All are staff members of the Children's Medical Center.

Clinical Summary

DR. NELSON: A white girl, age three years and four months, was admitted to Children's Medical Center Feb. 23, 1963, for fever and cough of two weeks' duration. On Jan. 16 she had been given an injection of gamma globulin (0.16 ml. per kg. body weight) for measles exposure. Approximately one month later she developed fever and skin rash which was diagnosed by her private physician as measles. Because of persisting fever and increasing cough she was treated with penicillin and tetracycline, but without improvement. Three days before admission she was seen as an outpatient in the hematology department. She had fever, a generalized maculopapular rash compatible with rubeola, and pulmonary findings consistent with diffuse bronchopneumonia. A throat culture showed no pathogens and the patient was started on chloramphenicol therapy. There was no improvement, and she was admitted to the hospital.

Past History.—The patient had been diagnosed as having acute lymphoblastic leukemia in October, 1960. Antileukemic therapy which was given is outlined in Table 1. One notable thing is that she was never treated with steroids. She was treated with 6-mercaptopurine and methotrexate for the periods noted, and was receiving methotrexate at the time of her final admission.

Physical Examination on Admission.—Weight 30 lbs., height 37 in., blood pressure 110/70 mm. of mercury, pulse 150 per minute, respirations 40 per minute and grunting, temperature 102 F. Examination revealed an acutely and severely ill dyspneic child with dry, scaly skin. The other positive findings were a bilateral periorbital puffiness, generalized tubular breath sounds without rales, and mild edema of the lower extremities. There was no hepatosplenomegaly and no significant lymphadenopathy.

Laboratory Findings. — The hemoglobin was 11.6 gm. per 100 cc. and the hematocrit 40 per cent. The white blood count was 13,100 per cu. mm. with the differential showing a shift to the left of 73 per cent segmented polymorphonuclear leukocytes, 13 per cent band forms, 12 per cent lymphocytes and 2 per cent eosinophils. There were no immature cells in the peripheral blood smear. Platelets were 66,000 per cu. mm. The urine had a specific gravity of 1.028 and contained traces of acetone and protein. The blood urea nitrogen was 8 mg. per 100

TABLE 1.—*Treatment for Leukemia.*

Medication	Dosage	Duration	Response
6-Mercaptopurine	2.5 mg./kg./day	10/17/60-5/16/61	Good
Methotrexate	2.5 mg./day	5/16/61-Final Admission	Good (Decreased platelets)

A child with leukemia in remission developed a fever, a maculopapular rash, and an unusual type of pneumonia. She died 30 hours after admission to the hospital.

cc. and the serum uric acid 0.8 mg. per 100 cc. A nasopharyngeal swab culture grew *Pseudomonas*. Two blood cultures were taken; one had no growth and the second contained *Micrococcus tetragenus*.

Radiologic Findings

DR. EBERTS: Posteroanterior and lateral views of the chest (Figs. 1 and 2) demonstrate a bilateral patchy diffuse infiltrate, involving all lobes and definitely nodular in some areas. The process is more confluent in the right lung with a suggestion of early consolidation and some loss of volume. The shift of the mediastinum is slightly greater than can be accounted for by rotation. The differential diagnosis must include a hematogenous granuloma, a severe extensive bronchopneumonia (probably viral) and a leukemic infiltrate. Pulmonary or uremic edema is unlikely. The cardiac silhouette is normal in size. A pleural effusion is not identified. The thoracic bony cage and soft tissues are not remarkable. The final impression then is of extensive bilateral bronchopneumonia; a hematogenous granulomatous process should be excluded.

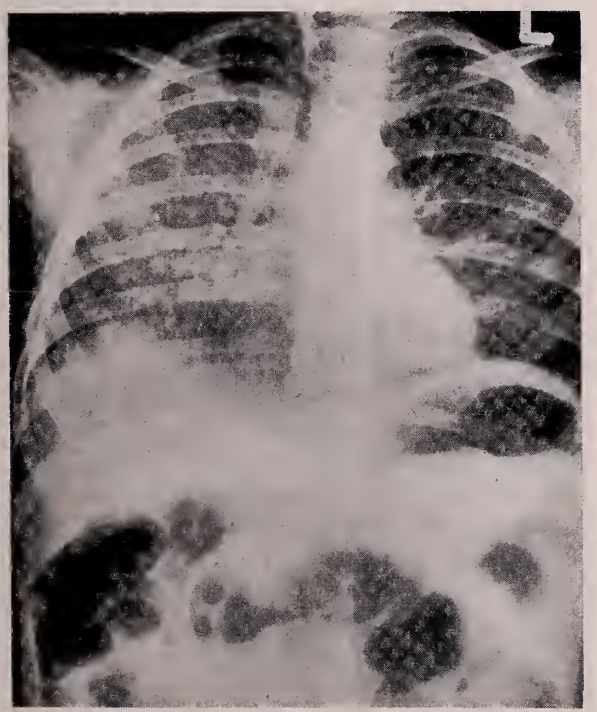

Fig. 1. Posteroanterior roentgenogram of chest.

Fig. 2. Lateral roentgenogram of chest.

Hospital Course

DR. NELSON: The patient was continued on chloramphenicol therapy and given parenteral penicillin. There was a slight decrease in fever but a progressive increase in respiratory distress, cardiac rate, and edema. Digitalis and diuretics were given and intramuscular colistin was added to the antibiotic therapy. The patient voided once shortly after admission, but during the remaining 27 hours of life did not void. Respirations became more labored, and there was a constant hacking cough. Attempts at endotracheal resuscitation were unsuccessful. The patient died 30 hours after admission.

Clinical Discussion

DR. THOMAS WILLIAMS: Had she had measles immunization?

DR. JOAN WINDMILLER: No.

DR. FRED BROOKSALER: Was a tuberculin skin test done or did she have any known contact wth tuberculosis?

DR. WINDMILLER: No.

DR. NELSON: In summary, this is a child with leukemia who was in hematologic remission when she developed an exanthematous disease and ten days later died of pneumonia. Now, we will make the assumption, hopefully a correct one, that this child really did have acute lymphocytic leukemia—that the original diagnosis was correct. All the evidence indicates that the leukemia was in remission. We do not have a bone marrow study performed after the child's hospitalization, but there is nothing in the peripheral blood to suggest that she was in relapse. Central nervous system leukemia involvement can occur without peripheral blood manifestations of leukemia, but we have no clinical or laboratory evidence from which we could draw such an inference in this case.

The child's terminal illness was pneumonia. What kind of infection could this be? The number one thing we have to consider is measles. She was seen by two or three different people, all of whom definitely thought she had measles. The fact that she had received almost a full protective dose of gamma globulin a month before she developed measles does not allow one to rule out the diagnosis of measles at all. Leukemic children have been shown by Mitus, and others,[5] to be highly susceptible to measles in spite of gamma globulin administration. To put it another way, high doses of prophylactic gamma globulin do not provide good protection against measles for leukemic children.

The lapse of 13 or 14 days between the onset of measles and death from pneumonia is compatible with measles pneumonia. In children with leukemia, the measles virus may persist for a long time and antibody formation is delayed or does not occur at all.[6]

If this child had measles virus pneumonia, or Hecht's giant cell pneumonia as it is called, we must consider the possibility of superimposed bacterial infection. This would certainly be a natural chain of events, particularly in a child who had received antibiotics which would tend to select the types of organisms in the respiratory tract. The fact that *Pseudomonas* was cultured from her nasopharynx doesn't mean that it was down in her lungs, but there might possibly be an association. In this regard, the roentgenograms are helpful to me. When I see a pneumonia that is as diffuse and extensive as this with a uniform granular involvement, I think of granulomatous or viral processes more than bacterial causes.

Another viral cause of pneumonia in children with leukemia is cytomegalic inclusion disease. In this case we have nothing in particular to support or refute this diagnosis. Children with leukemia, children who have been on steroids, and children with any sort of difficulty in their immune response are particularly prone to infections with *Candida,* Pneumocystis carinii, and *Toxoplasma,* but again there are no supportive data for any of these. A skin test was not done, and although there is no particular reason to suspect tuberculosis on the basis of history, the recent episode of measles would have abolished the Mantoux reaction in this child.[2]

What might have caused her death other than pulmonary disease itself? As I mentioned before, children who are in hematologic remission from leukemia can develop fatal central nervous system leukemia. We have no clinical evidence for this diagnosis. Likewise, fatal hemorrhage must also be considered since the platelets were moderately depressed; but this patient had no signs of bleeding in the central nervous system.

The periorbital puffiness and the pitting edema of the feet perplex me. One has to consider renal or cardiac failure. She had oliguria which could have been caused by

renal disease or by her poor oral intake before admission to the hospital. The normal BUN and routine urinalysis are not compatible with a diagnosis of renal insufficiency. The heart was not enlarged, the tachycardia was commensurate with the fever, and the liver was not enlarged. These findings would all be against a diagnosis of congestive heart failure, particularly in view of the fact that edema is one of the late manifestations of cardiac failure in children. I cannot explain the edema unless one assumes that she had hypoalbuminemia.

The isolation of *Micrococcus tetragenus,* a common contaminant of blood cultures, from the second blood culture I will dismiss as having no significance because of another sterile blood culture. There is no evidence, in my opinion, that this child had generalized bacterial sepsis.

In summary, the diagnoses I would favor are acute lymphocytic leukemia in remission and measles virus pneumonia.

DR. WINDMILLER: First it should be noted that this child was in complete hematologic remission on methotrexate during this terminal illness. In fact, she was able to mobilize a white count of 13,000 with a left shift. Despite a platelet count of less than 100,000, she had no bleeding; and, as Dr. Nelson mentioned, there was nothing to suggest leukemic involvement of, nor hemorrhage within, the central nervous system as the cause of the terminal event. "Full blown" leukemic relapse, therefore, cannot be incriminated as the cause of the fulminating nature of this child's pulmonary infection. Neither can steroid therapy, since this patient was never given steroids. The majority of bacterial infections seen in children with leukemia are caused by gram-negative organisms, especially *Pseudomonas* and *Escherichia coli.* These infections most commonly occur during periods of hematologic relapse or periods of drug toxicity, that is, at a time when the circulating level of granulocytes is decreased. This child had neither neutropenia nor characteristic skin lesions of gram-negative septicemia. Although I do not feel that the terminal episode was the result of leukemia per se I do feel that leukemia or the antileukemic therapy contributed to this child's death by alteration of her immune mechanism. For this reason, measles seems a very good possibility. In contrast to the bacterial infections which are most severe in the presence of neutropenia, fatal cases of measles are likely to occur, and frequently do occur, when the child is in hematologic remission. The four children reported by Mitus and others[6] were receiving methotrexate and not steroids or 6-mercaptopurine, as was true in the present case. All these drugs are known to suppress the immune response.

A number of interesting observations have been made regarding the course of measles in leukemic patients:

1. There is persistence of the virus for an unusually long time in the upper respiratory tract.

2. There is failure, in most, to develop specific antibodies. This particular feature may be a direct result of methotrexate therapy.[3]

3. These two unusual features seem to be associated with the host and not with the agent, since strains of the virus isolated from children with leukemia do not differ significantly from those taken from children with typical measles.

With these altered responses it seemed logical, therefore, to immunize leukemic children against measles. In one such investigation by Mitus,[5] 12 children with leukemia in remission were chosen for study. Eight were considered susceptible. They were all given the live attenuated measles vaccine and gamma globulin. Of the eight susceptible children, four developed normal antibodies; two had no antibody response; and two had a delayed response. One of the children with delayed antibody production developed measles one month following vaccination and died of measles pneumonia. I am not familiar with any studies using the inactivated vaccine for measles.

DR. ROBERT KRAMER: Is methotrexate toxic to the kidney? Might this patient have renal failure on the basis of drug toxicity?

DR. WINDMILLER: Methotrexate is toxic to buccal mucous membrane, gastrointestinal tract, and bone marrow. Symptoms of toxicity include anorexia, abdominal pain, ulceration of mouth and gastrointestinal tract, diarrhea, and alopecia. Methotrexate does not cause renal insufficiency. If, however, renal failure is present, blood levels of methotrexate will become elevated and the toxic symptoms will be accentuated.[1] The oliguria in this patient was probably secondary to dehydration and not to renal failure. The BUN was within normal limits and we have no evidence of preexisting renal dis-

ease. With a normal blood uric acid level, impaired renal function associated with hyperuricemia can be excluded in this patient.[7] Finally, renal failure secondary to leukemic infiltration of the kidneys has not been reported in childhood.[4]

DR. SHIRLEY KINDBERG: I realize that it would be extremely rare, but is it possible for leukemic infiltration of the lungs to occur during hematologic remission?

DR. WINDMILLER: Not to my knowledge. Even at the time of diagnosis or during periods of relapse pulmonary involvement is exceedingly rare. The overall incidence of pulmonary involvement in acute leukemia including mediastinal adenopathy, pleural effusion, and pulmonary infiltration is between 30 and 65 per cent. These are, for the most part, autopsy findings.

Pathological Findings

DR. DUTCHER: Dr. Nelson's first concern was the possibility that the patient did not have leukemia. I have reviewed the bone marrow sections and smears obtained at the time of the diagnosis and there is no doubt about the diagnosis of acute lymphocytic leukemia. Pathologists today do, on occasion, have difficulties in establishing a histological diagnosis of leukemia at autopsy. This is because the currently used chemotherapeutic agents significantly alter the classical appearances of leukemic infiltrates and frequently no definite foci of leukemia can be found.

The bone marrow obtained in our case at autopsy (Fig. 3) is hypocellular and the cells present are principally megakaryocytes and erythroid elements. There is granulocytic hypoplasia despite a reasonably adequate peripheral leukocytosis. No leukemic foci were found.

The lymph nodes have lost the normal architecture and this is manifested by the absence of the cortical lymphoid follicles. This is seen in a variety of conditions and is frequently associated with antileukemic therapy. Definite leukemic infiltration of the lymph nodes is not seen.

An interesting finding in the lymph nodes is the presence of multinucleated giant cells. These cells are scattered throughout the node and do not simulate the giant cells of granulomatous inflammation. At higher power (Fig. 4), eosinophilic inclusions are visible within some of the nuclei. These cells are known as Warthin-Finkeldey cells and they are specific for the prodromal stage of measles. One of the pathologist's minor delights in life is to find Warthin-Finkeldey cells in tonsils or an appendix and then inform the surgeon that within a few days the patient will have an overt measles rash.

Giant cells of this type have been found ten or eleven days prior to the rash but are usually seen three to four days before, and then disappear as the rash develops. It is unusual for them to persist after the onset of the rash; but, as noted by Dr. Nelson, in leukemic children the virus of measles may persist and this seems to be associated with persistence of the Warthin-Finkeldey cells.

The lungs are interesting. There is a giant cell pneumonia, and there is no evidence of a superimposed *Pseudomonas* or other bacterial infection. There are numerous giant

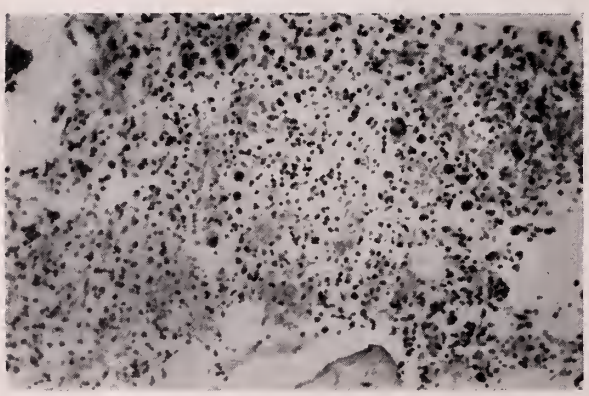

Fig. 3. Photomicrograph of bone marrow at autopsy. There is moderate hypocellularity and a predominance of nucleated red cells and megakaryocytes. Hematoxylin and eosin stain. Low power.

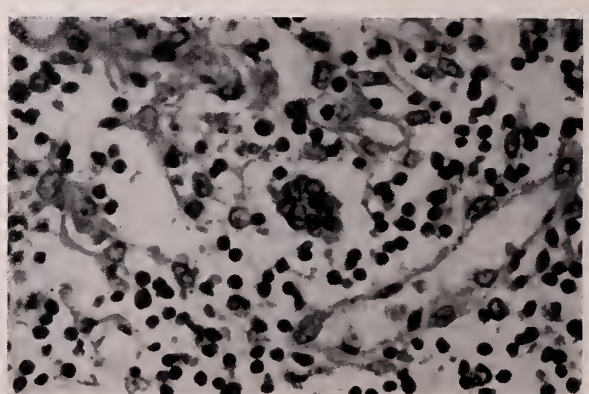

Fig. 4. Photomicrograph of lymph node. Multinucleated Warthin-Finkeldey giant cells and pale intranuclear inclusions are seen. High power.

cells, hyaline membranes on the alveoli, and a mononuclear cellular infiltrate which is principally interstitial but also present in the alveoli (Fig. 5). This histological combination was first described by Hecht in 1910 and was known to be associated with measles. It was not certain that Hecht's giant cell pneumonia was specifically a consequence of measles infection since it was occasionally seen in deaths which occurred without an antecedent measles rash. Through the years other etiologic agents for the development of giant cell pneumonia received speculative consideration—mainly the virus of canine distemper and vitamin A deficiency. This question was settled by Enders and his group in 1959. In companion papers cited previously by Dr. Nelson,[2, 6] this group identified and cultured measles virus from three cases in which clinical measles had not preceded the giant cell pneumonia, and subsequently cultured the measles virus from four patients with giant cell pneumonia which followed obvious clinical measles. Each of these latter four were children with acute leukemia in remission.

The giant cells in the lungs of this case display the characteristic features of this disease very nicely. At high power (Fig. 6), it can be appreciated that the individual giant cell is formed as a syncytium of several cells. Eosinophilic intranuclear inclusions are readily seen and most of the cells also have intracytoplasmic inclusions. In this group of affected cells, the cilia are still present and syncytial formation is not complete (Fig. 7). These changes are identical to the cytopathic effects of the measles virus on cells in tissue culture.

This child's liver is the site of a variety of lesions (Figs. 8, 9). There are marked fatty metamorphosis, focal acute necrosis with an acute inflammatory reaction, and numerous giant cells with intranuclear inclusions formed from hepatic parenchymal cells. It is possible that the changes in the liver might have given rise to hypoalbuminemia and consequent edema, but we cannot be certain of

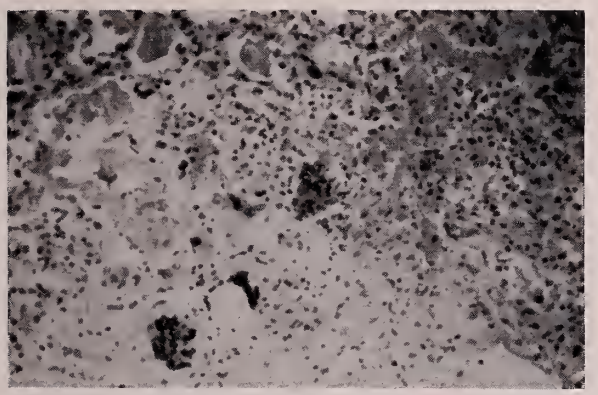

Fig. 5. Histologic section of lung showing pneumonia and large multinucleated giant cells. Low power.

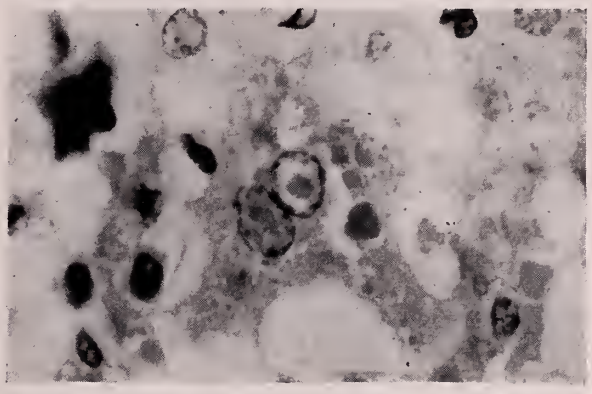

Fig. 6. High power view of lung with Hecht's giant cell pneumonia. Prominent, pale (eosinophilic) intranuclear inclusions are visible in this giant cell which is produced by a syncytial fusion of several cells.

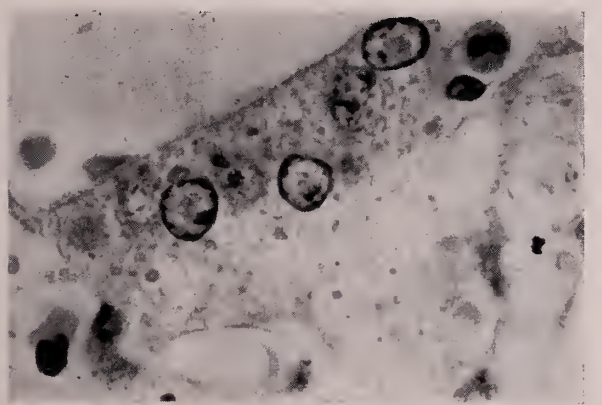

Fig. 7. High power view of affected group of bronchial epithelial cells in giant cell pneumonia. Cilia are still present, and intranuclear inclusions are noted. Such cell groups coalesce to form giant cells.

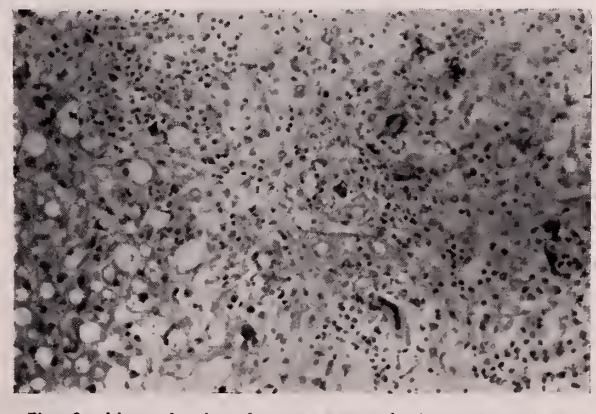

Fig. 8. Liver showing fatty metamorphosis, periportal inflammation, and Warthin-Finkeldey giant cells in periportal area. Hematoxylin and eosin stain. Low power.

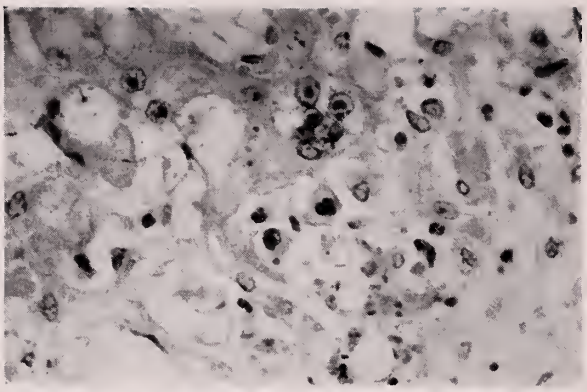

Fig. 9. A high power view of periportal area of liver, showing a multinucleated giant cell in the nuclei of which the typical viral inclusions can be seen.

this. No other cause for edema was found. There was no histologic evidence for passive congestion of heart failure. Giant cells were not found in the heart although such instances have been recorded in the literature. Except for a few giant cells in the epithelium of the renal pelvis there were no renal lesions.

Neither viral nor leukemic lesions were found in the central nervous system.

Of passing interest was the presence of giant cells within the lining epithelium of the uterus. Although giant cells have been described in nearly every tissue and organ of the body in this disease, I am not aware of any previous mention of involvement of the uterine epithelium.

In conclusion, this is a case of a child with acute lymphocytic leukemia in remission. She contracted measles and died from extensive measles virus infection which was particularly severe in the liver and lungs.

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Drs. Dutcher, Nelson, Windmiller, and Eberts, Children's Medical Center, Dallas.

Fast Process Developed To Detect Hidden Diabetes

An "instant" process for detecting most hidden diabetics has been developed, according to a report from the International Diabetes Federation.

A new blood testing process (Dextrostix, A. E. Ames Co., Elkhart, Ind.) requires only one minute to complete.

"For the first time, we have readily available a process that promises to lend itself to simplified mass surveys using blood samples," says the Federation's director of information, Dr. D. W. Clarke.

The process has been undergoing rigid clinical tests in Canada and the United States for the past six months.

Disclosure of the process came at a time when it has been speculated that the world's diabetic population may be around 200,000,000, more than triple the present estimate.

Dr. Clarke said that research has found that diabetes exists in an undetected state for as long as ten years in some persons and that by the time symptoms appear the disease has caused considerable damage.

NEWS

Judd, Fishbein, Newman, Popma, Wood To Speak at Public Relations Meet

THE ARRAY of featured speakers to be in Austin for the 1964 TMA Public Relations Conference on Sept. 19 includes Dr. Walter H. Judd, Dr. Morris Fishbein, Guy D. Newman, Th.D., Dr. Alfred M. Popma, and Dr. Donald E. Wood.

To be held simultaneously with the PR conference is an orientation program for new and transfer members. Both will be held at the TMA Headquarters Building, 1801 N. Lamar, and are due to start at 9 a.m. Physicians attending the orientation program will have an opportunity to hear all the featured speakers.

The day's events will also include meetings of committees and councils (others will be held Friday night), two panel discussions —"Working Together for Better Community Health Care" and "How To Preach What You Practice — Medicine's Basic PR Program," a school of instruction for members of the Auxiliary, and an address by TMA President Dr. Max E. Johnson. The Executive Board will meet Sunday morning.

Lunch will be served at the building free of charge to all those attending the meetings. A hospitality hour and buffet has been arranged for physicians, their wives, and Association guests by the Charles O. Finley Company. This will be at 5:30 p.m. at the Driskill Hotel. Complimentary bus transportation will be provided to the football game at Memorial Stadium at 7:30 p.m. The University of Texas, ranked the nation's No. 1 football team in 1963, will host Tulane University, and TMA has reserved a block of 500 tickets at $4 each for physicians, their wives, and guests.

Physicians may now make room reservations at the Austin hotel or motel of their choice. Leading facilities which have set aside rooms for members of the TMA are Stephen F. Austin Hotel, 701 Congress Avenue; Commodore Perry Hotel, 800 Brazos; Driskill Hotel, 117 East 7th; Gondolier Hotel, 1000 South Interregional Hwy.; Imperial '400' Motel, 901 South Congress; Terrace Motor Hotel, 1201 South Congress Avenue; and Villa Capri Motor Hotel, 2360 Interregional Hwy.

The doctor's place in public affairs will be the theme of Dr. Walter H. Judd's address. The statesman and former Congressman from Minnesota was a delegate to the recent Republican National Convention in San Francisco where his name was placed in nomination for President of the United States. At the 1960 Republican National Convention in Chicago, Dr. Judd gave the keynote address.

A former medical missionary to China, Dr. Judd is able to speak about Communism from personal experience because he worked in areas under its control as far back as 1930. He is a nationally recognized authority on U.S. foreign policy and was a member of the House Committee on Foreign Affairs for 16 years. A former delegate to the General Assembly of the United Nations, the Minnesota physician now devotes his time to speaking to interested groups all over the country and has appeared on many nationwide radio and television programs.

A long career in medical writing and editing has brought international recognition to Dr. Morris Fishbein. He is and has been identified with many publications; he is editor of *Medical World News*, contributor to *McCalls*, and former editor of the *Journal of the American Medical Association*, to mention a few.

A prolific contributor to the lay literature on medicine and health as well as a writer for the medical profession, the Chicago physician will speak to members of TMA on the subject of "The Evolution of Quackery."

"Politics and the Medical Profession" is the title of the address to be given by Dr. Don E. Wood of Indianapolis. Dr. Wood is chairman of the American Medical Political Action Committee, a member of the AMA Council on Legislative Activities, President of the Indiana State Medical Association, and professor of Medicine at the Indiana University School of Medicine.

Dr. Alfred M. Popma of Boise, Idaho, chairman of the American Medical Association Council on Voluntary Health Agencies, will speak on "The Physician and Voluntarism." Dr. Popma, a fellow of the American College of Radiology, is director of the Department of Radiology at St. Luke's Hospital in Boise and is a consultant at the

VA hospital there. He is past president of Idaho State Medical Association, Rocky Mountain Radiological Society, and the American Cancer Society. Other affiliations include membership in the Western Interstate Commission for Higher Education and chairmanship of the board of trustees, College of Idaho, Caldwell, Idaho.

Guy D. Newman, Th.D., the president of Howard Payne College in Brownwood, will give an inspirational talk on responsibilities of the individual in citizenship. A Baptist minister, he has been pastor of a number of Texas churches and has served on several denominational boards. He is the author of "When God Invaded History," the originator of the Democracy in Action program to promote Americanism on college and high school campuses, and the recipient of a Freedoms Foundation award in 1960.

Opposition Urged To Social Security

Texas physicians are being urged to express personally opposition to legislation in Congress which would require compulsory participation by self-employed physicians and interns in the Social Security retirement program.

In a letter to presidents and legislative chairmen of all county medical societies in Texas, Dr. John M. Smith, Jr., chairman of the Council on Medical Jurisprudence, asks physicians to appeal to Sen. John Tower for assistance in asking members of the Senate Finance Committee to delete the provision in H.R. 11865 which requires participation by self-employed physicians in Social Security.

"After having waged a successful battle for many years to prevent compulsory participation by physicians in Social Security, it is distressing that this legislation is sailing toward enactment by the 88th Congress. Only immediate, effective opposition by interested physicians can prevent this development," Dr. Smith stated.

Dr. Max E. Johnson, President of TMA, has also urged expression of opposition to the provision. Writing to presidents and legislative chairmen regarding opposition to any amendment providing health care financed through Social Security taxes, Dr. Johnson suggested that physicians express their opinions to the Texas senators and to Sen. Harry F. Byrd, chairman of the Senate Finance Committee, regarding the compulsory inclusion of self-employed physicians under the Social Security Act.

In his communication, which stressed that a strong effort will be made by some members of the Senate Finance Committee to amend the House bill to include Medicare, Dr. Johnson urged doctors and their friends to write the senators at once. Early action by the Senate Finance Committee and the Senate is expected.

The Social Security Amendments of 1964, H.R. 11865, was passed by the House of Representatives July 29, 388 to 8. Four of the eight dissenting votes were cast by Texans: J. J. Pickle, Joe R. Pool, and Walter Rogers, all Democrats; and Ed Foreman, Republican. The bill provides a 5 percent across-the-board increase in Social Security cash benefits and other provisions, *including the participation of self-employed physicians and interns in the program.*

Although the House Ways and Means Committee shelved the controversial Medicare proposal, the Committee voted for inclusion of self-employed physicians in Social Security coverage.

In voting this inclusion, the Committee reported, "Large numbers of doctors have requested coverage. Your Committee knows of no valid reason why this single professional group should be excluded."

H.R. 11865 was considered by the House of Representatives under the closed rule in which no amendments were permitted from the floor. Even though many Texas Congressmen supported physicians in their opposition to participating in the Social Security retirement program on a compulsory basis, they were unable to vote against that provision without voting against the entire bill.

At this point the best prospect for deleting this amendment is through the Senate Finance Committee, it is now believed.

The Texas Medical Association is firmly on record against compulsory inclusion of physicians in the Social Security System. The Association's position was based initially on a statewide poll in 1956 which was conducted among the entire membership of the Association. Texas physicians expressed opposition to compulsory coverage by a ten to one vote. This position has been reiterated almost annually by the House of Delegates.

The opposition of many physicians is based upon the following points:

1. Physicians desire the privilege of establishing retirement benefits for themselves on a voluntary basis.

2. Compulsory participation would represent another costly tax to physicians. The tax rate for the self-employed will be 5.7 percent on the first $5,400 of income in 1965; proposed increases in Social Security taxes are scheduled in 1966, 1967, and 1971, with the rate for self-employed reaching 7.2 percent in 1971.

3. Most physicians will have little prospect for enjoying Social Security retirement benefits until reaching age 72. Social Security retirement benefits are limited essentially to those who retire at age 65 and who have earnings of less than $100 monthly. The majority of physicians between the ages of 65 and 72 are still in active practice, and are ineligible for Social Security retirement benefits on the basis of their earnings.

Auxiliary Announces School of Instruction

The Woman's Auxiliary to the Texas Medical Association will hold its annual fall executive board meeting and school of instruction on Sept. 17 and 18.

The executive board meeting will be held on the afternoon of the 17th, preceded by meetings of various state committees. On this evening a dinner and style show will be held to which all persons attending the school are invited.

On the 18th all county presidents and presidents-elect have been invited to attend a school presented by the state officers and committee chairmen. The purpose of this school is to present to the county representatives all new information and material which has become available on the various functions and projects which comprise the life of the Auxiliary.

Two guest speakers will be on the program, and there is new material to be presented to help each auxiliary choose projects which will hold the interest and enthusiasm of its members. Three new films will be shown at the end of the school day for those desiring to see them.

1964 Public Relations Conference

Texas Medical Association Headquarters Building

1801 N. Lamar Austin Texas

Saturday the 19th of September

8:30—REGISTRATION AND COFFEE

OPENING SESSION

Sam Thompson Room

9:00—CALL TO ORDER AND WELCOME—Travis Smith, M.D., Abilene, Vice President, Texas Medical Association

9:10—UNITED WE STAND—Max E. Johnson, M.D., San Antonio, President, Texas Medical Association

9:25—THE WORLD DRIFT AND OUR WESTERN ALTERNATIVE—Guy D. Newman, Th.D., Brownwood, President, Howard Payne College

9:55—THE PHYSICIAN AND VOLUNTARISM—Alfred M. Popma, M.D., Boise, Idaho, Chairman, Council on Voluntary Health Agencies, American Medical Association

10:25—THE EVOLUTION OF QUACKERY—Morris Fishbein, M.D., Chicago, Editor, *Medical World News*

10:55—COFFEE BREAK

SECOND MORNING SESSION

Library Stack Area

11:10—Panel Discussion: WORKING TOGETHER FOR BETTER COMMUNITY HEALTH CARE—Robert Mayo Tenery, M.D., Waxahachie, Moderator, Immediate Past President, Texas Medical Association

1. COMMUNITY DENTAL CARE PROGRAMS—Crawford A. McMurray, D.D.S., Ennis, Secretary-Treasurer, Texas Dental Association
2. THE ROLE OF THE NURSING HOME IN A COMPLETE HEALTH CARE PROGRAM—C. B. Francis, Austin, President, Texas Nursing Home Association
3. NURSING PROGRAMS ESSENTIAL TO GOOD HEALTH CARE—Mrs. Lillian E. Taubert, R.N., San Antonio, Executive Director, Texas Nurses Association
4. PHARMACY AND COMMUNITY HEALTH—Representative of Texas Pharmaceutical Association
5. JOINT EFFORTS OF THE HEALTH PROFESSION WHICH WILL PROMOTE BETTER HOSPITAL CARE—George B. Pearson, F.A.C.H.A., Tyler, Past President, Texas Hospital Association

QUESTION AND ANSWER PERIOD

12:40—LUNCHEON—Basement, Compliments of Texas Medical Association, Catering by Walter Jetton, Fort Worth

FIRST AFTERNOON SESSION

Sam Thompson Room

David Wade, M.D., Austin, President-Elect, Texas Medical Association, presiding

1:30—POLITICS AND THE MEDICAL PROFESSION—Donald E. Wood, M.D., Indianapolis, Ind., Chairman, Board of Directors, American Medical Political Action Committee

SECOND AFTERNOON SESSION

Library Stack Area

2:10—Panel Discussion: HOW TO PREACH WHAT YOU PRACTICE—Introductions by Glenn D. Carlson, M.D., Dallas, Chairman, Council on Public Relations and Public Service, Texas Medical Association

MEDICINE'S BASIC PR PROGRAM—James R. Hickox, Chicago, Director, Program Services Department, Communications Division, American Medical Association

The Program as Seen by . . .

1. THE SMALL COUNTY MEDICAL SOCIETY—Sam A. Nixon, Jr., M.D., Floresville, Chairman, Public Relations Committee, Gonzales County Medical Society
2. THE MEDIUM SIZE COUNTY MEDICAL SOCIETY—C. Mac Geyer, M.D., Wichita Falls, Chairman, Public Relations Committee, Wichita County Medical Society
3. THE LARGE COUNTY MEDICAL SOCIETY—B. B. Markette, M.D., San Antonio, Chairman, Public Relations Committee, Bexar County Medical Society

QUESTION AND ANSWER PERIOD

CLOSING SESSION

Sam Thompson Room

Max E. Johnson, M.D., San Antonio, President of Texas Medical Association, presiding

3:50—PRESENTATION OF THE TEXAS MEDICAL ASSOCIATION'S DISTINGUISHED SERVICE AWARDS—1964 Recipients: George R. Herrmann, M.D., Galveston, and Milford O. Rouse, M.D., Dallas

4:00—THE DOCTOR'S PLACE IN PUBLIC AFFAIRS—Walter H. Judd, M.D., Minneapolis, Minn., Statesman and Former Congressman

4:30—ADJOURNMENT

EVENING FELLOWSHIP AND ENTERTAINMENT

5:30—HOSPITALITY HOUR AND BUFFET—Hosted by Charles O. Finley Company, Crystal Ballroom, Driskill Hotel

6:45—CHARTERED BUSES LEAVE FOR MEMORIAL STADIUM—Brazos Street Entrance, Driskill Hotel, Courtesy Charles O. Finley Company

7:30—FOOTBALL: TEXAS VS. TULANE—Memorial Stadium

Orientation Program

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10:55—COFFEE BREAK

SECOND MORNING SESSION

James D. Murphy, M.D., Fort Worth, Speaker, House of Delegates, Texas Medical Association, presiding

11:10—AN ACCOUNTING OF STEWARDSHIP; WHAT HAPPENS TO YOUR DUES—Byron P. York, M.D., Houston, Chairman, Board of Trustees, Texas Medical Association

11:30—THE BEST MEDICAL CARE FOR ALL TEXANS—C. Lincoln Williston, Austin, Executive Secretary, Texas Medical Association

11:55—THE PHYSICIAN'S RESPONSIBILITY IN PRESERVING VOLUNTARY HEALTH INSURANCE—A. Rex Kirkley, M.D., Dallas, Chairman, Committee on Health Insurance, Texas Medical Association

12:10—LUNCHEON—Basement, Compliments of Texas Medical Association, Catering by Walter Jetton, Fort Worth

FIRST AFTERNOON SESSION

Sam Thompson Room

David Wade, M.D., Austin, President-Elect, Texas Medical Association, presiding

1:00—MEDICAL ETHICS—OBLIGATIONS OF THE PHYSICIAN TO PATIENTS, COLLEAGUES, AND TO THE PROFESSION—James H. Sammons, M.D., Highlands, Secretary, Board of Councilors, Texas Medical Association

1:15—THE PHYSICIAN AS A CITIZEN—Carlos E. Fuste, Jr., M.D., Alvin, Vice Chairman, Board of Councilors, Texas Medical Association

1:30—POLITICS AND THE MEDICAL PROFESSION—Donald E. Wood, M.D., Indianapolis, Ind., Chairman, Board of Directors, American Medical Political Action Committee

SECOND AFTERNOON SESSION

Charles Max Cole, M.D., Dallas, Vice Speaker, House of Delegates, Texas Medical Association, presiding

2:10—SERVING THE DOCTORS OF TEXAS—Donald M. Anderson, Austin, Assistant Executive Secretary, Texas Medical Association

2:30—DOCTOR-PATIENT RELATIONS—Walter Walthall, M.D., San Antonio, Chairman, Board of Councilors, Texas Medical Association

2:50—THE VETERANS ADMINISTRATION HOSPITAL PROGRAM; ITS EFFECT UPON THE PRIVATE PRACTICE OF MEDICINE—Milton V. Davis, M.D., Dallas, Chairman, Committee on Military and Veterans Affairs, Texas Medical Association

3:10—LEGAL ASPECTS OF MEDICINE; MALPRACTICE—HOW TO AVOID IT—Philip R. Overton, LL.B., Austin, General Counsel, Texas Medical Association

CLOSING SESSION

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7:30—FOOTBALL: TEXAS VS. TULANE—Memorial Stadium

Personals

Miss Winifred Ann Killian of Junction City, Kan., became the bride of *Dr. Robert Alexander Wilson* of Dallas in a ceremony performed June 7 in Junction City.

Don Anderson, assistant executive secretary of Texas Medical Association, was recently elected president of the Texas Society of Association Executives.

Dr. W. S. Fry, Jr., of Denison has been named 1964 Texas Baptist Father of the Year.

Dr. William C. Baumann, Biggs Air Force Base, has been awarded a $1,000 scholarship for a year's general practice residency, one of 20 made this year by the American Academy of General Practice through funds provided by Mead Johnson and Co. The award was presented by Dr. A. Robert Nering, El Paso, director for the Texas Academy of General Practice, at a meeting of the El Paso County Medical Society.

Dr. Clarence Dale Snyder is replacing *Dr. R. F. Anderson* in his offices in Refugio. *Dr. Anderson* is serving a residency in internal medicine at the University of Texas Medical Branch.

Dr. Morris E. Hill of Fort Worth has accepted a position in Sweetwater with the Peters Clinic to be associated with *Drs. R. O. Peters* and *S. F. Supowit.*

Dr. E. F. Vernezobre is expected to move to Eldorado to begin practice. He has been practicing in Baird.

Dr. W. Compere Basom of El Paso has been elected president of the Texas division of the Intertional College of Surgeons.

Dr. C. E. Reynolds of Fort Worth is moving to Sulphur Springs. *Dr. Lester Hodges* of Sulphur Springs is leaving to specialize in pediatrics. Joining the staff of Hopkins County Memorial Hospital as a specialist in radiology is *Dr. B. O. Brian.*

Dr. Louis W. Breck of El Paso has been elected a fellow of the American Association for the Advancement of Science.

Dr. Louis J. Levy, Fort Worth, has been named chairman of the professional division of the 1964 Tarrant County United Fund "Crusade of Hope."

Dr. P. C. Clements of Lufkin has been named to the honorary staff of the Memorial Hospital there in recognition of a half century of "distinguished medical practice" both to the community and to Memorial Hospital since it was opened.

Dr. J. W. Young, Sr., 84, of Sweetwater, recently displayed a collection of his own craftwork, stone jewelry and woodwork, in the Jay-Rollins Library at McMurry College, Abilene.

Dr. Robert L. Raleigh of Longview spoke on "Industrial Approach to Alcoholism" at the seventh annual Institute on Alcohol Studies at the University of Texas in Austin July 26-30.

Dr. W. H. Buckholts, administrator of Dallas Veterans Administration Hospital since 1955, plans to retire before the end of the year.

Paul A. Weiss, Ph.D., of the Rockefeller Institute, New York, has been appointed university professor and dean of the Graduate School of Biomedical Sciences at Houston. Dr. Weiss succeeds *Dr. Grant Taylor*, who has been acting dean of the Houston institution, which was authorized by the Texas Legislature in 1963. Dr. Taylor will resume the deanship of continuing education, an assignment which he has held with distinction for several years.

Dr. Ridings Lee, Dallas, has been appointed chairman of the Texas Medical Advisory Committee to the U. S. Olympics.

Dr. Frank J. Kelly, Richland, Wash., has been appointed chief physician and medical director of the Whiting, Ind., refinery of American Oil Company. Dr. Kelly is formerly of Temple and Amarillo.

C. Lincoln Williston, Executive Secretary of Texas Medical Association, is a newly appointed member of the Association Committee of the Chamber of Commerce of the United States.

Dr. Stephen Ware of Corpus Christi is the new president of the Fifth and Sixth Districts Society. President-elect is *Dr. Sanford Glanz*, Corpus Christi; vice president, *Dr. B. M. Works*, San Antonio; and secretary-treasurer, *Dr. J. E. Albright*, Corpus Christi.

Dr. Philip O'B. Montgomery of the University of Texas Southwestern Medical School, Dallas, was designated as the official AMA representative to the fourth international photobiology congress in Oxford, England, July 26-30.

Dr. Ernest W. Keil has joined the staff of the VA Center in Temple as chief of psychiatry service.

Scott and White Sets Educational Afternoon

The fourth annual Educational Afternoon will be held at Scott and White Memorial Hospital in Temple Sept. 11 and 12.

Sponsored by the Educational and Research Division of Scott and White Memorial Hospital and Scott, Sherwood and Brindley Foundation, the meeting is being held in conjunction with the Scott and White Alumni Association reunion. The program is acceptable for four accredited hours by the American Academy of General Practice.

Guest speakers include Dr. Edmund B. Flink of Morganstown, W. Va., and Dr. Richard C. Lillehei, Minneapolis.

Pediatric Groups To Meet in Dallas

The 1964 meeting of the Texas Pediatric Society and the Texas Chapter, American Academy of Pediatrics, will be held at the Marriott Hotel in Dallas, Sept. 24-26.

The five guest speakers and their topics for the two-day program include Dr. Malcolm A. Holliday, Children's Hospital of the East Bay, Oakland, Calif., "Salt Losing States," "Acute Renal Failure, Poisoning and Dialysis," and "Renal Tubular Function and Clinical Pediatrics"; Dr. Irving Schulman, chairman, Department of Pediatrics, University of Illinois College of Medicine, Chicago, "Hemorrhagic Disorders of Specific Importance in Children," "Pathogenic Mechanisms in Anemias of Children," and "Biologic Significance of the Hemorrhagic Mechanism";

Dr. A. N. Boelsche, assistant professor of Pediatrics, University of Texas Medical Branch, Galveston, "The Role of the Pediatrician in Child Guidance Practice"; Dr. Leon Librik, instructor, Department of Pediatrics, Baylor University College of Medicine, Houston, "Studies in Dwarfism"; and Dr. John D. Nelson, assistant professor, Department of Pediatrics, University of Texas Southwestern Medical School, Dallas, "New Developments in Bacterial Meningitis."

TMA Officers, Councils, & Committees, 1964-1965

Officers

Max E. Johnson, President, San Antonio.
David Wade, President Elect, Austin.
Travis Smith, Vice President, Abilene.
J. M. Travis, Secretary, Jacksonville (1965).
C. Lincoln Williston, Executive Secretary, Austin.
Howard O. Smith, Treasurer, Marlin (1965).
James D. Murphy, Speaker of the House of Delegates, Fort Worth.
Charles Max Cole, Vice Speaker of the House of Delegates, Dallas.

Board of Trustees

Byron P. York, Chairman, Houston (1968).
J. B. Copeland, Vice Chairman, Austin (1966).
N. L. Barker, Secretary, Paris (1969).
Elliott M. Mendenhall, Dallas (1965).
C. P. Hardwicke, Austin (1966).
James A. Hallmark, Fort Worth (1967).
C. E. Willingham, Tyler (1969).

Committee on Physicians Benevolent Fund (special)
Travis Smith, Chairman, Abilene.
May Owen, Fort Worth.
G. V. Brindley, Sr., Temple.
Denton Kerr, Houston.
James A. Hallmark, Fort Worth.
Robert Mayo Tenery, Waxahachie.
Max E. Johnson, San Antonio.

Committee on Medical History (standing)
William M. Crawford, Chairman, Fort Worth (1965).
G. V. Brindley, Sr., Temple (1965).
W. D. Thames, Jr., Lufkin (1965).
Morris Polsky, Austin (1967).
Gerald A. King, Fort Worth (1966).
Lyman Blair, Houston (1966).
Joe C. Rude, Austin (1966).
Herbert Donnell, Waxahachie (1967).
H. Irving Schweppe, Jr., Houston (1967).[1]
W. B. Russ, San Antonio (Emeritus Member).
L. H. Reeves, Fort Worth (Emeritus Member).
H. Reid Robinson, Galveston (Emeritus Member).
Pat I. Nixon, San Antonio (Emeritus Member).

Advisory Committee to the Woman's Auxiliary (standing)
A. O. McCary, Chairman, 304 W. Broad St., Freeport (1966).
Martin L. Towler, 112 North Blvd., Galveston (1965).
Gray E. Carpenter, 2323 Montana, El Paso (1967).

Board of Councilors

First District:
Russell Holt, El Paso (1967).[2]
John C. Hundley, Fort Stockton, Vice Councilor.

Second District:
Henrie E. Mast, Midland (1966).
A. H. Daniell, Brownfield, Vice Councilor.

Third District:
Julian M. Key, Pampa (1965).
James G. Morris, Lubbock, Vice Councilor.

Fourth District:
S. Braswell Locker, Brownwood (1967).
Henry N. Ricci, San Angelo, Vice Councilor.

Fifth District:
Walter Walthall, San Antonio (1965), Chairman.
George H. Herrmann, Del Rio, Vice Councilor.

Sixth District:
Harold E. Griffin, Corpus Christi (1965).
Phil A. Bleakney, Harlingen, Vice Councilor.

Seventh District:
Jay J. Johns, Taylor (1966).
Ray L. Shepperd, Burnet, Vice Councilor.

Eighth District:
Carlos E. Fuste, Jr., Alvin (1966), Vice Chairman.
George E. Glover, Jr., Victoria, Vice Councilor.

Ninth District:
James H. Sammons, Highlands (1966), Secretary.
Haden E. McKay, Humble, Vice Councilor.

Tenth District:
Charles W. Castle, Liberty (1966).
John C. Greco, Bridge City, Vice Councilor.

Eleventh District:
R. H. Bell, Palestine (1967).
William C. Smith, Carthage, Vice Councilor.

Twelfth District:
Tom M. Oliver, Waco (1965).
Dick K. Cason, Hillsboro, Vice Councilor.

Thirteenth District:
Charles B. Dryden, Jr., Wichita Falls (1967).[3]
Drue O. D. Ware, Fort Worth, Vice Councilor.

Fourteenth District:
B. E. Park, Dallas (1967).
Horace A. Baker, Wills Point, Vice Councilor.

Fifteenth District:
Walter C. Barnes, Texarkana (1965).
William A. King, Omaha, Vice Councilor.

Committee on Contract Medicine (standing)
E. Peter Garber, Chairman, Galveston (1967).
Francis Hood, Jr., Sweetwater (1965).
William M. Ashe, Dallas (1965).
R. B. Johns, Abilene (1967).
H. W. Kilpatrick, III, Baytown (1966).
Charles A. Hulse, San Antonio (1967).[4]
Sanford Glanz, Corpus Christi (1966).[5]
James F. Fitch, McAllen (1965).[6]
Sterling H. Fly, Jr., Uvalde (1966).[7]

Committee on Liaison with State Bar of Texas (standing)
Frederick C. Lowry, Austin (1965), Chairman.
Mylie E. Durham, Jr., Houston (1965).
Robert Gossett, San Antonio (1966).
Jesson L. Stowe, El Paso (1965).
John W. Griffin, Port Lavaca (1966).
John L. Dean, Jr., Crockett (1967).[8]
Walter F. Speakman, Wichita Falls (1967).[9]
Wendell H. Hamrick, Houston (1967).[10]

1. Appointed April, 1964, to fill vacancy.
2. Elected on April 26, 1964, to replace Charles E. Oswalt, Jr., Fort Stockton, who had completed three consecutive terms.
3. Elected on April 26, 1964, to replace Travis Smith, Abilene, who had completed three consecutive terms.
4. Appointed June, 1964, to fill vacancy.
5. Appointed July, 1964, to fill vacancy.
6. Appointed July, 1964, to fill vacancy.
7. Appointed July, 1964, to fill vacancy.
8. Appointed April, 1964, to fill vacancy.
9. Appointed April, 1964, to fill vacancy.
10. Appointed June, 1964, to fill vacancy created when the

Arthur C. Ressmann, San Antonio (1966).[11]

Committee on Orientation (ex officio)
Chairman, Board of Trustees.
Chairman, Board of Councilors.
Chairman, Council on Medical Service and Insurance.
Chairman, Council on Medical Jurisprudence.
Chairman, Council on Public Relations and Public Service.

Delegates to the American Medical Association and Alternates

(terms expire on Dec. 31 of the year indicated)
J. B. Copeland, Chairman, Austin (1965).
George Turner, El Paso, Alternate.
Harvey Renger, Hallettsville (1966).
James H. Sammons, Highlands, Alternate.
John K. Glen, Houston (1966).
N. L. Barker, Paris, Alternate.
G. W. Cleveland, Austin (1966).
Ridings, E. Lee, Dallas, Alternate.
John L. Otto, Galveston (1966).
E. P. Hall, Jr., Fort Worth, Alternate.
T. C. Terrell, Fort Worth (1965).
Denton Kerr, Houston, Alternate.
M. O. Rouse, Dallas (1965).
R. D. Moreton, Fort Worth, Alternate.
J. C. Terrell, Stephenville (1965).
J. L. Cochran, San Antonio, Alternate.
R. W. Kimbro, Cleburne (1965).
J. E. Miller, Dallas, Alternate.

Executive Board

Ex Officio: President, Vice President, President Elect, Secretary, Treasurer, Speaker of the House of Delegates, Vice Speaker of the House of Delegates, Trustees, Councilors, Texas Delegates and Alternates to the AMA, Council on Medical Jurisprudence, Chairmen of Councils.

Council on Annual Session

Herman C. Sehested, Chairman, Fort Worth (1967).
John Meadows, Jr., San Antonio (1965).
John P. McGovern, Houston (1967).
Dennis M. Voulgaris, Wharton (1966).
John W. Chriss, Corpus Christi (1966).
Madison Lee, Jr., Tyler (1966).
Alice Smith, Dallas (1965).[12]

Committee on General Arrangements for Annual Session (special)
A. Fletcher Clark, Jr., Chairman, San Antonio.

Committee on Scientific Exhibits (standing)
Jasper H. Arnold, Chairman, Houston (1967).
John A. Welty, Harlingen (1965).
Charles C. Jones, Jr., Kerrville (1965).
J. Frank Beall, Nacogdoches (1965).
Walter Long, Austin (1967).
Nathan Cedars, Stephenville (1966).
W. L. Carrington, Mexia (1967).
Benedict E. Abreu, Galveston (1966).[13]
Donald S. Brown, Dallas (1966).[14]

Council on Constitution and By-Laws

J. T. Billups, Chairman, Houston (1967).

committee was enlarged to nine members.
11. Appointed June, 1964, to fill vacancy created when the committee was enlarged to nine members.
12. Elected on April 26, 1964, to fill vacancy.
13. Appointed April, 1964, to fill vacancy.
14. Appointed May, 1964, to fill vacancy.

Stephen R. Lewis, Galveston (1965).
J. J. Andujar, Fort Worth (1965).
George M. Jones, Dallas (1967).
M. D. Thomas, El Paso (1966).
Jack B. Lee, San Antonio (1966).[15]
Robert Mayo Tenery, Waxahachie (1966).[16]
(Ex officio members: Speaker and Vice Speaker of the House of Delegates.)

Council on Medical Education and Hospitals

G. V. Brindley, Jr., Chairman, Temple (1965).
John W. Lanius, Dallas (1965).
A. J. Gill, Dallas (1967).
M. H. Crabb, Fort Worth (1966).
Weldon G. Kolb, La Marque (1966).
Bryan V. Williams, Houston (1967).[17]
Jack Partain, San Antonio (1965).[18]

Committee for AMA Education and Research Foundation (standing)
Herbert A. Bailey, Chairman, Dallas (1967).
Edward S. Crocker, Houston (1965).
E. M. Addison, Huntsville (1965).
Douglas B. Black, Lamesa (1965).
H. E. Whigham, McAllen (1967).
Nelson L. Schiller, Austin (1967).
J. C. Terrell, Stephenville (1966).
P. W. Voltz, Jr., San Antonio (1966).
C. V. Chenoweth, Tyler (1966).

Advisers to Student American Medical Association
Homer A. Taylor, Jr., Houston (Baylor).
Charles LeMaistre, Dallas (Southwestern Medical School).
E. Sinks McLarty, Galveston (University of Texas Medical Branch).

Committee on Medical Careers (standing)
Peter Kamin, Chairman, San Antonio (1966).
John Wensley, Longview (1965).
Harry M. Shytles, Sherman (1965).
A. Fletcher Clark, Jr., San Antonio (1965).
John Bonnet, Temple (1967).
John B. Truslow, Galveston (1967).
Walter A. Brooks, Quanah (1967).
Gordon Black, El Paso (1966).
Norman L. Renfro, Corpus Christi (1966).[19]

Committee on Liaison with Medical Schools (standing)
Russell L. Deter, Chairman, El Paso (1966).
Frank Harrison, Dallas (1966).
Harry Spence, Dallas (1965).
L. Bonham Jones, San Antonio (1966).
Stanley Olson, Houston (1965).
May Owen, Fort Worth (1967).
Henry Renfert, Jr., Austin (1967).
Stephen Lewis, Galveston (1965).
Charles W. Yates, Rosenberg (1967).[20]

Committee on Hospital Accreditation (standing)
Ray L. Shepperd, Chairman, Burnet (1966).
Maynard Hart, El Paso (1965).
Hugh F. Rives, Jacksonville (1967).

15. Elected April 26, 1964, to replace David Wade, Austin, who resigned to become President-Elect.
16. Appointed May, 1964, to finish the unexpired term of Joe Donaldson, Pampa, who resigned.
17. Elected April 26, 1964, to replace James A. Greene, Houston, who resigned.
18. Elected April 26, 1964, to finish the unexpired term of Jack B. Lee, San Antonio, who resigned to become a member of the Council on Constitution and By-Laws.
19. Appointed June, 1964, to fill vacancy.
20. Appointed June, 1964, to replace W. H. Hamrick, Houston, who resigned.

R. E. Speegle, Garland (1967).
William F. Ross, San Benito (1966).
Drue O. D. Ware, Fort Worth (1967).[21]
Roy S. Bucy, Linden (1965).[22]
A. F. Herff, Jr., San Antonio (1965).[23]
Donald E. Fletcher, Wichita Falls (1966).[24]

Committee on Nursing Home Accreditation (special)
Ralph L. Coffelt, Waco.
Lloyd J. Gregory, Jr., Houston.
Arvil Ponton, Alpine.
Marshall D. Turnbull, Abilene.
R. R. Botkin, San Antonio.
Robert E. Higgs, Wichita Falls.[25]
Oliver M. Phillips, Fort Worth.[26]
Vacancy.
Vacancy.
Charles B. Dryden, Jr., Wichita Falls (ex officio).[27]

Committee on Nursing (standing)[28]
William D. Seybold, Chairman, Houston (1967).
Thomas J. Coleman, Fort Worth (1966).
E. Russell Hayes, Dallas (1965).
Walter B. King, Jr., Waco (1966).
Robert W. Shirey, Hillsboro (1965).
Emmett N. Wilson, Jr., Pearsall (1967).
Robert W. Grace, Dallas (1965).
Merle W. Delmer, San Antonio (1966).
Virginia H. Boyd, Abilene (1967).

Council on Medical Jurisprudence

John M. Smith, Jr., Chairman, San Antonio (1965).
Joe T. Nelson, Weatherford (1965).
Haden E. McKay, Humble (1967).
Tom Duke, Amarillo (1966).
Robert L. Daily, Wichita Falls (1966).
Courtney M. Townsend, Paris (1966).[29]
Doyle W. Ferguson, Dallas (1967).[30]

Committee on Military and Veterans Affairs (standing)
Milton V. Davis, Chairman, Dallas (1965).
James C. Whittington, Fort Worth (1967).
Richard H. Harrison, III, Bryan (1967).
James E. Watson, Jr., Houston (1966).
Ivey E. Lamberth, Tyler (1965).
George W. Tate, Longview (1966).
Joseph V. Land, Crockett (1966).[31]
Thomas G. Glass, Jr., San Antonio (1965).[32]
Andrew J. Magliolo, Dickinson (1967).[33]

Council on Medical Service and Insurance

Charles D. Bussey, Chairman, Dallas (1965).
William E. Sharp, Baytown (1965).
John E. Ballard, San Angelo (1965).
C. F. Jorns, Houston (1966).
J. G. Rodarte, Temple (1966).

21. Appointed April, 1964, to fill vacancy.
22. Appointed May, 1964, to fill vacancy.
23. Appointed June, 1964, to fill vacancy.
24. Appointed May, 1964, to fill vacancy.
25. Appointed June, 1964, to replace Charles B. Dryden, Jr., Wichita Falls, who became an ex officio member.
26. Appointed May, 1964, to fill vacancy.
27. Appointed ex officio member June, 1964.
28. Nine member standing committee created at 1964 Annual Session.
29. Appointed November, 1963, to finish unexpired term of N. L. Barker, Paris, and elected April 26, 1964, at Annual Session.
30. Elected April 26, 1964, to replace Floyd A. Norman, Dallas, who resigned.
31. Appointed June, 1964, to fill vacancy.
32. Appointed June, 1964, to fill vacancy.
33. Appointed June, 1964, to fill vacancy.

David O. Johnson, Austin (1967).
Robert F. Gossett, San Antonio (1967).[34]

Committee on Association Insurance Programs (standing)
Alford R. Hazzard, Chairman, Austin (1965).
G. J. Pruitt, Lufkin (1965).
A. L. Hewitt, Lubbock (1967).
C. C. Shotts, San Antonio (1967).
Donald S. Gibbs, Fort Worth (1966).
Charles W. Klanke, Houston (1966).
Laurance Nickey, El Paso (1966).

Committee on Professional Insurance (standing)
A. W. Bronwell, Chairman, Lubbock (1965).
R. E. Read, Lufkin (1965).
Bill Robins, Houston (1967).
Patrick H. Pappas, Lubbock (1966).
Edward G. Naslund, Amarillo (1966).
E. A. Cox, Wichita Falls (1966).
Porter K. Mason, Dallas (1965).
William F. Pipes, San Antonio (1967).
Frank L. Bynum, Fort Worth (1967).[35]

Committee on Health Insurance (standing)
A. Rex Kirkley, Chairman, Dallas (1965).
William M. Sherrill, Houston (1965).
Jack L. Eidson, Weatherford (1965).
E. A. Maxwell, San Antonio (1967).
Norman West, Hillsboro (1967).
Mario E. Ramirez, Roma (1967).
George L. Walmsley, Baytown (1966).
John Douglas, Austin (1966).
Billy B. Markette, San Antonio (1966).

Subcommittees of Committee on Health Insurance:
Committee on Liaison with Blue Shield (standing)
Everett C. Fox, Chairman, Dallas (1965).
R. W. Kimbro, Cleburne (1965).
Harvey Renger, Hallettsville (1965).
Olin B. Gober, Temple (1967).
R. H. Bell, Palestine (1967).
J. B. Copeland, Austin (1967).
E. A. Rowley, Amarillo (1966).
Denton Kerr, Houston (1966).
James A. Hallmark, Fort Worth (1966).

Appointees of Hospitals-Insurance-Physicians Joint Advisory Committee
Charles D. Bussey, Dallas (1965).
William M. Sherrill, Houston (1965).
Ernest A. Maxwell, San Antonio (1965).

Committee on Workmens Compensation Insurance (standing)
Edward T. Smith, Chairman, Houston (1965).
J. B. Chester, Dallas (1965).
Richard C. Bellamy, Liberty (1967).
Ralph E. Donnell, Jr., Abilene (1966).
James L. Mims, Jr., San Antonio (1966).
Edward F. Thomas, Amarillo (1966).
W. Harry Ledbetter, Wichita Falls (1967).[36]
Robert W. Milam, McAllen (1967).[37]
William O. Hargrove, Fort Worth (1965).[38]

Council on Public Relations and Public Service

Glenn D. Carlson, Chairman, Dallas (1965).

34. Elected April 26, 1964, to replace Gail Medford, Lufkin, who had completed three consecutive terms.
35. Appointed May, 1964, to fill vacancy.
36. Appointed May, 1964, to fill vacancy created when the committee was enlarged to nine members.
37. Appointed May, 1964, to replace F. C. Rehfeldt, Fort Worth, who had completed three consecutive terms.
38. Appointed May, 1964, to fill a vacancy created when the committee was enlarged to nine members.

G. M. McCaskey, Corpus Christi (1965).
J. T. Ainsworth, Houston (1967).
Van D. Goodall, Clifton (1966).
Carroll T. Adriance, Galveston (1967).[39]
Ben M. Primer, Sr., Austin (1965).[40]
O. E. Harper, Abilene (1966).[41]

Committee on Aging (standing)
F. G. Dorsey, Chairman, Houston (1967).
C. J. Ruilmann, Austin (1965).
John Dupre, Levelland (1965).
Edward J. Lefeber, Galveston (1965).
Elizabeth Gentry, Austin (1967).
Ernest W. Keil, Austin (1966).
Marvin Tavarez, McAllen (1967).[42]
John M. Church, Fort Worth (1966).[43]
A. Bryan Spires, Jr., Taylor (1966).[44]

Committee on Disaster Medical Care (standing)
Ben A. Merrick, Chairman, Dallas (1967).
Joe R. Shaeffer, San Antonio (1965).
T. June Melton, Midland (1965).
W. D. Nicholson, Freeport (1965).
J. L. Johnson, Amarillo (1966).
O. A. Fly, Jr., Houston (1967).
Joseph Selman, Tyler (1966).
Robert A. Dennison, Jr., Austin (1967).
Charles R. Queen, Edinburg (1966).[45]

Committee on Medicine and Religion (standing)[46]
Edwin Goodall, Chairman, Breckenridge (1967).
Frank Cohen, Fort Worth (1967).
John P. McGraw, Houston (1967).
Robert F. Boverie, El Paso (1966).
J. Layton Cochran, San Antonio (1966).
Norman E. Halbrooks, Tyler (1966).
Joseph F. Peters, Jr., Alvarado (1965).
Robert J. Kellar, Weslaco (1965).
Grant F. Begley, Fort Worth (1965).
Milford O. Rouse, Dallas (ex officio).
Travis Smith, Abilene (ex officio).
Howard O. Smith, Marlin (ex officio).

Committee on Transportation Safety (standing)
Heinrich Lamm, Chairman, Harlingen (1965).
Otto Lippmann, Austin (1965).
William H. Neil, Fort Worth (1965).
Boyd D. Alexander, Waco (1967).
Charles H. Wilson, Wichita Falls (1966).
Vernon V. Goss, Tyler (1966).
Dean C. Kipp, Dallas (1967).[47]
Oren H. Ellis, Jr., El Paso (1966).[48]
Vacancy (1967).

Committee on Industrial Health (standing)
Robert J. Potts, Chairman, Dallas (1966).
Robert A. Wise, Houston (1965).
G. V. Launey, Dallas (1965).
John C. Parsons, San Antonio (1965).
Jesse G. Burdick, Pasadena (1967).
F. W. Wilson, Port Arthur (1966).
E. E. Baden, Raymondville (1966).

39. Appointed December, 1963, to finish unexpired term of Courtney M. Townsend, Paris, and elected April 26, 1964, at Annual Session.
40. Elected April 26, 1964, to finish unexpired term of John H. Bohmfalk, San Antonio, who resigned.
41. Elected April 26, 1964, to finish term expiring in 1966.
42. Appointed May, 1964, to fill vacancy.
43. Appointed May, 1964, to fill vacancy.
44. Appointed May, 1964, to fill vacancy.
45. Appointed June, 1964, to fill vacancy.
46. Formerly special committee. Elevated to standing status with membership of nine at 1964 Annual Session.
47. Appointed May, 1964, to fill vacancy.
48. Appointed July, 1964, to fill vacancy.

E. Ross Kyger, Jr., Fort Worth (1967).[49]
Vacancy (1967).

Committee on Public Health (standing)
Sam A. Nixon, Chairman, Floresville (1966).
Lorence W. Feller, Fredericksburg (1965).
Charles A. Pigford, Houston (1965).
W. V. Bradshaw, Jr., Fort Worth (1966).
J. E. Peavy, Austin (1966).
George L. LeBeau, Jr., Wichita Falls (1967).
William H. Wade, El Paso (1967).
Thomas Romo, Jr., San Antonio (1965).[50]
Ivan W. Kuhl, McAllen (1967).[51]

Committee on Rural Health (standing)
Curtis R. Haley, Chairman, San Augustine (1966).
J. G. Sanders, Bremond (1965).
Charles L. Bishop, Ganado (1965).
Lloyd Sherrill, Ozona (1967).
George V. Pazdral, Somerville (1967).
Dennis W. Short, Commerce (1966).
Erwin G. Pink, Frisco (1966).
Harry Tubbs, Post (1967).[52]
Ed. W. Schmidt, Pecos (1965).[53]

Committee on School Health (standing)
Edwin L. Rippy, Chairman, Dallas (1967).
R. K. Arnett, Lufkin (1965).
A. G. Arrant, Abilene (1965).
John W. Stack, Mesquite (1966).
E. E. Addy, Jr., Cisco (1966).
R. M. Hampton, Pampa (1966).
R. L. Penn, Jr., Midland (1965).[54]
Percy E. Luecke, Jr., Dallas (1967).[55]
James W. Caldwell, McAllen (1967).[56]

Council on Scientific Advancement

J. E. Miller, Chairman, Dallas (1965).
Herbert C. Allen, Jr., Houston (1965).
John W. Middleton, Galveston (1965).
Howard R. Dudgeon, Waco (1967).
Robert E. Leslie, El Campo (1966).
William J. McGanity, Galveston (1966).
P. C. Talkington, Dallas (1967).

Committee on Athletics (standing)
Robert G. McCorkle, Chairman, Houston (1966).
Harry W. Slade, Waco (1966).
Charles Sadler, Amarillo (1967).
J. Frank Beall, Nacogdoches (1965).
Louis Levy, Fort Worth (1966).
Alfred E. Voigt, El Paso (1965).
William Horn, Jr., Fort Worth (1967).
Dan Spivey, Lufkin (1967).
Vacancy (1965).

Committee on Cancer (standing)
Howard R. Dudgeon, Chairman, Waco (1965).
Tom B. Bond, Fort Worth (1965).
John F. Thomas, Austin (1965).
William S. Brumage, Austin (1967).
W. Q. Budd, Amarillo (1966).
Jabez Galt, Dallas (1966).

49. Appointed May, 1964, to fill vacancy.
50. Appointed April 6, 1964, to fill vacancy created by death of J. C. Ellington, San Antonio, in October, 1963.
51. Appointed June, 1964, to replace Ben Primer, Sr., Austin, who had completed three consecutive terms.
52. Appointed June, 1964, to replace T. T. Walton, Jr., Bryan, who resigned.
53. Appointed May, 1964, to fill vacancy.
54. Appointed November, 1963, to fill vacancy created by resignation of Zenas W. Hutcheson, Jr., Andrews.
55. Appointed May, 1964, to replace H. C. Chancellor, Honey Grove, who resigned.
56. Appointed May, 1964, to replace M. A. Childers, San Antonio, who resigned.

A. K. Morris, Baytown (1967).[57]
Norman H. Jacob, Jr., San Antonio (1966).[58]
R. H. Chappell, Texarkana, Ark.-Tex. (1967).[59]

Committee on Cardiovascular Diseases (standing)
Robert E. Leslie, Chairman, El Campo (1965).
S. W. Thorn, Houston (1965).
W. Frank McKinley, Marlin (1967).
William B. Adamson, Abilene (1966).
John R. Derrick, Galveston (1966).
K. C. Sherman, Harlingen (1967).
John M. Verosky, El Paso (1966).
James E. Kreisle, Austin (1967).
T. T. Walton, Jr., Bryan (1965).[60]

Committee on Maternal Mortality (standing)
William J. McGanity, Chairman, Galveston (1966).
J. C. Rucker, Jacksonville (1965).
Donald M. Gready, Houston (1965).
Fred W. Lurting, Big Spring (1965).
Stephen W. Cobb, Dallas (1965).
R. P. McDonald, Fort Worth (1967).
Henry C. McGrede, Jr., Longview (1967).
Warren T. Kable, Jr., Wichita Falls (1967).
J. S. Lancaster, Austin (1966).
William E. Strozier, San Antonio (1966).
Emmett K. Blewett, Austin (1966).
James R. Morgan, El Paso (1967).
Matthew H. Talty, Jr., Houston (1965).
John B. Miller, Brownsville (1967).[61]
John E. Kirschvink, Taylor (1966).[62]
Peggy Joyce Whalley, Dallas (ex officio).
Carl F. Moore, Austin (ex officio).
Lt. Col. Charles E. Gibbs, Lackland AFB (ex officio).[63]

Committee on Mental Health (standing)
P. C. Talkington, Chairman, Dallas (1965).
Ivan Bruce, La Marque (1965).
George Constant, Victoria (1965).
P. C. Palasota, Abilene (1967).
Leroy A. Boriack, Corpus Christi (1966).
Holland C. Mitchell, Waco (1966).
Charles H. Brown, Wichita Falls (1966).
C. B. Goolsby, Crockett (1967).[64]
H. L. McCauley, Jr., Fort Worth (1967).[65]
Dorothy Wyvell, Midland (ex officio).[66]

Committee on Nuclear Medicine (standing)
Herbert C. Allen, Jr., Chairman, Houston (1965).
E. E. Anthony, Jr., Fort Worth (1965).
C. C. Shullenberger, Houston (1965).
J. Allen Chamberlin, Houston (1967).
Lloyd R. Hershberger, San Angelo (1967).
Elbert DeCoursey, San Antonio (1966).
J. R. Maxfield, Jr., Dallas (1966).

Committee on Nutrition (standing)
Ralph G. Greenlee, Chairman, Midland (1965).
William Levin, Galveston (1965).

57. Appointed May, 1964, to replace R. Lee Clark, Jr., Houston, who had completed three consecutive terms.
58. Appointed May, 1964, to fill vacancy.
59. Appointed July, 1964, to fill vacancy.
60. Appointed June, 1964, to replace David Miesch, Paris, who resigned.
61. Appointed May, 1964, to replace W. H. Jondahl, Harlingen, who had completed three consecutive terms.
62. Appointed May, 1964, to replace R. E. Moon, San Angelo, who had completed three consecutive terms.
63. Appointed ex officio member May, 1964.
64. Appointed May, 1964, to replace A. D. Pattillo, Austin, who had completed three consecutive terms.
65. Appointed May, 1964, to replace Dorothy Wyvell, Midland, who had completed three consecutive terms.
66. Appointed ex officio member June, 1964.

D. J. Sibley, Austin (1965).
Robert S. Nelson, Houston (1967).
Per H. Langsjoen, Temple (1966).
William J. Block, San Antonio (1966).
Charles Robinson, Fort Worth (1966).
Charles M. Manhoff, San Antonio (1967).[67]
Walter A. Sievers, Gonzales (1967).[68]

Committee on Rehabilitation (standing)
Edward Krusen, Jr., Chairman, Dallas (1965).
Oscar Selke, Houston (1966).
Ridings E. Lee, Dallas (1965).
Ira Budwig, El Paso (1967).
D. J. Turell, Houston (1966).
Robert A. Murray, Temple (1966).
Wade H. Lewis, San Antonio (1965).[69]
Franklin W. Yeager, Corpus Christi (1967).[70]
Robert E. Key, Fort Worth (1967).[71]

Committee on Tuberculosis (standing)
John W. Middleton, Chairman, Galveston (1966).
R. B. Morrison, Austin (1965).
James L. Reagan, Beeville (1965).
Samuel Topperman, Tyler (1967).
Richard F. Allison, Sanatorium (1967).
David McCullough, San Antonio (1966).
Daniel E. Jenkins, Houston (1966).
Howard Barkley, Houston (1967).[72]
Charles A. LeMaistre, Dallas (1965).[73]

Scientific Section Officers

Digestive Diseases
Marcel Patterson, Chairman, Galveston.
O. Roger Hollan, Secretary, San Antonio.

General Practice
George A. Burgess, Chairman, Eagle Pass.
Nelson L. Schiller, Secretary, Austin.

Internal Medicine
Horace E. Cromer, Chairman, Austin.
Hugh H. Hanson, Secretary, Houston.

Obstetrics and Gynecology
Wayne F. Baden, Chairman, Temple.
Garland H. Lang, Secretary, Midland.

Ophthalmology
Everett R. Veirs, Chairman, Temple.
Harold E. Hunt, Secretary, Paris.

Otolaryngology
Ben T. Withers, Chairman, Houston.
Thomas W. Folbre, Secretary, San Antonio.

Pathology
Jack P. Abbott, Chairman, Houston.
George Race, Secretary, Dallas.

Pediatrics
David M. Mumford, Chairman, Houston.
William S. Conkling, Secretary, Navasota.

Public Health
William R. Ross, Chairman, San Antonio.
Charles A. Pigford, Secretary, Houston.

Radiology
George F. Crawford, Chairman, Beaumont.
Winston E. Riley, Secretary, Corpus Christi.

Surgery
R. H. Baskin, Chairman, Waco.
A. O. Singleton, Secretary, Galveston.

67. Appointed May, 1964, to fill vacancy.
68. Appointed July, 1964, to fill vacancy.
69. Appointed May, 1964, to fill vacancy.
70. Appointed May, 1964, to fill vacancy.
71. Appointed May, 1964, to fill vacancy.
72. Appointed June, 1964, to fill vacancy.
73. Appointed June, 1964, to fill vacancy.

Lubbock Lectures Slated For September 26

The Lubbock Surgical Society lectureship on pediatric surgery and the fifth annual Methodist Hospital lectureship in medicine will be held in Lubbock on Sept. 26 at the Koko Palace convention center.

Subject of the medical lecture is hypertension, and guest lecturers will be Dr. Irvine H. Page of Cleveland and Dr. Thomas A. Stamey of Palo Alto, Calif. Dr. Mark Ravitch of Baltimore and Dr. William H. Clatworthy of Columbus, Ohio, are the featured speakers for the surgical society lectures.

Entertainment for those attending will include the Texas University vs. Texas Tech football game at 8 p.m.

Additional information may be obtained from Oren Art, director, development and public relations, Methodist Hospital, 3615 19th Street, Lubbock.

Texas Junior Colleges Train Medical Assistants

Several Texas junior colleges offer training for medical assistants and medical secretaries.

A list prepared by the education committee of the American Association of Medical Assistants includes Del Mar College, Corpus Christi; San Antonio College, San Antonio; Southwestern Union College, Keene; and Temple Junior College, Temple.

All are accredited institutions.

"I'd like to see the Surgeon General appoint an advisory committee to study gluttony."

AMA Occupational Health Congress Scheduled for Houston Sept. 26-27

HOUSTON is host Sept. 26-27 to the American Medical Association's 24th annual Congress on Occupational Health.

Welcoming to Texas those concerned with occupational health problems will be Dr. M. O. Rouse of Dallas, Speaker of the House of Delegates of the AMA, and Dr. Max E. Johnson of San Antonio, President of Texas Medical Association. The two-day meeting is being held at the Rice Hotel.

Saturday morning's opening session will be devoted to a discussion of "Cardiology in Industry." Physician participants include Dr. George Burch, chairman, Department of Medicine, Tulane University School of Medicine, New Orleans; Dr. Fredrick Stare, professor of nutrition, Harvard University School of Public Health, Boston; Dr. John Moyer, professor and chairman, Department of Medicine, Hahnemann Medical College and Hospital, Philadelphia; Dr. Raymond Pruitt, professor and chairman, Department of Internal Medicine, Baylor University College of Medicine, Houston.

Immediately preceding the noon recess the 1963 Physician's Award of the President's Committee on Employment of the Handicapped will be presented to Dr. John S. Young, Denver, for his outstanding work in the employment of the handicapped. Making the presentation will be Harold Russell, Washington, D. C., chairman of the Committee.

The conferees will tour the new NASA Manned Spacecraft Center Saturday afternoon and will be told of the progress in America's Gemini and Apollo space programs by Drs. C. A. Berry, chief of the Center, and W. R. Hawkins, assistant chief of occupational medicine. An astronaut will address the gathering.

A symposium on "Treatment of Burns" will open the Sunday morning program. Participants will include members of the Department of Surgery at the University of Texas Medical Branch including Drs. T. G. Blocker, Jr., Curtis P. Artz, S. R. Lewis, and J. B. Lynch.

The morning session will close with a three-point discussion of "Problems of General Physicians." Topics and speakers include "Investigation and Medical Testimony on Death from Doubtful Causes," Dr. J. A. Jachimczyk, chief medical investigator, Harris County; "The Nurse Who Works Alone," Nathan Hershey, LL.B., Pittsburgh; and "A General Practice Plan for Periodic Health Examinations in Very Small Employee Groups," Dr. V. C. Baird, medical director, Humble Oil and Refining Company, and vice chairman of the AMA Council on Occupational Health, Houston.

Pesticide poisoning, toxic exposures, and health programs for radiation workers will be the subjects covered in a Sunday afternoon symposium entitled "Environmental Health Services." Speakers and topics include Dr. R. T. Johnstone, Los Angeles, "Diagnosis and Treatment of Pesticide Poisoning"; James Hammond, chief industrial hygienist, Humble Oil and Refining Co., Houston, "Methods of Surveying Toxic Exposure in Occupational Environment"; and Dr. W. D. Norwood, Richland, Wash., "Health Program for Radiation Workers."

The concluding session will be devoted to a discussion of the practical expectations for rehabilitation of the severely impaired person, with case presentations from the Baylor Rehabilitation Center by Drs. W. A. Spencer and Lewis A. Leavitt.

The Texas Industrial Medical Association, the Louisiana Industrial Medical Association, and possibly others, will utilize the Congress for their annual scientific sessions.

The Congress is acceptable for accredited hours of study for the members of the American Academy of General Practice.

Registration will take place at the Rice Hotel. There is no fee. All persons with an interest in occupational health are invited to attend.

Hotel reservations should be made directly with the Rice Hotel where a block of rooms has been reserved for those attending this meeting.

M. D. Anderson To Undergo 65 Percent Enlargement

THE UNIVERSITY of Texas M. D. Anderson Hospital and Tumor Institute has received approximately $7,759,000 to support a 65 percent enlargement of the cancer research institution's physical plant amounting to an increase of 230,000 square feet.

Dr. R. Lee Clark, Anderson's director and surgeon-in-chief, said the $7,759,000 came from private, philanthropic, and federal sources.

Final plans have been accepted by the University's Board of Regents. Invitations for bids will be made in the immediate future.

Funds allocated by federal agencies include $1,400,000 from the Hill-Burton Fund; $732,252 from the National Cancer Institute of the U. S. Public Health Service, and $1,898,748 from Health Research Facilities.

Dr. Clark said the Health Research Facilities grant was awarded on the premise that the M. D. Anderson Hospital and Tumor Institute, at the completion of its expansion program, will constitute one of the nation's leading cancer research resources.

Private and philanthropic organizations which have given funds for the building program include the estate of the late Mose Gimbel, $850,000; M. D. Anderson Foundation, $1,500,000; Houston Endowment, Inc., $750,000; Moody Foundation of Galveston, $200,000; Fondren Foundation, $100,000; Foley Foundation, $15,000, and miscellaneous sources, $313,000.

Additions are planned in four areas of the present building. Clinical laboratories and a radiation therapy section at the north end of the plant will be expanded and will be known as the Gimbel Building. A five-story addition at the end of the east wing will be the Basic Science Research Building. A subsurface driveway and an area above the present loading dock will be enclosed to provide more laboratory space.

New additions will increase the institute's educational potential and accelerate the research program. Extra space will permit the addition of new basic science programs in immunochemistry, clinical physiology, and experimental pharmacology and therapeutics. Existing programs in immunology and nuclear medicine will be expanded. Space will be provided for training students in the basic sciences, for laboratories for visiting scientists and for further development of a computer data processing system.

More room for medical specialty studies is envisioned, as is an expanded diagnostic clinic for new patients which will be separated from presently overcrowded follow-up and special service clinics.

Additional storage space for research and treatment supplies will be provided. Some offices and departments will be relocated to place personnel next to their primary work areas.

Distinguished scientists will be added to the present staff to participate in increased programs of research, education, and patient care, Dr. Clark said.

The new building program, approved last year by the Board of Regents and in 1959 and 1961 by the Legislature, provides for critically needed space. Studies of space requirements, under consideration for the past eight years, were reviewed periodically to insure that the most modern laboratory facilities were incorporated in the expansion program.

Anderson's present physical plant, exclusive of land but including the building, its furnishings and equipment, is valued at $12,800,000. The structure contains a total of 350,000 square feet of floor space.

When built in 1954, the M. D. Anderson Hospital and Tumor Institute was one of the first cancer institutions to combine under one roof the three functions of patient care, research, and education. Innovations in function and design incorporated in the Anderson building have been copied in a number of hospitals and research institutions in the U. S. and abroad.

State Medical Societies Report Nursing Liaison

Findings of a questionnaire survey to determine the current status of nursing liaison activities within state medical societies have been released by the AMA Committee on Nursing.

Fifty of 52 state societies responded to the questionnaire sent out in early 1963. Forty-one of the 50 respondents then maintained liaison with the nursing profession in their state.

Texas Medical Association added a Committee on Nursing to its organization at the last annual session. The survey reported that committees on nursing or comparable groups are responsible for liaison with nursing organizations in 35 states.

Release Awaited Of Mental Health Plan

The Texas effort to develop a comprehensive statewide and community plan for mental health services has been in progress since shortly before the first of the year. Sometime this month the result of that effort, the proposed plan, is expected to be released to the public. It then will go to the Governor and the 1965 Legislature.

The new state plan for mental health services is being written by a 112-member planning committee made up of citizens throughout the state. (See January issue of *Texas State Journal of Medicine.*)

The actual planning work has been done by the statewide committee in five work sessions in Austin.

The federal government has provided funds for comprehensive mental health planning in all 50 states, including $182,000 for Texas.

Dallas To Host Heart Association

The Texas Heart Association will meet in Dallas Sept. 25-27 at the Sheraton-Dallas Hotel.

The program for Friday, Sept. 25, includes "Prevention of Recurrent Arrhythmias and Discussion of Maintenance Quinidine Dosage," Dr. Maurice Sokolow, San Francisco, Calif.; "Electric Shock in the Treatment of Arrhythmias," Dr. Paul M. Zoll, Boston; "Arrhythmias in Sudden and Unexpected Death," Dr. Raymond D. Pruitt, Houston; "Electrical Pacing of the Heart," Dr. Zoll; panel discussion on Recognition and Treatment of

Cardiac Arrhythmias, Dr. Joe O. Arrington, Lubbock, moderator, Dr. Leonard J. Flohr, Dallas, Dr. Milton R. Hejtmancik, Galveston, Dr. Pruitt, Dr. Zoll, and Dr. Sokolow;

"Hypertrophic Subaortic Stenosis—Clinical, Physiologic and Surgical Considerations," Dr. Eugene Braunwald, Bethesda, Md.; "Management of Cerebrovascular Insufficiency," Dr. Irving S. Wright, New York; "The Natural History of Hypertension—Use of a Portable Blood Pressure Recorder," Dr. Sokolow; "Catecholamines and Myocardial Function," Dr. Braunwald; and "The Effects of Hypertensive Agents on the Splanchnic Blood Flow," Dr. Fouad A. Bashour, Dallas.

Saturday's program will include "Anatomic and Hemodynamic Problems of the Left Ventricle and Its Outflow Tract," Dr. Jesse E. Edwards, St. Paul, Minn.; "A Critique on Aortic Valve Surgery, the Conservative Approach," Col. Hu Blake, San Antonio; "Total Valve Replacement," Dr. Denton A. Cooley, Houston; panel discussion on "Management of Problems of the Left Ventricular Outflow Tract," Dr. Alfred W. Harris, Dallas, moderator, Colonel Blake, San Antonio; Dr. Braunwald, Dr. Cooley, Dr. John R. Derrick, Galveston, Dr. Edwards, and Dr. Hugh E. Wilson, III, Dallas.

September Designated Sight Saving Month

September is Sight Saving Month, a time when the Texas Society for the Prevention of Blindness will conduct a statewide educational campaign to alert Texans to threats to their eyesight.

At present there are 24,900 blind persons living in Texas, and an estimated 1,750 individuals lose their sight each year. Much of this blindness can and should be prevented, states Dr. E. E. Garrett of Houston, state medical chairman of the Society.

Since its inception in 1955, the Texas Society has worked closely with the medical profession in developing glaucoma and preschool vision screening programs. The Society is also working for improved eye safety conditions in industry, at school, and in the home, as well as for better educational services for partially-seeing children.

MEDICOLEGAL NOTES

Telling Record Items to Insurance Company May Invade Patient Privacy

DISCLOSURE of items from a patient record to insurance companies without proper authorized consent in Texas may very well be an invasion of the right of privacy of that patient. For this reason, consent from the patient should be required before information from the patient record is made available to a third party. Before third parties, such as insurance companies, are given access to patient records and information with respect to patients, properly authorized consent from the patient should be first obtained. The only exception recognized in Texas is where the request is by a workmen's compensation carrier. In those cases, because of the structure of the Workmen's Compensation Law, the position has been taken that consent is implied. This does not mean, however, that a patient may not, even in workmen's compensation cases, request the physician or hospital not to make the information with respect to his physical condition available to the compensation carrier. Should the patient decline to make the information available to the compensation carrier, of course, the carrier is under no obligation to pay for the physical examination of the patient to the physician or for any hospital expenses. The physician and hospital would be obliged to look to the patient for the payment of his bill under these circumstances.

The underlying philosophy of the law in Texas with respect to disclosure of information from the patient record is that the relationship between the hospital and a patient is one containing the highest confidentiality. Though not recognized by statute in Texas as a "privileged communication," as between a hospital and a third party, the hospital should consider the wishes of its patients when deciding whether or not to release information from the patient record, and to do otherwise is to invite litigation.

An additional exception might be commented on here. This is the one contained in Article 5506(a), 4a. The important thing to remember with respect to this particular exception is that it applies only in cases where the hospital has filed a lien under the Hospital Lien Law. The Article reads as follows:

> Sec. 4a. Any person or persons, firm or firms, corporation or corporations legally liable, or against whom a claim shall be asserted for compensation for such injuries, shall be permitted to examine the records of any such association, corporation, or other institution or body maintaining such hospital in reference to such treatment, care and maintenance of such injured person, under such reasonable rules and regulations as such hospital may require, and the hospital record with respect to the injured person or persons may be admitted in evidence in any proceeding with respect to the recovery of damages.

With respect to who should give the consent, a man or woman in Texas over 21 years of age may give their consent. It is not necessary, though advisable, for a married man to consent to release of hospital information regarding his wife.

With respect to minors, either the mother or father may properly consent. The minor in Texas is anyone who has not attained the age of 21 years, except that marriage dissolves the disabilities of minority with respect to both males and females.

—PHILIP R. OVERTON, LL.B., Austin.

Postgraduate Courses

Anesthesiology. — Temple, September-May. Refresher courses in anesthesiology for the general practitioner will be conducted continuously, beginning each Monday during the months from September through May.

For additional information, physicians may write to Dr. J. Arnold Bargen, medical director, Scott and White Memorial Hospital and Scott, Sherwood and Brindley Foundation, Temple.

Blue Cross Survey Shows 78.3% Insured

A Blue Cross survey of its member hospitals in regard to the over age 65 group has shown that 25 per cent of the total inpatients are over 65 and that 78.3 per cent of all patients have some form of health insurance.

Of a total of 25,537 patients in 500 hospitals, 27.4 per cent had OAA only; 12.8 per cent had personal Blue Cross; 1.3 per cent, OAA plus Blue Cross; 7.8 per cent, OAA plus commercial; 28 per cent, commercial; 21.7 per cent, no insurance; and 1.1 per cent, other.

Texas Jaycees Undertake Project of VD Education

Venereal disease education, a statewide project of the Texas Junior Chamber of Commerce for the coming year, has been a successful undertaking of the Houston Jaycees since 1962. The Houston project is endorsed by the Harris County Medical Society and the City of Houston Health Department.

Requests for endorsement by Texas Medical Association of the statewide project have come from both the Texas Junior Chamber of Commerce and the Harris County Medical Society and, in accordance with established procedures, have been referred to the Committee on Public Health.

The ultimate goal of the Jaycee project, according to its chairman, Jack V. Casebolt of Houston, is to make venereal disease, specifically syphilis, an intolerable disease in the community and to encourage the schools to integrate venereal disease information into the curriculum so that young people will be informed about the dangers of these diseases.

The Houston project has included publication of an informative pamphlet, "Teenage Venereal Disease; A Problem in Houston"; a citywide meeting attended by 300 youth leaders, teachers, clergymen, and others who deal with young people; establishment of a speakers' bureau which has arranged for Jaycees to speak more than 50 times about VD to Houston groups; placement of venereal disease posters in the toilets of service stations, bars, night clubs, and restaurants in areas of the city where there is high incidence of the disease; placement of 310 posters in city buses; and interviewing of 1,281 people getting off the buses to evaluate the effectiveness of the posters.

American Fracture Association Announces Philadelphia Meet

The American Fracture Association will hold its annual meeting Oct. 4-8 in Philadelphia.

The program will be given by the orthopedic surgery section of Hahnemann Medical School, Temple University of Medicine, and various other medical centers of Philadelphia.

Physicians desiring additional information may write to Dr. W. Compere Basom, first vice president, American Fracture Association, 1220 Stanton Street, El Paso.

Medical Exams To Be Dec. 7-9

The next meeting of the Texas State Board of Medical Examiners when examinations will be given and reciprocity applications considered is scheduled for Dec. 7-9 at the Blackstone Hotel, Fort Worth.

Completed examination applications for graduates from United States medical schools must be filed 30 days prior to the meeting date, and those for graduates of foreign medical schools, 60 days prior to the meeting date. Completed reciprocity applications must be filed 60 days prior to the meeting date to be given consideration.

All applications should be filed with Dr. M. H. Crabb, secretary, Texas State Board of Medical Examiners, 1714 Medical Arts Bldg., Fort Worth.

Baylor-Rice Team To Develop Heart

A project to develop an implantable artificial heart has been launched by surgeons at Baylor University College of Medicine and engineers at Rice University.

Supported by an initial $653,324 grant from the Public Health Service, this unique research investigation is set up to cover a five-year period and could require as much as $4,500,000 in support of the project. Within the next three to five years surgeons at Baylor hope to undertake heart replacement using an artificial device.

Dr. Michael E. DeBakey, professor and chairman of the Department of Surgery at Baylor, will direct the work there and at the Methodist Hospital under a $395,000 grant. W. W. Akers, Ph.D., professor and chairman of the chemical engineering department at Rice will direct the $258,000 project at Rice.

Texans Elected to American College of Physicians

At the 45th annual session of the American College of Physicians held in Atlantic City in April the following Texas physicians were elected fellows of the College:

Dr. Eugene P. Frenkel, Dr. John W. Hyland, Dr. David E. Streitmatter, and Dr. Bryan Williams, Dallas; Dr. Victor H. Malagon-Diaz, McAllen; and Dr. Harvey B. Schneider, Houston.

Elected as associates to the College were the following:

Dr. George Henry Carman, Dr. Paul K. Conner, Jr., Dr. Arthur C. DeGraff, Jr., and Dr. Arthur Sherwin Weston, Dallas; Dr. Harry Eugene Sarles, Galveston; Dr. Thomas Turner Walton, Jr., Bryan; and Dr. James Fair Witten, Corsicana.

Dr. Alfred W. Harris of Dallas was elected Governor for Northern Texas. Dr. Hatch W. Cummings, Jr., of Houston is Governor for Southern Texas.

AMA Releases Results of Study On Cost of Medical Care

RESULTS of a broad three and one-half year study of the cost of medical care were released by the American Medical Association at its 113th Annual Convention in San Francisco.

Contained in four volumes, totaling 513 pages, the study examines the past and analyzes the present economic structure of the provision of medical care.

The research included a study of changing patterns of hospital care, based on some 34,000 medical records from 64 hospitals. Another project compiled lists significant medical advances of the past 25 years and analyzed the economic implication of five separate advances.

Also studied were the operations of 12 local professional review mechanisms which through physician audit of claim forms seek to secure the wisest use of prepayment and health insurance premiums. Other projects included examinations of the Consumer Price Index, the economics of solo and group practice, and conditions affecting the demand for medical care.

The study of changing patterns of hospital care was undertaken to determine whether advances in medical science, changes in patterns of illness, and a variety of other influences have had an effect on expenditures for hospital services.

The study showed that "medical care purchased in 1946 is not the same as that purchased in 1961. Even allowing for better quality, the conditions being treated are not the same. Patients are being treated in hospitals today for illnesses different from those in 1946. These changes in diagnoses also affect the cost of care, since today's most common diagnoses tend to utilize more expensive diagnostic or therapeutic agents and procedures."

Between 1946 and 1961, it was found that the average length of hospital stay fell from approximately 11 days to about 8 days. During this same interval, laboratory use increased by 99 per cent, x-ray diagnosis and therapy increased by 179 per cent, and the use of drugs increased by 56 per cent.

The conclusion was: "All this suggests that the treatment given the hospital patient in 1961 was more intense and complex than in 1946. More 'things' happen to him in a shorter length of time."

The study also showed that average patient charges for a stay in the hospital rose from $82 in 1946 to $259 in 1961—a 216 per cent increase. The per day charges for hospitalization, however, increased from $7.41 per day to $30.94 per day—a 318 per cent rise.

"It is obvious," the report said, "that the more services rendered in a given day, the greater will be the cost of that day to the patient."

Following are a few of the highlights of the study:

—The average age of those hospitalized increased by almost five years from 1946 to 1961. Cerebral thrombosis, arteriosclerosis, coronary heart disease, cataracts, and arteriosclerotic heart disease constitute an increasing percentage of the hospitalized population.

—Hospital admissions from rural areas increased from 30 per cent in 1946 to 37 per cent in 1961, indicating a trend of increasing utilization by persons residing in the nation's less densely populated locations.

—The five leading disease categories for which persons are hospitalized have not changed since 1946. These are: deliveries and complications of pregnancy, diseases of the respiratory system, diseases of the digestive system, injuries, and diseases of the genitourinary system.

—Hospitalized illnesses of greater proportional significance in 1961 than in 1946 include: diseases of the digestive, circulatory and genitourinary systems; injuries; diseases of the bones and organs of movement; neoplasms; congenital malformations; diseases of the nervous system and sense organs; and allergic, endocrine, metabolic, and nutritional diseases.

—There was a decreasing ratio of surgical to medical admissions, from approximately two-thirds in 1946 to one-half in 1961. At the same time, length of stay for surgical diagnoses decreased from 17.9 days to 12.7 days, while medical diagnoses decreased from 12.8 to 9.5 days.

—Admissions for acute coronary occlusion increased from .47 per cent in 1946 to 1.2 per cent in 1961, but at the same time a substantial decrease in the mean length of stay of 4.3 days was observed.

—Improved mortality figures were seen for virtually all diagnoses examined.

The study of significant medical advances showed that many of the items which are now considered essential to medical practice were not available a quarter-century ago. An analysis of five significant medical advances, documented not only their importance in providing medical care but also their economic and social benefits.

The in-depth cost-benefit analysis of the economic significance of the polio vaccine showed that during the period 1955-1961, an estimated $6.7 billion in medical care costs and gross lifetime income loss were avoided through use of the Salk vaccine. The cost of developing and administering the vaccine totaled $653 million. Thus, the economic benefit during this seven-year period was calculated as being more than $6 billion.

The Commission study found that techniques used for the early diagnosis of cancer of the uterus produced economic benefits by saving lives and by avoiding the higher cost of treatment for advanced cancer.

A study of the economic significance of surgical correction of congenital heart defects showed that because of the low incidence rate and the relatively high cost of treatment, the economic benefit realized by society may be less than that associated with other medical advances. However, the direct economic benefit per person may be great, since surgery is performed early in life and the correction in many cases enables the individual to survive and lead a productive life.

Another "in depth" analysis showed that new drugs for the treatment of tuberculosis have been primarily instrumental in dropping

the death rate for the disease from 55 per 100,000 population in 1935 to 6 per 100,000 in 1960. The principal drugs are streptomycin, para-aminosalicylic acid, and isoniazid.

In 1960, it was estimated that there were almost 76,000 survivors of tuberculosis and that their earnings in 1960 alone contributed $187.4 million to the Gross National Product. It was further estimated that an annual hospitalization cost of $50 million was avoided.

Another Commission study examined the economic impact of the use of disposable syringe-needle sets. It was estimated that hospitals realized net savings of $4.1 million in 1963 through use of this disposable product.

With increased future use of disposable syringe-needle and anticipated increases in hospital labor costs, it was estimated that hospitals would save $87 million between 1963 and 1970 through use of this product.

During the past decade, there has been a growing realization that the American system of private medical practice is dependent in a large measure, on the success of voluntary health insurance and prepayment plans.

The Commission's study of 12 professional review programs provides a description of some of the techniques presently used by medical societies and others to prevent the abuse of voluntary health insurance and prepayment plans.

The Commission's study of the Consumer Price Index showed that medical care goods and services have risen over the long inflationary period between 1935 and 1963 only slightly more than the average price levels of all goods and services—136 per cent compared with 123 per cent. The most rapid and extended rise occurred in the index for hospital daily service charge which increased 480 per cent since 1935.

It was found that the index for medical care, less hospital rates and hospitalization insurance, shows that the average price of the other medical care items rose considerably less than the general price level —91 per cent compared with 123 per cent. Physicians' fees increased 112 per cent and the index of prescriptions and drugs rose 43 per cent.

However, since 1957, the annual rate of increase has been declining gradually and steadily from 4.8 per cent for 1957-58 to 2.2 per cent for 1962-63.

GROUND-BREAKING ceremony for the Shriners Burns Institute of Shriners Hospitals for Crippled Children at Galveston was held June 23. Left to right are: Harold C. Close of Hartford, Conn., Imperial Potentate of the Shrine of North America; Dr. Truman G. Blocker, Jr., acting Executive Director, University of Texas Medical Branch; and Dr. Curtis P. Artz, Shrine Professor of Surgery and Chief Surgeon of the Shriners Burns Institute.

Shriners will provide funds and guidance of the institute, and the University of Texas Medical Branch will assemble the professional staff and assume the responsibility for the technical operation in achieving the objectives of the institute: patient care, research, and teaching.

Authors of the report were quick to point out that the Medical Care Price Index does not account for the vast amount of quality changes in medical care which have occurred.

The study of "demand for medical care" showed that "the variable that was most significant was average family income." The interpretation of the income effect is that for a $1,000 increase in annual income, $29 more would be spent for medical care.

The Commission also undertook a pilot study to determine the feasibility and possible methods of measuring differences, if any, in the cost of an illness to persons attended by physicians in two contrasting systems of medical care—solo and group practice.

"The pilot study reveals," the report said, "a surprisingly close relationship in the total overall costs of providing medical care under the two systems reviewed. The results, however, are not presented either as proof of a similarity in cost pattern under the two systems or to establish the economic advantage of one system over the other."

The Commission also developed an economic description of the "medical care industry" in which an effort was made to analyze the determination of the price of medical care in terms of the familiar economic forces of supply and demand.

New Books In TMA Library

Bondy, P. K., and Epstein, F. H., eds.: **Year Book of Endocrinology, 1963-4 Series**, Chicago, Year Book Medical Publishers, Inc., 1964.

Boniuk, M.: **Ocular and Adnexal Tumors, New and Controversial Aspects**, St. Louis, C. V. Mosby Co., 1964.

Cain, A. H.: **Cigarette Habit: An Easy Cure**, New York, Dolphin Books, Doubleday & Co., Inc., 1964.

Consultants Magazine: Common Complaints, Philadelphia, Smith Kline & French Laboratories, 1964.

Emmons, C. W., et al.: **Medical Mycology**, Philadelphia, Lea & Febiger, 1963.

European Society for the Study of Drug Toxicity: **Proceedings of the Symposium in Paris, January, 1963, Vols. 1-2**, Amsterdam, Netherlands, Excerpta Medica Foundation, 1963.

Cate, G. T.: **Teratology; Abstracts and Titles of Papers on Congenital Malformations Collected from the 1962 Medical Literature**, Amsterdam, Excerpta Medical Foundation, 1962.

Gilbert, C. R. A.: **Better Health for Women**, Garden City, N. Y., Doubleday & Co., Inc., 1964.

Hardy, J. D., ed.: **Physiological Problems in Space Exploration**, Springfield, Ill., Charles C Thomas, 1964.

Hershey, S. G.: **Shock**, Boston, Little, Brown, and Co., 1964.

Hippocrates: **Theory and Practice of Medicine**, New York, Philosophical Library, 1964.

Hirsh, B. D.: **Business Management of a Medical Practice**, St. Louis, C. V. Mosby Co., 1964.

Marti-Ibañez, F.: **Crystal Arrow**, New York, Clarkson N. Potter, Inc., 1964.

Master, A. M., et al.: **Electrocardiogram and Chest X-Ray in Diseases of the Heart**, Philadelphia, Lea & Febiger, 1963.

Missildine, W. H.: **Your Inner Child of the Past**, New York, Simon and Schuster, 1963.

O'Malley, C. D., ed.: **Andreas Vesalius of Brussels, 1514-1564**, Berkeley, Calif., University of California Press, 1964.

Owen, C. A., Jr.: **Diagnostic Radioisotopes**, Springfield, Ill., Charles C Thomas, 1959.

Raffel, S.: **Immunity**, 2nd ed., New York, Appleton-Century-Crofts, Inc., 1961.

Ross Conferences on Pediatric Research: Physical Diagnosis of the Newly Born, Report of the 46th Conference, Columbus, Ohio, Ross Laboratories, 1964.

Reich, W. J., and Nechtow, M. J.: **Pitfalls in Gynecologic Diagnosis and Surgery**, New York, McGraw-Hill Book Co., Inc., 1962.

Sliepcevich, E. M.: **School Health Education Study 1961-3 Summary Report**, Washington, D. C., School Health Education Study, 1964.

Strunk, W., Jr.: **Elements of Style**, New York, The Macmillan Co., 1959.

U. S. Department of Health, Education and Welfare, Public Health Service: **National Cancer Institute, Research No. 2, Virus-Cancer Research (Public Health Service No. 1130)**, Washington, D. C., Government Printing Office, 1964.

Wartman, W. B., ed.: **Year Book of Pathology and Clinical Pathology, 1963-4 Series**, Chicago, Year Book Medical Publisher, Inc., 1964.

New Films In TMA Library

MGS Educational Series A—1963, sound, color, six minutes.

THESE TEN FILMS are designed for use in special educational classes for educable mentally retarded children at the elementary level. The major educational objective is to help introduce and strengthen concepts related to physical and mental well-being: self-identification; self-orientation; learning to work with others; learning to share; learning who helps one at home, at school, and elsewhere, and how one can help others. Emphasis is given also to self-care (including health and safety), social skills, activities of daily living, work, recreation, and creative activity.

These can be used with parent groups and others to show special education classes at work, for in-service programs, for teacher training courses, for regular teachers, and in recruitment progams.

"He sure is paranoid."

The One Who Heals—1963, sound, color, 28 minutes.

A SERIES of dramatic vignettes which clearly show the relationship of physician and clergy in the total care of the individual patient compose this film. Each vignette indicates that faith is a factor in the treatment of a patient, and though problems are presented, their solutions are not given, rather, discussion is encouraged.

Texas, Today's Fun-Tier—1963, sound, color, 28½ minutes.

A DELIGHTFUL TOUR of the state of Texas in which the viewer enjoys some beautiful scenes in the Big Bend National Park, along the Mexican border, through the western part of the state, along the Gulf Coast, among the palm trees in the Rio Grande Valley, in central and northern Texas, and in the pine country of the eastern part of the state.

New Journal in TMA Library

The TMA Library now receives *Modern Treatment*, a new journal just started this year and published by Hoeber Medical Division of Harper & Row, New York.

A bimonthly publication, *Modern Treatment* in each issue deals with the complete treatment of a single disease.

Recent Deaths

DR. G. W. WALDRON

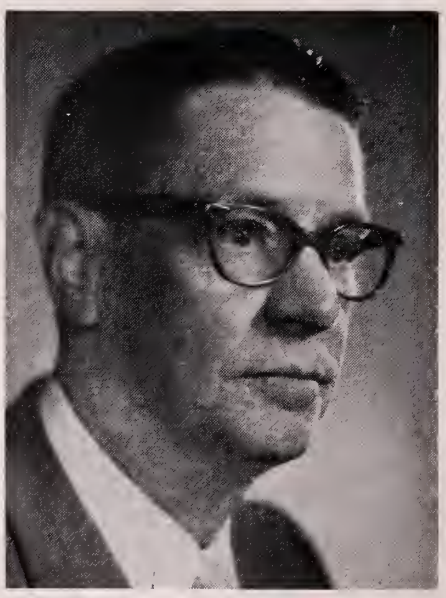
Dr. George W. Waldron, surgeon, teacher, and leader of medical organizations, died Feb. 15 in Houston.

The 59-year-old physician had served as the 97th President of the Texas Medical Association, and he had been governor of the American College of Surgeons nine years.

A clinical professor of surgery at Baylor University College of Medicine since 1948 and the University of Texas Postgraduate School of Medicine since 1950, he was the author of 35 published medical papers.

Dr. Waldron had been chief of surgery at Hermann Hospital since 1948; he had also served as chief of surgery at St. Joseph's and St. Luke's Episcopal Hospitals and as president of the Methodist Hospital staff.

Born in 1904 in Santa Barbara, Calif., his parents were Robert George and LaVera (Van Kirk) Waldron. He attended Rochester High School, Rochester Junior College in Rochester, Minn., and received a B. S. degree from the University of Minnesota in Minneapolis in 1926. During his senior year at the University of Minnesota Medical School, he received the Rollin E. Cutts Award for research in experimental surgery. He served his internship at Garfield Memorial Hospital in Washington, D. C., and returned to Rochester in 1929 as a fellow in surgery of the Mayo Foundation.



DR. G. W. WALDRON

He served as a first assistant in pathologic anatomy from 1931 to 1932 and as a first assistant on the surgical service of Dr. E. Starr Judd from 1933 to 1934. He left Rochester in 1934 to become senior medical officer in charge of a 43-bed hospital in Fort Peck, Mont. In 1937 he opened his practice of surgery in Houston.

In addition to the hospitals named, Dr. Waldron was a consulting surgeon at Memorial Baptist and Southern Pacific Hospitals, and a member of the thoracic surgical staff of the Houston Tuberculosis Hospital.

He married Rosalind Deakyne in 1935, who preceded him in death. They had one son, George Van Kirk Waldron. In 1950 Dr. Waldron married Olivia Morgan of Houston.

In 1961, a group of Houston doctors who had studied under Dr. Waldron formed the George W. Waldron Surgical Society to further postgraduate studies in surgery.

Dr. Waldron was a member of 25 medical organizations. Certified as a fellow of the American College of Surgeons in 1941, he represented that group to President Eisenhower's National Safety Council. From 1941 to 1945 he was a member of the Houston Selective Service Board. He served as president of the Harris County Medical Society in 1950, the Houston Surgical Society in 1960, and the Texas Surgical Society in 1953. In 1962 he was named chairman of the American College of Surgical Advisors Committee for Northeast Texas.

He also belonged to the Texas Traumatic Surgical Society, Southwestern Surgical Congress, Southern Surgical Association, American College of Chest Physicians, American Medical Association, Association of American Physicians and Surgeons, American Thyroid Association, International Society of Surgery, Alumni Association of the Mayo Foundation, and Nu Sigma Nu professional

medical fraternity.

His social and civic organizations included the Kiwanis Club, River Oaks Country Club, Houston Club, Allegro, Doctor's Club, and the Houston Chamber of Commerce.

He was a vestryman and senior warden of Palmer Memorial Episcopal Church and a former president of the Houston Episcopal Churchmen's Association.

Survivors include his wife; son, George Van Kirk of Santa Anna, Calif.; and a brother, Robert G. Waldron of Houston.

DR. H. TAYLOR, JR.

Dr. Holman Taylor, Jr., 47, Houston dermatologist, died of a heart attack at his home on June 14.

The son of the late Dr. Holman Taylor, Sr., and Mrs. Frances Lake Taylor, he was born on Oct. 18, 1916, in Corpus Christi. He moved as a child with his family to Marshall and later to Fort Worth. He attended Fort Worth schools, Texas Christian University, and the University of Texas where he received a B.A. degree in 1939. His M.D. degree was conferred by the University of Texas Medical Branch in 1942. He interned at Hermann Hospital, Houston, and did his residency at the University of Michigan's University Hospital.

He was a member of the American Medical Association, the Texas Medical Association, Harris County Medical Society, and the American Dermatological Association. He was certified by the American Board of Dermatology and Syphilology in 1947.

He began medical practice in Houston 15 years ago.

He belonged to the Methodist church, Kappa Sigma fraternity, and Sons of the American Revolution.

Dr. Taylor was crippled by poliomyelitis when he was a child, but he maintained his interest in sports through reading and television.

He was not married. Survivors include three aunts, Mrs. E. B. Hayes and Miss Emma Lake, of Marshall; Mrs. Christine Taylor Walker, Oklahoma City; and one uncle, Wood Lake, Marshall.

DR. W. C. BARNARD

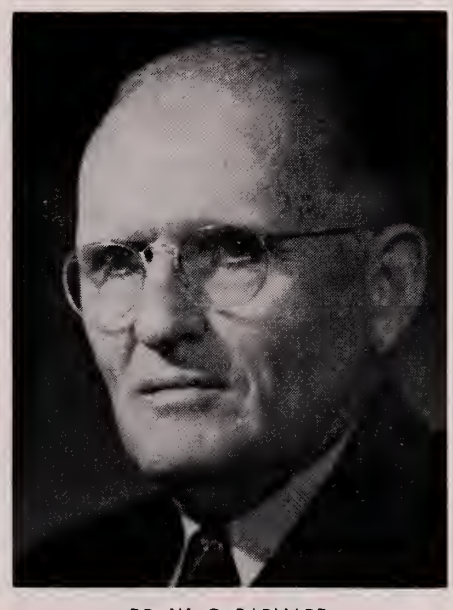
Dr. William Calvert Barnard, Corpus Christi's oldest active medical practitioner, died June 17 of a heart attack as he dressed to call on a patient. He was 83.

He had practiced medicine in Corpus Christi since 1911, except for time served in the Army Medical Corps in World War I.

Dr. Barnard was born in Cleburne, Tex., Aug. 24, 1881, the son of James and K. Calvert Barnard.

He received a B.A. degree from Texas Christian University in 1905 and his medical degree at the University of Louisville School of Medicine. He interned at St. Vincent's

DR. H. TAYLOR, JR.



DR. W. C. BARNARD

Hospital in Cleveland before opening his medical practice. At St. Vincent's he met Eleanor Erb whom he married June 19, 1913. She died in 1951.

He was an honorary member of Nueces County Medical Society and Texas Medical Association and belonged to the American Medical Association.

He was a Mason, Shriner, Knights Templar, an American Legionnaire, a member of the Woodmen of the World, and a member of the Chamber of Commerce.

Dr. Barnard was an avid sports fan.

He was deacon emeritus of the First Christian Church of Corpus Christi.

He is survived by three sons, Dr. James L. Barnard, Mayor of Corpus Christi, Dr. Cornell Barnard, a dentist, and William C. (Bill) Barnard of New York, a general executive with the Associated Press; and 11 grandchildren.

DR. G. H. JENKINS

Dr. Gaines Horace Jenkins, 88, died in a California hospital on May 29. He had practiced medicine at Bynum, Hill County, Tex., for 51 years before his retirement in 1957 when he moved to Lemon Grove, Calif., to live with his daughter, Mrs. J. B. Stevens.

Dr. Jenkins was born in Martling, Ala., March 8, 1876, the son of Francis Marion and Eliza Jenkins. There were seven sons in the family, of whom six became medical doctors.

He attended public schools in Martling and later attended the private institute of Capt. Henry Baker. He graduated from Grant University in 1901, a school which later merged with the University of Tennessee. He began his first year of practicing medicine at Howard, Tex., and stayed there five years. In 1906 he went to Bynum.

He was a member of the Texas and American Medical Associations through the Hill County Medical Society. He was elected to honorary membership in TMA in 1951.

He was a 32nd degree Mason, a member of the Independent Order of the Odd Fellows, the Shrine, and the Methodist Church.

Dr. Jenkins was married on June 17, 1906, to Miss Elizabeth Mae Parker, Waxahachie, Tex.; she died in 1947.

Three of their five children survive. They are Mrs. Pearl Dodson of Santa Monica,

DR. G. H. JENKINS

Calif., Mrs. Stevens and Miss Jimmie Fred Jenkins of Lemon Grove, Calif.; and two grandsons.

DR. M. L. MAFFETT

Dr. Minnie L. Maffett, Dallas gynecologist and surgeon, died in Honolulu, Hawaii, on May 26, 1964. Dr. Maffett became ill several weeks before her death while en route back to Dallas after attending the dedication ceremonies for the Dr. Minnie L. Maffett Nurses' Residence at the National Defense Medical Center at Taipei, Taiwan.

She was a native of Falls County where she received her education in the public schools before attending Sam Houston State Teachers College, the University of Texas, and the University of Texas Medical Branch. She received her M.D. degree in 1914.

A former teacher, she had been principal of the State Home High School in Corsicana and of a ward school in Cleburne.

She interned at New York Infirmary for Women and Children, and later received additional training in urology under Dr. Hunner, Johns Hopkins, Baltimore, and at New York Post Graduate Hospital. She was an associate professor of Gynecology and Obstetrics at Baylor University College of Medicine prior to its removal to Houston. She also had served as a professor of clinical gynecology at Southwestern Medical School and later as a consultant in gynecology there.

She was elected to honorary membership

DR. M. L. MAFFETT

in Texas Medical Association through the Dallas County Medical Society in 1963. She belonged to the American Medical Association; Southern Medical Association; Texas Association of Obstetricians and Gynecologists, which organization she served as secretary-treasurer, vice president, and as president; Central Association of Obstetricians and Gynecologists which she served as vice president and as a member of the council; and Alpha Epsilon Iota medical women's fraternity. She was also a Fellow of the American College of Surgeons.

Dr. Maffett was a member of the American Medical Women's Association, and was serving as a guest editor of their journal at the time of her death. She was also serving on the board of directors of the American Bureau for Medical Aid to China. She had recently served as advisor for workshops on health for the Pan-Pacific and Southeast Asian Women's Association in Tokyo, Japan, as well as in the same capacity for the International Federation of Business and Professional Women's Clubs in Southern Rhodesia, South Africa. In 1951, she was a member of a panel appointed by the State Department of the federal government for a study called Status of Women's Clubs in West Germany Five Years After Hitler. In connection with this study, she spent a number of months in Germany. She organized the health center at Southern Methodist University, Dallas, and served as its director until her retirement from that position several years ago.

She also was a member of Zonta International, Delta Kappa Gamma, and the University Women's Club of Southern Methodist University.

The Free Chinese Government had awarded her the Rosette of the Order of the Brilliant Star. She was a member of the Chinese Relief Legion, and a Certificate of Merit was awarded her by the American Bureau for Medical Aid to China. She was the recipient of the Zonta Club Award in 1944, and had received plaques from Theta Sigma Phi, and from the Texas Association of Obstetricians and Gynecologists.

Dr. Maffett was the first president of the Texas Federation of Business and Professional Women's Clubs. She served the National Federation of Business and Professional Women's Clubs as health chairman and as second vice president prior to her election to the presidency of that organization, an office she held from 1939 to 1944. It was during her tenure as president that the interest of this organization became focused upon nursing conditions in the Republic of China which culminated in their erection of the nurses' residence bearing her name.

Dr. Maffett was on the National Civilian Advisory Committee to the War Department during World War II, and for two years thereafter. She was chairman of the subcommittee whose activities pertained to health and recreation of the Women's Army Corps.

She was a member of Highland Park Methodist Church in Dallas.

Dr. Maffett is survived by four sisters, Mrs. Paul R. Winston and Mrs. James G. Pickens of Dallas, Mrs. Boyd Garrett of Groesbeck, and Mrs. Blanche Garrett of Center Point, as well as by several nieces and nephews.

DR. H. G. BEVIL

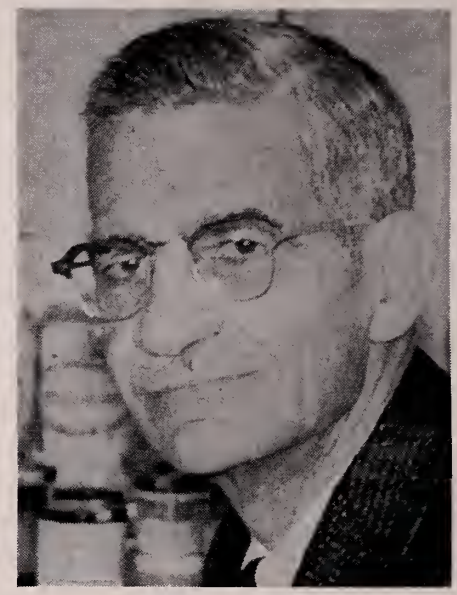
Dr. Henry Grady Bevil, Beaumont internist, died May 18 at the age of 70.

A native of Lee's Mill, Tex., he started to school in Kountze and completed his education at the University of Tennessee where he was graduated in medicine in 1915.

Dr. Bevil began practice in 1917 in Fisher, Ark., after interning at Memphis General Hospital, Memphis, Tenn. Other places of practice included Doucette, Tex., Silsbee, Tex., and Beaumont.

He was married in Memphis on Dec. 23, 1918, to Mary Eunice Patrick of Jonesboro,

DR. H. G. BEVIL



DR. G. A. HART

Ark. She was killed in a car accident Dec. 23, 1958. Doris Baron and Dr. Bevil were married Dec. 1, 1960.

During World War I he served in the Army Medical Corps at Fort Riley, Kan., and held the rank of first lieutenant.

Dr. Bevil, who had published several medical papers, was a fellow of the American College of Physicians and belonged to the American Association of Internal Medicine, American College of Angiology, American Geriatrics Society, American Medical Association, Texas Medical Association, Texas Academy of Internal Medicine, Jefferson County Medical Society, and the Beaumont Academy of Medicine.

He was a Mason, Shriner, and member of and deacon of Westminster Presbyterian Church in Beaumont.

Survivors include his wife; a daughter, Mrs. Genevieve Ream of Beaumont; two sisters, Mrs. J. W. Bettis of Shreveport and Mrs. F. M. Hughes of Houston, and one grandson, Gene Patrick Ream.

DR. G. A. HART

Dr. Gaylord Anthony Hart, 58, died June 22 in a Dallas hospital.

A Dallas resident for 42 years, he was a member of Dallas County Medical Society, Texas Surgical Society, Dallas Southern Clinical Society, Texas Medical Association, the American Medical Association, and Southwestern Surgical Congress. He was a fellow of the American College of Surgeons.

He was a graduate of Rice University and the University of Texas Medical Branch. He interned at Parkland Hospital and served his residency at Methodist Hospital, both in Dallas.

During World War II, he served in the medical corps for two years in the European theater. He was discharged as a full colonel.

Dr. Hart was a native of Hackensack, N. J. He was a member of Cochran Chapel Methodist Church in Dallas.

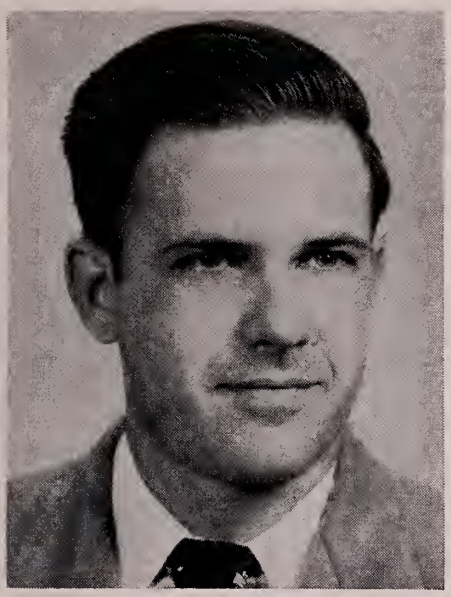
He is survived by his wife, the former Anne Colclough Longfellow of Lamar, S. C.; a daughter, Miss Jan Hart; a son, Gaylord A. Hart, Jr.; his mother, Mrs. James S. Hart; and a sister, Mrs. Fritz W. Glitsch, Jr., all of Dallas.

DR. C. L. GREEN

Dr. Charles Leslie Green, 39, died unexpectedly May 31 after an illness of several weeks.

Reared in East Texas, the El Paso physician was a graduate of Southern Methodist University and Southwestern Medical School. He interned at El Paso General Hospital and served residencies at Veterans Hospital, McKinney, and St. Paul's Hospital, Dallas.

Dr. Green was a pathologist and was certified by the American Board of Pathology in 1957. He was the author of several published medical papers.



DR. C. L. GREEN

Dr. Green was born Nov. 26, 1924, in San Antonio. He attended school in Jacksonville, Tex., and served in the Army from 1943-1945. Botany and golf were his hobbies.

He began a general practice in Silver City, N. Mex., but left it to take a residency in pathology. In 1956, he entered a group practice in El Paso. He also was associate pathologist at Hotel Dieu Hospital in El Paso, gynecologic consultant to William Beaumont General Hospital, El Paso, and clinical assistant professor of pathology at the University of Texas Southwestern Medical School, Dallas.

He served as treasurer of El Paso Medical Society in 1963 and was a member of the El Paso chapter of the American Cancer Society, American Society of Clinical Pathologists, Texas Society of Pathologists, Inc., the Academy of Anatomical Pathology, Texas Medical Association, and American Medical Association.

Dr. Green was a member of the board of stewards at Trinity Methodist Church in El Paso.

Survivors include his widow, Mrs. Anne Green; and four daughters, Charlotte, Carolyn, Katherine, and Margaret, all of El Paso.

DR. W. E. SCHULKEY

Dr. William E. Schulkey, 65, collapsed and died in the lobby of an Austin motel May 29. The San Angelo physician was visiting in Austin.

He was born Dec. 10, 1898, in Decatur. He attended schools and junior college in Decatur and took premedical and medical courses at Baylor University in Dallas, graduating in 1925.

After one year of internship at Baylor Hospital, Dr. Schulkey became a resident surgeon there. In 1927, he moved to San Angelo.

During World War I, he enlisted as a private in the Army and served overseas with the 36th Division participating in the Saint-Mihiel and Meuse-Argonne engagements. During World War II he served on the District Medical Advisory Board of the Selective Service and the District Appeals.

In December, 1951, a stroke ended his medical career.

An avid sportsman, he was a Baylor basketball letterman.

He was a fellow of the American College of Surgeons and a member of the Southwestern Surgical Congress. He was a member of the American Medical Association and an honorary member of Tom Green Eight Counties Medical Society and of the Texas Medical Association.

He was a member of the American Legion, president of the Kiwanis club, and vice president of the school board.

He is survived by his wife, the former Helen Lenore Manett of Dallas whom he married in 1926; a son, William E. Schulkey, Jr., of San Angelo; a sister, Miss Alma Schulkey of Dallas; a brother, Carl Schulkey, of Little Rock; and two grandchildren.

DR. W. E. SCHULKEY

DR. I. G. WILSON

Dr. Iva Gordon Wilson, 54, a radiologist, died June 14 in Big Spring after a long illness. Cause of death was glioblastoma multiforme.

Dr. Wilson was born March 6, 1910, in Bessmay, Tex. He graduated from Port Gibson College high school in 1926 and attended Millsaps College in Jackson, Miss. He received his M.D. degree from Tulane University School of Medicine in New Orleans. He specialized in radiology at the Ochsner Clinic in New Orleans.

He served in the Army Medical Corps in the Asiatic and Pacific theaters from 1942 to 1946 and retired with the rank of major.

Dr. Wilson married Evelyn Robins Dec. 4, 1937, at Beaumont. He was a member of Alpha Omega Alpha honorary medical fraternity, the American College of Radiology, the North American Society of Roentgenology, the American Medical Association, and had been elected to inactive membership in TMA in 1964. He was certified by the American Board of Radiology.

He was a Methodist.

Before moving to Big Spring in 1957 he had been in general practice in Beaumont.

His hobby was woodworking.

Survivors include his widow of Big Spring; two sons, Eric Wilson, Lubbock, and David Wilson, Beaumont; one granddaughter; his parents, Mr. and Mrs. O. E. Wilson, Pineland, Tex.; and one brother, C. P. Wilson, Houston.

DR. I. G. WILSON

DR. J. S. FINE

Dr. Jacob S. Fine, 53, a Dallas pediatrician since 1946, died June 10.

He was born Feb. 11, 1911, in Peabody, Mass., and attended Bowdoin College in Brunswick, Me., where he received a B.A. degree. He received his M.A. from Princeton and his M.D. degree from Yale University School of Medicine in 1939 and interned at Gallinger Municipal Hospital in Washington, D. C. He served residencies at Children's Hospital in Washington, D. C., at Herman Kiefer in Detroit, and at Children's Medical Center, Dallas, where he was chief resident in 1946.

Dr. Fine was a member of Temple Emanu-El.

He was a member of Dallas County Medical Society, Texas Medical Association, American Medical Association, and the Dallas Pediatric Society. He was certified by the American Board of Pediatrics.

He was a clinical instructor in pediatrics at Southwestern Medical Foundation and a staff member at Parkland Hospital and Children's Medical Center, Dallas.

He is survived by his wife, Mrs. Dorothy Fine of Dallas; two daughters, Miss Karen Fine and Miss Barbara Fine, both of Dallas; two sons, Richard Fine and Lawrence Fine, both of Dallas; and four sisters, Mrs. Theodore Bedrick of Crawfordville, Ind., Mrs. Nathan Silk of Falls River, Mass., Mrs. Morton Bedrick of New London, Conn., and Mrs. Abraham of Horvitz of New Bedford, Mass.

DR. J. S. FINE

COMING MEETINGS

American Medical Association, Clinical Meeting, Miami Beach, Fla., Nov. 29-Dec. 2, 1964. Annual Meeting, New York City, June 20-24, 1965. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

AUGUST

American Congress of Physical Medicine and Rehabilitation, Boston, Aug. 23-28, 1964. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Hospital Association, Chicago, Aug. 24-27, 1964. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

SEPTEMBER

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Medical Association Congress on Occupational Health, Houston, Sept. 26-27, 1964. Dr. Henry F. Howe, 535 N. Dearborn St., Chicago, Sec.

Southwest Regional Cancer Conference, Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, Houston, Sept. 26-27, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, San Francisco, April 12-15, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, New York, Oct. 24-29, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, New York City, June 17-21, 1965. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, New York, Oct. 25, 1964. Dr. Robert A. Peterman, 3 Craig Court, Totowa Boro, N. J., Sec.

American College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians, Los Angeles, Oct. 8-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Oct. 2-3, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 4-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Dermatological Association, Boca Raton, Fla., Feb. 25-March 2, 1965. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society, New York, May, 1965. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Medical Women's Association, New York, Oct. 16-20, 1964. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Neurological Association, Atlantic City, N. J., June 14-16, 1965. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 11-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., New Orleans, La., Oct. 8-10, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Chicago, Nov. 6, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons, Mexico City, Oct. 6-11, 1966. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico, Phoenix, Ariz., Dec. 9-12, 1964. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum. Dr. Purcell Smith, Jr., P. O. Box 7008, Little Rock, Ark., Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Grady Morrow, 1900 N. Oregon, El Paso, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Waco, Nov. 8, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. Leonard R. Robbins, 3618 Yoakum, Houston, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. Charles Peterson, 1333 Moursund, Houston, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, San Antonio, May 1-2, 1965. Dr. Fred P. Thomas, 2342 Maroneal, Houston, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section, San Antonio, April 29-May 2, 1965. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc., Houston. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 7-9, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Surgical Society, San Antonio, Oct. 4-6, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, Sec.

United States-Mexico Border Public Health Association, Los Angeles, April 26-29, 1965. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 9-10, 1965. Dr. J. E. Albright, 2481 Medical Center, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

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Of Friction, Medicine And a Musical Bedfellow

ALMOST SUBMERGED in what sometimes seems to be an endless round of unsigned insurance forms, unread journals, and incomplete hospital records, we are often tempted to speculate on what would have happened if the choice had not been made to practice medicine. There is the conceit of throwing over a vocation for a life of hunting, of beachcombing and fishing, perhaps in one of those attractively described Mexican resorts where you are supposed to be able to live for a pittance; or perhaps to follow the bent of an avocation and become, as some physicians have, writers, painters, musicians.

Yet, all of us were taught the concept in physiology that a muscle, or a heart, contracts with maximum efficiency when it works against a certain amount of resistance—not too much, but just enough. So, too, may it be with people. Most of us now in medicine are here, have always been here, because we are driven, and by a complex and subtle series of motivations. And endless series of days in which we eat, fish, loll in the sun, and eat again, far, far away even from the telephone and far, far removed from all thought of major responsibility makes a pleasant reverie. But having been accustomed to the demanding discipline of medicine, we would probably require something more complicated and arduous, something more stimulating, and if it were not quickly forthcoming we would manufacture it.

Demanding too is the discipline of art, particularly of music. Relatively few men have built a background in medicine and then turned from it to become known to us in later years for their abilities in performing or composing. The two disciplines, medicine and music, make admirable companions when one allows medicine first place, and music becomes the avocation. Indeed, the examples of Schweitzer and his baroque organ studies, and of Billroth with his viola playing, come quickly to mind. It may well be

that proficiency and public name in any field depend primarily not on genius and inspiration, but rather on hard and often dull work, whether it be in practicing the tying of surgical knots, or of orchestrating the treble and bass of a musical score. We should like to think that each of us has, or at least had, within him almost limitless capacities for development in many directions and that they lie fallow for want of world enough and time. There is Schopenhauer's dictum: "The world is will and idea."

There is one physician who did a fairly competent job in both the disciplines of medicine (he was a physician and professor of chemistry) and of music. About 100 years ago, Alexander Borodin, then in his early thirties, was writing his first symphony in addition to his professorial duties at the St. Petersburg Academy of Medicine. We know him primarily as a composer, and probably best for his music for *Prince Igor,* but we also know that his livelihood depended upon medicine, and that his musical output is small in volume. We infer from this that music occupied a secondary role in his life, at least from the standpoint of the demands of environment, and of time. Even though we do not know him now as a resplendent celebrity either in medicine or in music, he did at least combine the two fairly successfully and, we may hope, happily.

Making the hurried rounds between home and hospital, office and operating room, it is entertaining to examine one of the contemporary hallmarks for the definition of maturity: the realization and acceptance of one's limits. Are we truly mature only when we can accept that we are limited? And does the physiological principle of muscle contraction against resistance also apply to the optimum functioning of people? It is fun to wonder.

To say much the same things, but in a different way:

Borodin's "Polovetsian Dances" were
Surging
From the radio:
How sybaritic, sensing those
Opulent chords flood past
Knowing, even though I dawdled,
Deliberately courting traffic lights, hoping
They'd turn red, the wondrous
Barbaric passion still would
Sparkle in the air, unheard by me when I
Arrived at work and turned the motor off
Before the music finished.

Borodin was a physician too, and said to be
a good one.
If he carried to his work a fraction of the
Power of his music he at least was potent.
And did the music which was there when other
things intruded
Leaving fragments glistening unwritten,
uncaptured
In the air about him
Heighten the glory of his medicine?
Did he find dissatisfaction adding ferment to
his art?

What sort of man would he have been
Had he only written music, or never helped
the sick?
Had the forces of his intellect
Been goaded toward one goal
Would he resound, another Beethoven;
Uncover further microcosms than Pasteur's?
What sounds would echo in a later generation's ear:
Music, or revelations in the essence of the cell?
Or would it be that later listening
We heard nothing?

—VAN L. LAWRENCE, M.D., Houston.

The Neglected Infertile Couple

AT THE PRESENT rate of progress in medical science, it is inevitable that there will be delays in the widespread, practical application of new knowledge. There is less justification for failure to use our available knowledge and resources when the components have been well studied and widely recognized for many years. Yet such is the case with the evaluation and treatment of the infertile couple.

It may seem strange, in an era when overpopulation threatens to destroy civilization as we know it, to be greatly concerned about this problem, which affects one out of seven marriages. Yet it should be a basic privilege of every married couple to have the children they want when they want them; the interplay of personal wishes and environmental forces on the average will achieve a successful ecologic balance in the family, in the community, and in the culture. From a philosophic, as well as from a medical point of view, it is just as important to enable a couple to have children when they want them, as it is to help them limit their family to the personally desired number.

In 1950, the American Society for the Study of Sterility published in their journal

a summary of the *minimal* procedures considered necessary for the evaluation of a barren marriage. These criteria have recently been brought up to date and presumably serve as the framework for a proper diagnostic investigation for physicians active and interested in the problem of infertility. There are 59 Texas members in the American Society for the Study of Sterility. Undoubtedly, these members are consulted by only a small fraction of the couples who seek medical advice for infertility. By far the largest proportion of these patients first consult their generalist or obstetrician. And here is where the misfortune lies, for chances of finding a physician who is interested, let alone one who will carry out a thorough sterility survey, appear to be slim indeed. The story is monotonously the same: the wife has been given a general physical and pelvic examination, the husband has been sent uninstructed to a laboratory for a semen examination; and when these two procedures have failed to reveal any grossly apparent pathology, the couple has been reassured that "time" would cure all. It is difficult to understand why this routine, which was declared inadequate 15 years ago, should still be so widespread, even among gynecologists. Sterility work is time-consuming; it is occasionally tedious and often frustrating; it involves an often distasteful invasion of the patients' most sacred privacy; its yield of cures is disappointingly low. Yet cannot much the same be said of oncology, rheumatology, cardiology, or of medicine generally before the turn of the century?

A barren couple has not been given the medical attention they are entitled to unless, as a minimum, the husband's sperm population has been *adequately* sampled by repeated examinations of properly collected specimens, the wife's endocrine and ovulatory ovarian function have been *clearly* defined, tubal patency *established*, cervical and vaginal factors *carefully* investigated. This minimum workup, as is well known, has therapeutic value of its own. Pregnancies, for instance, occur far more often after salpingography than may be expected by chance. Testosterone rebound therapy for the mildly oligospermic male may be successful in only 10 percent of cases, but in those where it does work, a 100 percent baby may be the result. Irregular ovulation may be amenable to improvement by judicious use of the variety of potent oral estrogens and progestins now available; even more powerful agents such as clomiphene citrate and human gonadotropin are just over the horizon. Cervical and vaginal hostility to spermatozoa may result from unfavorable bacterial flora which can be altered by antibiotics or even simpler measures. Nor should it be forgotten that careful inquiry into the patients' knowledge of reproductive physiology and procedure, followed by gentle, informative and corrective discussion, may be all that is required to solve a problem that was previously fobbed off on Father Time.

—Joseph W. Goldzieher, M.D.,
San Antonio.

Nutritional Nonsense

One hears and reads much nonsense these days about nutrition. We are told about calories not counting, the therapeutic benefits of honey and vinegar, the "poison" some "misguided" individuals want to compel us to add to our water, and the wonders of natural foods and those fertilized organically.

Actually, calories do count; honey and vinegar do not have any unusual therapeutic effects, nor for that matter does most of the other "stuff" available in health food stores including natural foods and organically fertilized foods; and fluoride is a mineral nutrient, not a poison.

Good nutrition comes from good food available at any grocery store or supermarket. So-called "health foods" and nutritional supplements are not necessary to provide the best in good nutrition. Variety in foods eaten is probably the single most important factor in obtaining good nutrition—variety in meats, fish, cereals, dairy products, fruits, and vegetables—because through variety one is sure to receive all of the 50 some nutrients—vitamins, minerals, amino and fatty acids, and others—known to be necessary for good nutrition. And good nutrition is most important for good health. Poor nutrition has an important role to play in three diseases most important in family and personal health: the commonest types of cardiovascular disease (coronary heart disease and cerebral hemorrhage or stroke), obesity, and tooth decay.

Comment on the fluoridation of public water supplies is most important in any discus-

sion of nutritional nonsense. Fluorine is a mineral nutrient we must have to enable us to construct a dental enamel that is maximally resistant to decay. Fluoridation currently offers the only effective means to reduce dental decay by 50 percent or more. A few people will have less decay if they drastically reduce the intake of sugar, but only a few. Many individuals, including these few, benefit from fluoridation. Fluorides can be given in pill or drop form, and there are fluoridated toothpastes; but none of these methods begins to approach fluoridation of water supplies in effectiveness and economy. The beneficial results of fluoridation are permanent as long as one continues to receive fluoridated water. Furthermore, recent studies suggest that optimal fluoride ingestion may prove to be helpful in the prevention of osteoporosis, a common disorder of our older citizens. Fewer fractures, and quicker and stronger healing when they do occur, would indeed be a boon to persons of advancing years. Optimal intake of the mineral nutrient fluoride throughout life may help to achieve this goal.

Enrichment of certain foods—iodine to salt, vitamin D to milk, vitamin A to margarine, three key vitamins and one mineral to flour, and fluoride to water—has been most important in bringing better nutrition and better health to the entire family—both young and old—in America. Enrichment of a few basic staples helps give assurance that good nutrition is readily available to all via every grocery store or supermarket.

So-called "health food stores" with their "health foods," "nature foods," and "food supplements" are not necessary, and in my opinion, are a great waste of money. But, of more importance, the claims that are frequently made for many of their products are deceptive, frequently fraudulent, and represent quackery and charlatanism at their worst.

Family members in their advancing years are particularly gullible, because of the eternal hope of living longer and keeping one's health. Sea water and sea salts have long been popular with the food faddists. Like most health nonsense they are foisted primarily on old folks. The idea that sea salts have some beneficial effects is based on the ancient but widely held misconception that because sea water contains numerous mineral and trace elements, and the body needs some of these elements, it is healthful to take a little sea water every day. From this premise it is only a step to flagrantly false claims that sea water is a panacea for all the diseases of mankind. Ignored is the fact that the same mineral elements are present in common foods and that, with the possible exception of iodine, they are abundantly supplied by the ordinary diet.

This is just another adaptation of the false premise that modern foods are nutritionally inadequate—a theory that is relied on and promoted by practically all nutrition quacks and food faddists.

How do you tell a food or nutrition quack?

1. He or she usually goes around saying that our foods are no good nutritionally; "they are over-processed, devitalized, and poisoned."

2. They pretend to have some unusual understanding of health and disease. They claim that many of the ills of mankind—specific ones: heart disease, liver disease, kidney disease, poor skin, and others—are due solely to poor foods.

3. They frequently have something to sell —food supplements.

4. They promise quick and sure results and testimonials to back up their comments.

Because Americans are probably the most health-conscious people in the world, nutrition quackery has become one of the smoothest and best-organized rackets in the country. Five hundred million dollars is the take from the innocent, according to the American Medical Association. This business is carefully nurtured by a relatively small group of unscrupulous health charlatans. Competition is keen, and profits are high.

Proper eating to provide proper nutrition for the entire family—oldsters, youngsters, or the middle-aged—can be achieved from the wise selection of foods available in ordinary grocery stores. Consuming "health foods" or "dietary supplements" is not necessary. For most people these so-called health foods and dietary supplements are a waste of money, and many families have no money to waste. Unfortunately, all who can read or understand are good game for the health hucksters with their phony nutrition advice in books and pamphlets and on radio and television.

Variety and moderation in food consumption are the keys to proper nutrition. Avoid

the quack with his special foods, diet supplements, and phony advice.

—FREDRICK J. STARE, M.D., Boston.

The Hearing Aid Question

WITH MORE than passing interest, otologists have watched the advances in the hearing aid industry that have taken place simultaneously with forward strides in otology. The improvements both in the distribution and manufacture of hearing aids have been overshadowed by some of the startling surgical advances.

A properly fitted hearing aid remains the single most valuable tool in the rehabilitation of people with a hearing loss. Granted that there are still improvements to be hoped for, and indeed being worked upon at this very time, we should give credit where credit is due, and recognize that the hearing aid industry has made great strides. In some places, the relationship between the otologist and the hearing aid distributor are much better than in others. Some otologists decry some of the advertising and business practices that are resorted to by some hearing aid dealers, but at the same time persons in the business of distributing hearing aids have some legitimate criticisms to level at physicians who give advice to prospective hearing aid purchasers.

The best approach to the hearing aid question (and it is better referred to as a question than a problem) seems to be cooperation similar to that on limb prostheses followed by orthopedists and the prosthetics distributors. The objective is the same: rehabilitation of people with a hearing problem, and both physicians and distributors recognize the objective. A more careful look at the problem faced by the hearing aid industry would be effort well spent by interested physicians. We may be assured that the hearing aid industry has carefully looked at our problems.

In addition to recommending the use of a hearing aid, the otologist is obligated to advise his patient on which ear to use (or both when appropriate) and on which type of aid, air or bone conduction, to buy. He also should briefly initiate the training period usually required for the patient to obtain maximum benefit. Beyond this, the knowledge and technical skill of the distributor must be relied upon.

By mutually approaching the rehabilitation of deafened people, we in no wise compromise our position as physicians. The careful fitting and care of hearing aids is one aspect of the overall rehabilitation problem which the physician has neither the training nor the inclination to carry out. We should be on the best of terms with those who do this aspect of the job.

—LOUIS E. ADIN, JR., M.D., Dallas,
President, Texas Otolaryngological Association.

personal opinions and views

Survival of the "Unfittest"

IN 1859, CHARLES DARWIN explained how, as the result of the struggle for existence, Nature selects the fittest for survival. Left undisturbed by man, Nature prevents overpopulation by weeding out that life which is least capable of competing for survival. Through his tampering with Nature's balance, however, mankind has created the explosive problem of survival of the "unfittest." Civilized man is living beyond his usefulness, and afflicted people who are kept alive reproduce inferior offspring.

Recently, Dr. John D. Thompson, chairman of the Department of Obstetrics and Gynecology at Emory University School of Medicine, told delegates to the White House Conference on Mental Retardation that the quality of human reproduction is "poor and getting poorer." With the existing poor quality of human reproduction and the inadequate resources available to cope with the problem, he predicted that "the situation will deteriorate rapidly in the next decade to utter chaos."

The common belief that undesirable hereditary traits, especially if dominant, tend to increase percentagewise as the population increases is not true according to Mendel's law of segregation and aggregation. This law assumes that large populations breed at random and is not contradictive to the fact that a race of inferior human beings is developing.

Advancement, particularly in the medical sciences, has made it possible to keep alive persons with undesirable hereditary traits who otherwise would have died in infancy and early childhood. Phenylketonuria, galactosemia, retinoblastoma, diabetes, and schizophrenia are but a few hereditary diseases which now can be controlled, making it possible for more of these afflicted persons to propagate and pass their mental and physical handicaps on to countless generations.

Humans with low intelligence often produce large numbers of offspring of similar quality. Dr. Franz J. Kallmann, chief of psychiatric research at New York State Psychiatric Institute, stated that "a striking rise in the birth rate among schizophrenic patients poses both medical and social problems for the future." Following a longitudinal study of two samples of 2,700 schizophrenic patients in 11 state hospitals for the years 1934-1936 and 1954-1956, he disclosed that the reproduction rate of schizophrenic women in the later series increased 86 percent as against 25 percent in the general population. This represented an increase of 60 children per 100 women—"practically doubling the rate of the general population." In line with this thinking, Thompson said that the "massiveness" of the antepartum clinics in charity hospitals is "unbelievable." A survey of expectant mothers at Emory University's hospital showed that 17 percent were functionally illiterate and 34 percent delivered illegitimately.

What avenues of approach are open to cope with the problem of survival of the "unfittest"? When future programs of research and patient care are established, top priority must be directed toward producing the optimum human product. Facilities for adequate maternal care, especially of the indigent expectant mother, must be provided. Persons with serious hereditary disorders should be counselled concerning the possibility of producing afflicted children, and instructions and assistance in birth control should be offered to these persons. Better nutrition and strict observance of the principles of hygiene may help to upgrade the quality of the human race. Man someday may be able to improve the species by altering the structure of genes, but much remains to be learned.

Whereas, each physician adheres to the principle that his best efforts must be expended toward keeping all of his patients—fit or unfit—alive and as healthy as possible, he must face the responsibility to help guide the mental and physical destiny of mankind.

—EVERETT R. VEIRS, M.D., Temple.

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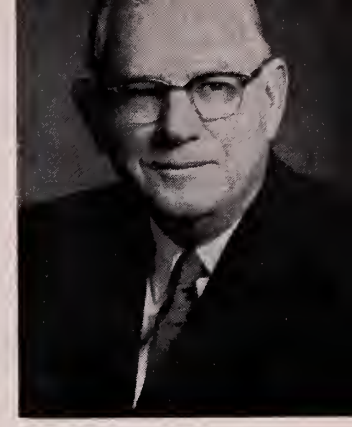

A Proposal for Orientees

Doctors as individuals are among the best educated people in America, but as a group they know less about their county, state, and national associations than others.

The Texas Medical Association, realizing this, made it mandatory that each new member attend an orientation program during his two years of probationary membership.

The program has been proved a great success. It has been of tremendous value to doctors entering practice. The men who have conducted this program are to be highly commended.

I would like to suggest one additional requirement. I believe if each doctor taking the orientation program is required to attend one Annual Session of Texas Medical Association, both the doctor and the Association would be greatly benefited.

He would be required to attend (as a guest) the opening session of the House of Delegates, then select at least one resolution submitted to the House, follow it through the reference committee, participating in the deliberations, and then follow it back to the House for final action.

He would also attend one session of the Executive Board to familiarize himself with the duties of the various councils, officers, and committees.

A member completing such a course of orientation would be aware of the mechanics of medical organization. He would become familiar with its philosophy and policies, and of the means it uses to accomplish its aims. He would be a better member of medical groups and more likely to take an active part in his society's program. He would be a well-informed, participating member who would have much to contribute to his community and to his society.

Mat E. Johnson
President
Texas Medical Association

Menopause and Postmenopause Management

DON G. HARREL, M.D.

CLINICAL MEDICINE

THE POSTMENOPAUSAL woman is currently the subject of a controversy, the center of which is her alleged need for, as well as the safety and value of, prolonged hormone therapy.

Two factors have brought this controversy to attention:

First is the increasing number of women beyond the menopause. In 1900, a woman's average life expectancy was 48.7 years.[4] Fifty years later her expectancy had risen to 72.4 years. It has been suggested that the geriatric use of hormones is an important measure to assure that this nearly one quarter of a century which has been added to a woman's life represents fuller years of living rather than merely additional years of life.

The second factor spotlighting this subject today is that several prominent teachers in both gynecology and endocrinology have recently advocated the routine prolonged use of hormones in all postmenopausal women. Estrogen has long been employed as replacement therapy in the menopause in such clinical disorders as osteoporosis and senile vaginitis. Now, however, the concept of estrogen as the "metabolic strength" of women—of its beneficial effect on practically every system, organ, and tissue of the body—has given rise to a new and challenging idea that estrogen administration should be continued beyond the limits of actual menopause for its protective influence on vital processes, notably the cardiovascular system, bone, and cellular metabolism. The advocates contend that such a program will provide a rational means of retarding, or even preventing some of the degenerative and incapacitating disorders of women in later years. It has long been recognized that the castrate as well as the premature menopause should be treated with cyclic estrogens with or without progesterone. By so doing physicians have been able to forestall premature aging, maintain relative cosmetic elegance, insure a functional coital tract, prevent osteoporosis, and protect against atherosclerosis.

Dr. Robert A. Wilson says, "A girl becomes a woman when estrogen and progesterone arrive. When they depart, she is a woman by courtesy only—in reality a castrate."[6] He considers it medical barbarism to deny full replacement therapy to women in their 20's and 30's who have of necessity had their ovaries removed. He thinks this is also true when the denial is later on, but the insult (he feels) decreases with age. Dr. Allen Barnes, chairman of the Department of Obstetrics and Gynecology at Johns Hopkins Medical School, feels that there are many factors which point to the fact that the menopause is a pathologic rather than a physiologic epoch in a woman's life.[1] He points out that the human ovary is an example of a vital organ dying well in advance of its owner and that *Homo sapiens* is the only species that undergoes this phenomenon. In lower animals, ovarian life expectancy and general life expectancy coincide. He feels that the human is no better equipped than any other species to live one-third of a lifetime without the important ovarian hormones.

These men, and others, point out that the reasons given for full replacement therapy to the castrate and to the premature menopause patient are valid reasons also for giving hormones to the woman with naturally occurring menopause and contend that these women, for all intents and purposes, are castrates. Griffith[3] cites evidence to show much more widespread occurrence and rapid progression of atherosclerosis in women de-

Arguments are presented both for and against the universal routine use of hormones.

prived of estrogen at an early age and feels that this progression can be arrested by full doses of estrogen. Similar results in the male who has had a first coronary occlusion and who has been treated with estrogen have been reported. These reports suggest that estrogen may be the protective mechanism against atherosclerotic disease in the premenopausal woman and that the continuance of exogenous estrogen after the menopause might offer similar protection.

The average gynecologist, general practitioner, or internist treating the menopausal and postmenopausal patient has been taken somewhat by surprise by this seeming change in attitude and teaching of some of the leaders in endocrinology and gynecology. The average practitioner probably has the attitude that 85 percent of menopausal patients have symptoms, for the most part symptoms are mild and short lived, 50 percent need hormones for a time, treatment can be discontinued when symptoms disappear, and "new hormone balance" is achieved.

Contrast this with those who say that the routine giving of estrogen with or without progesterone or androgen will do the following: prevent premature aging, act as a prophylaxis against mental aberrations in postmenopausal women, reverse negative nitrogen balance, prevent osteoporosis, and prevent or slow atherosclerotic processes.

If these are true, then what reasons are advanced by those who argue against routine use of hormones? There are four—possible production of cancer, withdrawal bleeding, prolonged change, and "interference with nature."

Carcinogenesis

For years the lay press has presented frightening generalities as to the possible relationship between hormones and the production of cancer. This idea has arisen largely from two sources. First, animal research has shown that in strains of animals highly susceptible to the spontaneous occurrence of breast, uterine, and skin malignancy the percentage occurrence can be increased by the use of estrogens, but in dosages far exceeding, on a mg. per kg. ratio, any ever given to humans. Secondly, the idea has arisen from the general lay knowledge that certain tumors are hormone-dependent and that the deprivation of hormones results in palliation of the tumor. Among best examples of this hormone dependency is the improvement seen following the removal of ovaries in cancer of the breast, and of the removal of the testicles in cancer of the prostate.

Greenblatt[2] says that we should have little fear in regard to the production of cancer in humans by estrogen. He calls attention to the fact that since 1930 when significantly potent hormones first became available for clinical use there has been no significant increase in breast cancer, and there has been an actual decrease in uterine cancer—this in spite of relatively widespread use of estrogens in millions of women during the three ensuing decades.

Withdrawal Bleeding

All of us have been taught since medical school days that postmenopausal bleeding is an extremely significant symptom and sign. Spontaneously occurring, it means cancer, either of the cervix or endometrium in 30 to 40 percent of the cases in which it occurs. The giving of estrogen to a woman past the menopause frequently results in uterine bleeding

since the responsiveness of the uterus as an end-organ does not parallel the disappearance of function of the ovaries. There is also much variability in this sensitivity so that one woman can take large doses of estrogen without bleeding, while others can be stimulated to bleed with extremely small doses of estrogens.

This frequent occurrence of bleeding gives rise to the question, "How is one to determine which postmenopausal bleeding is significant?" Many patients also have been conditioned to the idea that bleeding is a "danger signal," and these patients cannot readily be reassured.

If one takes the attitude that any such bleeding which occurs in a woman taking hormones is due to the exogenous hormones, he must in time give himself false assurance and miss a spontaneously occurring malignancy. If, on the other hand, he takes the attitude that all such bleeding must be investigated by curettage, then he will certainly subject many women to an unnecessary trip to the operating room.

The advocates of the routine use of hormones answer this in two ways:

1. They advise the use of androgen or progesterone with estrogen. These hormones tend to decrease the tendency for estrogen-induced bleeding, or

2. They tend to minimize the importance of this symptom and suggest[6] that periodic "lavage" of the endometrium by bleeding prevents hyperplasia and may even cure early in situ endometrial carcinoma.

Withdrawal bleeding six or seven times a year has been advocated and has been termed "bleeding by plan" and considered to be an acceptable consequence of hormone therapy even in women in the seventh or eighth decade of life.

Prolonged Change

It has been calculated that the average premenopausal woman at her production peak each month secretes 200 μg. of estrogen per 24 hours as compared to 40 μg. per 24 hours in the postmenopausal woman. This abrupt change with the accompanying pituitary stimulation has been named as the causative factor in the hot flashes complained of by menopausal and postmenopausal patients. The introduction of exogenous estrogens not only relieves the hot flashes but prevents an abrupt change in the hormone environment of the tissues and does, in effect, prolong the change since so long as the exogenous estrogen is supplied the hormonal deprivation of the tissues is prevented. The objective of the advocates of routine use of hormones is not to prolong the change, but to prevent the change.

"Interferes with Nature"

Barnes[1] says the principal argument that the climacteric is physiologic is that it happens to all women. He counters this argument by saying that death too is universal, but this does not deter us from taking every step possible to prevent its coming. He points out also that when he called his ophthalmologist to complain that the telephone directories were being printed in smaller and smaller type he was not given the answer, "Middle age presbyopia is universal, so we will not treat it." Instead, he prescribed the necessary optical correction and he was able to compensate for the aging process. Since the ovary appears to be the "Achilles' heel" of the human female[5] it is suggested that this "interference with nature" may be such a compensation.

Summary and Conclusions

Recognition has been given to the increasing number of postmenopausal women, and arguments have been presented both for and against the universal routine use of hormones in postmenopausal women. To attempt to present conclusions at this time would be presumptuous; however, several observations may be timely and worthy of thought.

(1) The average practitioner has been too frugal in his use of hormones. (2) Most, if not all, women need hormones at menopause for a long or a short period of time. (3) Postmenopausal bleeding, its interpretation and management, presents the greatest obstacle to routine use of hormones. (4) Further study is needed in the adjustment of dosage and combination of hormones less likely to cause uterine bleeding. (5) There is a need for large groups to be studied so that more accurate comparisons may be made.

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Dr. Don G. Harrel is a Dallas obstetrician and gynecologist. He presented this paper to the Section on General Practice at the 1964 TMA Annual Session.

Dr. Stare Authors Book

Dr. Fredrick J. Stare of Harvard, author of the editorial "Nutritional Nonsense" on p. 705 and a 1964 TMA Annual Session speaker, is the author of a book entitled "Eating for Good Health" which came from the presses of Doubleday in August.

He also writes a twice-weekly syndicated column which appears in more than 40 papers in the U. S., Canada, and abroad and is called "Food and Your Health."

All proceeds received by Dr. Stare from these publications go to a fund that is used to help support researches of Harvard's Department of Nutrition.

Dr. Fredrick J. Stare was born in 1910 in Columbus, Wis. He studied biochemistry at the University of Wisconsin and was awarded his first doctorate (in science) in 1934. Later he studied in Cambridge, England, and in Hungary with Nobel Laureate, Albert Szent-Gyorgyi. He worked with another Nobel Prize winner, Paul Karrer, in Zurich and then returned to pursue research in enzymes in cancer tissue at Wisconsin. In 1941 he received his medical degree at the University of Chicago. He has his internship at Barnes Hospital in St. Louis and passed his Missouri boards.

In 1942, Harvard invited Dr. Stare to organize the first Department of Nutrition at any great health and medical center. It has been one of the fastest growing units on the campus and now has an annual budget nearing $1,000,000 and a staff of more than 100. Through this department, Harvard has done much with the study of cholesterol in coronary heart diseases and strokes and in the field of obesity.

Dr. Stare is consultant on nutrition to the U. S. Public Health Service, the Veterans Administration, and the Inter-Departmental Committee on Nutrition for National Defense. The World Health Organization has called on him to help train health workers in Indonesia and to report on health and nutrition in Africa, South and Central America.

Vaginal Cytology as an Aid In Predicting Normal Pregnancy

Garland H. Lang, M.D.; Walter S. Parks, Jr., M.D.; C. Roy Johnson, M.D.;
Charles P. Guerriero, III, M.D.; Martha Madsen, M.D.; and Yolan Zimmerman, M.T., C.T.

RESEARCH & INVESTIGATION

ALTHOUGH the vaginal cytology of pregnancy was first described by Papanicolaou[3] in 1925, the more important cytologic detection of cancer overshadowed these findings for many years. Only after 1949 when numerical criteria were introduced for the evaluation of cytology during pregnancy was this method considered practical. Such investigators as Pundel of Luxembourg[4] and Gaudefroy of France[1] have reported large series of cases using the vaginal smear as an indication of the normal hormone balance of pregnancy. Nesbitt[2] of New York reported a series of cases from which he concluded that cytodiagnosis of hormonal status using a vaginal smear was of great significance in forecasting pregnancy outcome.

In the first 12 weeks of pregnancy, the vaginal cytology reveals the effect of an adequate progesterone level by the presence of more intermediate cell types. The number of mature cells with small dense nuclei is reduced to a percentage less than 20. There is also a decreased number of eosinophilic cells, and after the placental hormones begin to increase, many of the proliferating intermediate cells take on a characteristic form and are termed "navicular" cells. These cells usually predominate in the vaginal smear during the last two trimesters of pregnancy.

Method of Study

Smears from the lateral vaginal wall are taken at the time of the first pelvic examination of obstetric patients. These smears are fixed and stained according to the Papanicolaou technique and read by the same technician. The slides are studied under low and high power and classification made on the basis of the karyopyknotic index. One hundred cells are counted and the total number of pyknotic cells, both eosinophilic and basophilic, is expressed as a percentage, or the karyopyknotic index (KI). Smears are reported as abnormal, borderline, or normal for pregnancy. A karyopyknotic index of 20 or below is considered normal, a value of 20 to 30 is borderline, and a value over 30 is reported as abnormal.

Material and Results

Over the last two and one-half years, 772 private obstetric patients have had a hormone evaluation done by the use of the vaginal smear technique described. The following statistics are accumulated and evaluated in Table 1. Seven hundred three patients had a normal KI with 660 patients going to term and 35 aborting, a 4.9 percent abortion rate. In the borderline group there were 30 patients, 28 of these patients going to term and 2 patients aborting, a 6.6 abortion rate. There were 39 patients with an abnormal smear and 27 of these patients went to term with 11 aborting, an abortion rate of 28.2 percent. The abortion rate in the total of 772 patients was approximately 6.2 percent.

In Table 2 these patients are divided according to the year in which the study was done, beginning in 1961 and continuing through most of 1963. As our experience increased in interpreting the slides and also in repeating smears on patients showing an abnormal KI, it was found that our percentage of abortion in the normal patients dropped from 6.2 to 3.4; in the borderline group the percentage increased from 0 to 16.6 percent; and in the definitely abnormal

In a study of 772 patients from whom vaginal smears were taken, those with a normal karyopyknotic index were found to have four times less chance of aborting than those with an abnormal index. In this series, hormone therapy did not increase the fetal salvage rate.

TABLE 1.—*Comparison of Pregnancy Outcome in 772 Gravid Women in Relation to Karyopyknotic Index of Vaginal Smear.*

Karyopyknotic Index	Total Patients	Pregnancy Outcome				
		Term	Premature	Immature	Abortion No.	Abortion %
Less than 20	703	660	3	5	35	4.9
20 to 30	30	28	0	0	2	6.6
Over 30	39	27	0	1	11	28.2
Total	772	715	3	6	48	6.2

TABLE 2.—*Comparison of Pregnancy Outcome in 772 Gravid Women in Relation to Karyopyknotic Index of Vaginal Smear According to Year of Series.*

Year	Karyopyknotic Index	Total Patients	Pregnancy Outcome				
			Term	Premature	Immature	Abortion No.	Abortion %
1961	Less than 20	258	242	0	0	16	6.2
1962	Less than 20	270	251	1	5	13	4.8
1963	Less than 20	175	167	2	0	6	3.4
1961	20 to 30	5	5	0	0	0	0
1962	20 to 30	13	13	0	0	0	0
1963	20 to 30	12	10	0	0	2	16.6
1961	Over 30	15	12	0	0	3	20
1962	Over 30	18	11	0	1	6	33.3
1963	Over 30	6	4	0	0	2	33.3

group the abortion rate increased from 20 to 33⅓ percent.

In reviewing our findings and evaluating them as to their significance in predicting pregnancy outcome, we compared these with the series reported by Nesbitt and others in 1961.[2] Table 3 gives a comparison of his series and of the present series. In the series reported by Nesbitt, patients with a normal KI had an abortion percentage of less than 1 percent whereas in the present series the rate was almost 5 percent. In the borderline group of patients his series showed an abortion rate of 4.4 percent and in the present series, 6.6 percent. In the group with abnormal KI, Nesbitt showed an abortion percentage of 46.7 whereas in the present series this abortion percentage was 28.2 percent. In the final total the abortion rate for the present group was 6.2 percent and for the series reported by Nesbitt the rate was 11.1 percent. The percentages reported from this series are very much out of line with those reported by Nesbitt[2] and also with those reported by Pundel[4] and Gaudefroy.[1]

In attempting to explain the discrepancy in abortion rates in the two series compared, one factor could be the difference in experience of the technicians reading the slides, and the staining methods used. One purpose of this study was to determine the accuracy and feasibility of using a slide stained by the Papanicolaou technique, and read by a cytotechnologist using only the karyopyknotic index as an indicator of normal pregnancy hormone balance. Obviously we have not approached the accuracy reported by others.[2, 4] Another possible reason for the discrepancy

TABLE 3.—*Comparison of Results of Present Study with Series of Cases Reported by Nesbitt and others.*[2]

Series	Karyopyknotic Index	Total Patients	Pregnancy Outcome				
			Term	Premature	Immature	Abortion No.	Abortion %
A	Less than 20	703	660	3	5	35	4.9
B	Less than 20	304	290	13	0	1	0.3
A	20 to 30	30	28	0	0	2	6.6
B	20 to 30	91	81	5	1	4	4.4
A	Over 30	39	27	0	1	11	28.2
B	Over 30	109	46	9	3	51	46.7
Total A		772	715	3	6	48	6.2
Total B		504	417	27	4	56	11.1

A = series reported by Lang and others.
B = series reported by Nesbitt and others.[2]

in results is that in the first year of work in this series there were no follow-up smears on patients with an abnormal karyopyknotic index so that a certain number of these patients may actually have reverted to normal. Later in our series an effort was made to follow all patients showing an abnormal KI at regular intervals until the patient passed the 16th week of gestation, or the index reverted to normal, either spontaneously or on hormone therapy. It was noted that approximately 50 percent of abnormal smears revert to normal on repeat examination.

Effect of Hormones On Predicted Pregnancy Outcome

Beginning in 1962 more attention was paid to the presence of abnormal smears and more patients were treated with a progestational agent when they showed an abnormal vaginal smear. In the 1962 series there were 31 abnormal smears and 15 patients were treated with a progestational agent. Twelve of these patients delivered full-term normal infants. There were 3 abortions, 2 of which were classified as missed abortions. In the 1963 series there were 18 abnormal smears and 4 of these patients were treated with a progestational agent. Of the 4 treated, 2 delivered at term, 1 delivering a mongoloid infant, and 2 patients aborted with 1 classified as a missed abortion. This gave us a total of 49 women with abnormal smears of which 19 were treated with a progestational agent. Fourteen of the treated patients delivered at term with 13 normal infants and 5 aborted for an abortion percentage of 26.3 percent. Of the 30 patients not treated, 5 aborted giving an abortion percentage of 16.6. Statistically it would be better to withhold hormone therapy in these cases.

Dr. Garland H. Lang practices obstetrics and gynecology in Midland as do his co-authors, Drs. Parks, Johnson, and Guerriero. Co-author Dr. Madsen practices pathology there and Mrs. Zimmerman is a medical technologist there.*

We feel that hormones should be administered to women with abnormal smears having a history of previous abortions, and sterility patients pregnant for the first time. Close observation of uterine growth to detect possible missed abortion is important when patients are receiving hormone therapy.

Based on our experience we offer the following observations:

1. Gravid women whose vaginal smear reveals a normal karyopyknotic index have a 4.9 percent chance of aborting.
2. Those having an abnormal karyopyknotic index, that is, over 30, have a 28 percent chance of aborting.
3. The abortion rate is four times as great in the abnormal group as it is in the normal group.
4. Statistically, in this series hormone therapy in a pregnancy showing an abnormal cytologic smear did not increase the salvage rate.

Summary

1. A short resume of vaginal cytology during pregnancy and interpretation of cytologic smears by one method has been given.

*Dr. Lang presented this paper to the TMA Section on Obstetrics and Gynecology on April 25 during the 1964 Annual Session in Houston. At the same time, Dr. Raymond Kaufman presented the discussion which follows.

2. The results of cytologic smears taken during the first 16 weeks of gestation in 772 private obstetric patients has been reported; 4.9 percent of the pregnancies with a normal smear ended in abortion; 6.6 percent of pregnancies with a borderline smear ended in abortion; 28.2 percent of pregnancies with an abnormal smear ended in abortion. Smears with a karyopyknotic index of less than 20 percent were considered normal.

3. In this series, hormone therapy in gravid patients showing an abnormal vaginal cytology did not increase the fetal salvage rate.

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DISCUSSION

RAYMOND H. KAUFMAN: The use of vaginal cytology in predicting pregnancy outcome has been utilized for many years outside of the United States. Only in recent years has this prognostic tool been put to use in this country. There is no doubt about the fact, and Dr. Lang's figures confirm this, that an abnormal smear carries with it a much more ominous significance than a normal smear. The percentage of patients with normal smears who aborted in Dr. Lang's series is higher than reported by most other authors. Gaudefroy[1] and Nesbitt[3] both reported an abortion rate of less than 1 percent in the presence of a normal smear obtained during the first trimester of pregnancy. The reasons for this difference may well be as Dr. Lang suggested, a lack of experience in reading the slides, which will be rectified as time progresses. One other possible factor may be that he is only considering the karyopyknotic index (KI) in evaluating the smears. Hormone production by the ovary and the placenta is reflected by the KI, but other factors in the smear will also be influenced, such as the tightness or looseness of the cell clusters, the percentage of isolated cells, the thickness of cell cytoplasm, cytoplasmic glycogen, and cytoplasmic curling. It has been our policy to take all of these factors into consideration and as a consequence, we will occasionally report a smear as abnormal even with a KI of less than 20 percent. A perfect example of this is a patient who had a KI of below 10 percent on repeated smears. Just prior to abortion, a loosening of the cell clusters and thinning of the cell cytoplasm was noted. This was considered an abnormal smear, even though the KI was within normal limits.

We concur with Dr. Lang's findings that *repeated* smears during pregnancy are necessary if an adequate prognostic evaluation is to be made. This is especially true in the presence of an initial abnormal smear. The cytologic findings may vary during the first trimester of pregnancy, and on occasion, the KI may rise above 20 percent only to fall rapidly back to within normal limits. Pundel[4] has noted a transitory rise in the KI at the end of the third month of pregnancy in some cases. A single abnormal smear that rapidly returns to normal is probably of no great significance, however, the persistence of an abnormal smear does signify the possibility of spontaneous abortion occurring in a significant number of cases.

Stamm and others[4] have indicated that an intense estrogen stimulation in the presence of progesterone is probably an essential requirement for a smear showing a normal pregnancy cytology. Stamm feels that estrogen insufficiency is probably the principal cause of an elevated KI during pregnancy. Their findings indicated that the majority of cases with pathologic smears during pregnancy had an estrogen insufficiency *without* luteal insufficiency, whereas cases of threatened abortion with normal cytology usually had a normal level of estriol. Studies by Greene[2] have indicated that placental failure late in pregnancy is associated with a great decrease in urinary estriol excretion. This is also probably true during the early months of pregnancy.[5] The decrease in urinary estriol excretion reflects a decreased hormone production by the placenta and/or the ovary, and this in turn is reflected by the abnormal changes noted in the vaginal cytogram. This abnormality in hormone excretion may be merely transitory as indicated by the fact that over two-thirds of the patients with abnormal smears in Dr. Lang's series did not abort, however, in a significant number of cases the abnormality is a permanent one, leading to subsequent abortion.

The amount of time and energy expended in evaluating smears for pregnancy prognosis is considerable, and a natural question to raise is whether the information derived is worth this effort. If one could obtain the results of Gaudefroy and Nesbitt where they have noted an abortion rate of less than 1 percent in the presence of a normal smear, it would be of some psychological benefit to the patient to be able to give her the assurance that she has over a 99 percent chance of having a normal pregnancy. This alone, however, does not justify the effort. The women on whom abnormal smears were found had an abortion rate which was higher than that noted in the patients with normal smears. From Dr. Lang's figures, it appears that the administration of progesterone at random to a group of these women had no effect on their pregnancy outcome. In my opinion, the outstanding potential of the vaginal cytogram during pregnancy would be to enable us to select those patients with abnormal smears who might be successfully supported with endocrine therapy. If this could be done, the fetal salvage obtained would make this effort worthwhile. At the present time, such a study is being conducted at Baylor University College of Medicine by Dr. Bao-jen Jing and myself. It has been amply demonstrated by Wied[6] and Gaudefroy that in the presence of a normal pregnancy, the vaginal mucosa is not sensitive to the effects of estrogen, that is, there

is no increase in the KI following the administration of estrogens. In the presence of a dead fetus or inadequately functioning placenta, however, the administration of estrogens, intramuscularly or orally, results in an increase of the KI. Wied has noted that the prognostic value of the cytologic evaluation during pregnancy becomes excellent if one administers estrogens as a test for the determination of the presence or absence of a proliferative response (increase in KI). He has indicated that in cases where an abnormal smear is converted to a normal smear following the administration of estrogens, hormonal therapy may be helpful. Also, some cases with an initial normal smear may show an abnormal response to estrogens, thus allowing detection of an otherwise unrecognized abnormal pregnancy. We have been obtaining vaginal cytograms from patients after the diagnosis of pregnancy has been confirmed, following which the women have been given Premarin orally for several days. A repeat smear is then obtained and the patients are classified into four groups:

1. Those from whom normal smear was taken initially and following estrogen. Prognosis is good.
2. Those from whom normal smear was taken initially; abnormal smear after estrogen. Prognosis is poor.
3. Those from whom abnormal smear was taken initially; improved after estrogen. Prognosis is guarded. Progesterone therapy may be indicated.
4. Those from whom abnormal smear was taken initially, becoming more abnormal after estrogens. Prognosis is poor.

At the present time our series is too small for statistical evaluation, however, our findings have been promising. Our ultimate aim is to be able to select those patients who will be benefited by endocrine therapy in support of their pregnancies. This is especially true for the group of patients who are habitual aborters.

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If They'd Played it Cool, Where Would We Be?

It is generally agreed that what our country needs today is patriotism, nationalism, morality, courage, dedication, and religion, and in a way that these have never been needed before.

These virtues are not the attributes of the modern "angle players," declared James E. Palmer, Jr., a professional staff member of the U. S. Senate Committee on Banking and Currency, in a commencement address.

Had they been angle-players, he said, some of our national heroes might have answered the call to duty like this:

Paul Revere: Why pick on me? It is the middle of the night. I cannot ride through every Middlesex Village. Besides, I am not the only man in Boston with a horse.

Patrick Henry: Yes, I am for liberty but we must be realistic. We are small compared to the British and someone is going to get hurt.

George Washington: Gentlemen, you honor me. I am just getting some personal matters settled and have much to do at Mount Vernon. Why don't you try General Gates? Also, you might say I have served my time against the French. . . .

Benjamin Franklin: I'm over 75 years old. What you need as a Minister to France in these strenuous times is a younger man. Let a new generation take over. I want to rest.

Management of Obstetric Emergencies In a Small Hospital

J. COLLIER RUCKER, M.D.

REVIEW ARTICLES

FOR A FRAMEWORK of reference, a small hospital may be designated as one of 150 beds or less, with an obstetric division of 15 beds or less.

Obstetric emergencies are not numerous, and most can be prevented by adequate preparation and constant anticipation of their possibilities. They fall into seven major categories: (1) abdominal bleeding (intrapartum and postpartum), (2) eclamptogenic toxemias, (3) anesthetic emergencies, (4) drug sensitivities and overdosage, (5) emergencies incident to delivery, (6) acute abdominal emergencies complicating pregnancy, and (7) vascular accidents.

In considering proper hospital facilities for the management of obstetric emergencies, only basic facilities are needed such as: (1) reasonably qualified laboratory personnel and facilities for performing standard laboratory procedures such as blood counts, blood typing, cross matching, and routine electrolyte studies, (2) a blood bank, or convenient connections with regional blood bank facilities, (3) simple safety devices in the delivery room, and (4) nursing personnel who are reasonably well-oriented in obstetrics. The most important factor, however, is that the attending physician be alert to the possibility of emergencies. There is no field of medicine in which a high degree of suspicion is more important, no field in which normal physiology can change to life-endangering complications in a briefer period of time, and no field where prophylaxis pays a higher dividend.

Certain simple basic precautions and procedures will anticipate and prevent most obstetrical emergencies, and will successfully manage those that do occur. These precautions and procedures can be carried out in any hospital, regardless of its size.

Obstetric bleeding (abortion, ectopic pregnancy, placenta praevia, abruptio placentae, rupture of the uterus, and postpartum hemorrhage) is the most frequent emergency encountered. This still ranks first of the three causes of maternal death in Texas (32 percent). Of these, postpartum hemorrhage accounts for approximately 25 percent.

Postpartum Hemorrhage

The causes of postpartum hemorrhage are uterine inertia, retained secunda, laceration of the birth canal and uterus, or rarely, defects in clotting mechanism.

The best management of postpartum hemorrhage is prophylaxis consisting of: (1) manual removal of the placenta when it is not easily expelled, (2) thorough digital exploration of the uterine cavity, (3) careful attention to the tone of the uterus after delivery of the placenta, especially in the over-distended and over-used uterus (notation of how well the uterine cavity is obliterated, and how well the cervix is retracted), (4) thorough massage of the uterus and maintenance out of the pelvis until uterine contraction is well established, (5) routine inspection of the cervix and vaginal wall for

The basic facilities in every good hospital, small or large, are adequate for obstetric emergencies. Far more important to the patient are a physician who is alert to the possibility of emergencies and able to manage them wisely, and reasonably well-trained nursing and laboratory personnel.

lacerations, (6) initiation of the episiotomy repair approximately 1 cm. above the incision or laceration, (7) observation of the patient until lochial discharge is minimum (no less than one hour postpartum). Proper prophylaxis after the patient returns to her room includes careful observation through the frequent noting and recording of blood pressure and pulse, and careful notation and recording of the number and character of pads (soiled or soaked). Attentive observation should continue for a minimum of three hours, or longer if the lochial discharge is at all excessive. One cardinal point in the prevention of postpartum hemorrhage is to remember that a patient can exsanguinate and go into shock from multiple small bleeding episodes as well as from a massive hemorrhage. It is always important to be particularly watchful of the grand multipara.

Treatment of postpartum hemorrhage is well established and requires no special equipment. Management of uterine inertia consists of adequate administration of oxytocics (Ergotrate derivatives, either intravenous or intramuscular, oxytocin intramuscularly or by drip) and prompt and early intravenous administration of lactated Ringer's solution and blood when blood loss appears to be more than 500 cc. It is rare that surgical removal of the uterus is necessary.

Retained secunda, except in the extremely rare case of placenta accreta, can be easily removed by finger curettage, particularly when gauze is utilized.

Lacerations of the uterus can be detected by manual palpation, and surgical removal of the uterus is usually necessary. Lacerations of the cervix can be seen with adequate light and exposure. Hemostasis either in the cervix or along the vaginal tract can be stopped by continuous suturing.

It is extremely important that lactated Ringer's solution be started at the first indication of excessive bleeding. A large needle or catheter is best to assure that a vein may be kept open and to maintain adequate volume and electrolytes until blood is obtained.

The secret of adequate management of postpartum hemorrhage is fourfold: (1) always expect it, (2) recognize it when it occurs, (3) stop it, and (4) replace blood and fluid loss.

Postpartum hemorrhage usually can be prevented and always can be controlled with no more facilities or personnel than an alert obstetrician, a reasonably well-informed circulating nurse, adequate delivery room lighting, instruments for exposure, and available electrolyte solutions and blood.

Third Trimester Bleeding

Significant third trimester bleeding usually is caused by placenta praevia or abruptio placentae.

Briefly, the management of placenta praevia consists of correction of shock and proper diagnosis. In the interest of the baby, it is important to recognize that the first bleeding episode usually is not serious, but there must be close observation for the second episode, with delivery then performed by the simplest means and attention given to adequate blood replacement (1000 ml. of blood is the minimum that should be available). In placenta praevia, the later in pregnancy the bleeding occurs, the less amount of cervix is usually compromised.

The management of abruptio placentae also depends on proper diagnosis, prompt delivery, and correction of shock. The most serious problems in abruptio placentae are the occurrence of fibrination and depletion of the factors significant to hemostasis.

Since fibrination may occur intermittently and recurrently before placental separation becomes complete, prompt emptying of the uterus is of prime importance; this is to prevent further fibrination, with the resultant pulmonary embarrassment, panhemostatic deficiency, and shock out of proportion to the hemorrhage.

Proper management of shock is first dependent on accurate evaluation of the panhemostatic deficiencies. Defibrination is a frequent one. This can easily be determined by one of several rapid tests. In the clot observation test, 5 ml. of maternal blood in a 15 ml. test tube is gently agitated four or five times. If a clot occurs within six minutes, it can be assumed that the fibrinogen level is greater than 150 mg. per 100 ml. If clotting time is more than six minutes with a poor clot, the fibrinogen level probably will be between 100 and 150 mg. per 100 ml. No clot in 30 minutes indicates a fibrinogen level of less than 100 mg. per 100 ml. For the topical thrombin test, 1 ml. of fresh blood is added to a tube containing .1 ml. of topical thrombin. A clot will appear immediately if

Dr. Rucker, who practices in Jacksonville, prepared this article in cooperation with the TMA Committee on Maternal Mortality, of which he is a member, to further eliminate maternal deaths in the state.

the fibrinogen level is adequate; with decreased fibrinogen, there will be either no clot or a very poor clot. Confirming laboratory tests can be done in any laboratory. Other tests for hemostatic deficiencies are: (1) determination of circulating fibrinolysin (5 ml. of unclotted maternal blood is added to 5 ml. of clotted normal control blood and held at room temperature; lysis of normal clot within one hour indicates the presence of fibrinolysin), and (2) determination of heparin-like factor (5 ml. of unclotted maternal blood is added to 5 ml. of normal control blood; failure of the combined specimen to clot indicates the presence of a heparin-like factor).

Besides prompt delivery, the most important treatment of abruptio placentae is the administration of whole blood, because of the associated hemorrhage and the generalized disturbance in the hemostatic mechanism. Excessive transfusions, particularly prior to emptying of the uterus, should be avoided because of the possibility of aggravating an acute cor pulmonale caused by the fibrination. If the uterus has been emptied and the fibrinogen level, according to tests, is below 100 mg. per 100 ml., a minimum of 4 gm. of fibrinogen should be furnished. The expense and rarity of need for fibrinogen should not deter any hospital, regardless of its size, from making provisions for a minimum of 4 to 6 gm. to be available. This usually can be accomplished by the pooling of available sources in hospitals within a 50 to 100 mile radius. With 2 gm. in each hospital, and with good road connections, adequate supplies can be obtainable within a short time.

Presence of a heparin-like factor can be corrected by cautious administration of protamine sulfate, 20 to 50 mg. intravenously. This always should be given slowly, and should not be repeated unless persistence of the heparin-like factor can be subsequently demonstrated.

A significant concentration of fibrinolysin usually can be corrected with an initial intravenous dose of 200 mg. of hydrocortisone, followed by 100 mg. every four hours during the first 24 hours.

In summary, correct management of abruptio placentae is by delivery in the simplest manner possible, accurate evaluation of the hemostatic defects, adequate replacement of blood, and replacement of specific hemostatic defects when they become evident.

First Trimester Bleeding

In the first trimester, obstetric bleeding usually is caused by abortion or ectopic pregnancy. Spontaneous abortion rarely causes fatal bleeding, therefore observation and adequate oxytocic agents (when indications are that the abortion is incomplete), plus the administration of blood and electrolytes are usually sufficient. Dilation and curettage ordinarily will correct the problem when oxytocics fail.

However, it should always be kept in mind that cases of infected abortion can result in fatal bacterial shock. Important in management of these cases are alert suspicion, intensive broad spectrum antibiotic therapy, and administration of vasospastics (such as Aramine) until the blood pressure is stabilized. Cortisone sometimes is helpful.

Problems in the management of ectopic pregnancy revolve essentially around the diagnosis of its tubal abortion or rupture, adequate blood replacement, and at the same time, the performance of a laparotomy to stop the bleeding at its source.

Intrapartum obstetrical bleeding can be managed adequately by the alert physician who is able to accurately evaluate bleeding episodes and has available sufficient electrolyte solutions, blood facilities, and average laboratory, delivery room, and operating facilities.

Eclampsia

Eclampsia (convulsive toxemia) usually can be prevented by: (1) simple prenatal observation of weight changes, urinalysis, and blood pressure determinations, particularly the diastolic levels, and (2) adequate sodium restriction and propitious administration of diuretics which will extract sodium at the kidney level.

Should toxemia occur, the treatment is based on: (1) adequate sedation (20 cc. of

50 percent solution of magnesium sulfate intramuscularly, followed by 10 cc. of the same solution every four to eight hours), (2) reduction of external stimuli, (3) prevention of injury (the eclampsia patient should never be left alone), (4) close observation of intake and output, (5) reduction of vasospasm and, secondarily, blood pressure, (6) elimination of sodium retention, (7) oxygen administration through the easiest source available, (8) digitalization if pulmonary edema is present, and (9) an available tracheotomy set.

None of these procedures requires anything more than an alert obstetrician, attentive nurses, and reasonably adequate laboratory facilities.

Anesthetic Emergencies

Here, as in other obstetric emergencies, prevention is the best treatment. The most frequent anesthetic emergency is aspiration of stomach contents or respiratory tract secretions. This can be prevented in most instances by two simple procedures and one item of apparatus. Routinely, the physician should determine what and when the patient has eaten prior to admission to the hospital. If the patient has eaten within the last six hours, or if she has not completely emptied her stomach prior to admission to the delivery room, an agent other than general anesthesia should be used. Respiratory secretion drying agents (atropine or hyoscine) always should be given before admission to the delivery room. Every delivery room should have an adequate suction and endotracheal apparatus easily available and in good working order. In our opinion, water suction is the best (this can easily be built into the wall, and is always operable).

If a saddle or caudal block procedure is used, intravenous fluid replacement should be active to maintain vein patency. Blood pressure observations should be every 15 minutes, and drugs for the correction of sudden hypotension should be available.

Other anesthetic emergencies usually are associated with the use of anesthetic agents by unqualified personnel.

The size of the hospital has little or no influence on the prophylactic management of anesthetic emergencies. All that is required is an alert obstetrician who uses agents with which he is completely familiar, simple gustatory information from patients on admission to the hospital, adequate suction and endotracheal facilities in the delivery room, and drugs for the correction of hypotension.

A word should be said about the use of phenothiazine derivatives, namely that their combination with general or major conduction anesthetic agents frequently causes severe hypotension, which is difficult to treat.

Emergencies Incident to Delivery

Emergencies incident to delivery management (especially problems in cephalopelvic disproportion and abnormal positions and presentations) never have any relation to the size of the hospital per se. These problems usually arise as a result of inadequate observation by the obstetrician during the time the patient is in labor, that is, failure to have adequate evaluation of the patient's pelvis, and improper diagnosis of the degree of cervical dilatation and fetal position. Pelvic evaluation can be accomplished by a thorough clinical examination and supplemented, if necessary, by the assistance of a reasonably qualified x-ray technician and standard x-ray equipment. Any obstetrician can easily acquaint himself with the procedures of either the Snow or Ball technique of pelvimetry.

Acute Abdominal Emergencies

Hospital facilities to take care of usual abdominal emergencies are completely adequate for those emergencies when they complicate pregnancy. The woman during pregnancy is subject to all the surgical disorders that occur in the nonpregnant state. The principles of management are essentially the same for both groups. Acute abdominal conditions which require surgery should be treated without delay, regardless of the stage of gestation. Persistent abdominal pain and evidence of peritoneal irritation are the most frequent and reliable symptoms of acute disorders, and demand the ultimate in vigilance. It is of primary importance to realize that a pathologic condition exists, and that the symptoms are not caused by the pregnancy itself. When early diagnosis is combined with proper therapy, fetal salvage and suc-

cessful outcome for the mother usually are insured.

Vascular Accidents

Vascular embolisms (nonseptic thrombus embolism, air embolism, and amniotic fluid embolism) account for approximately 10 percent of the obstetric deaths. Nonseptic thrombus embolism usually can be prevented by proper treatment of varicosities and early ambulation. Should it occur, prompt anticoagulant therapy is the procedure of choice.

Preventive measures against air embolism include care in the manual delivery of placentas, and foregoing postpartum procedures which facilitate ballooning of the vaginal tract (knee-chest position, intravaginal douching, and insufflations). Successful treatment is based on rapid diagnosis; sudden cyanosis, unconsciousness, loud churning noises over the precordium, and convulsions are symptoms. The two most important therapeutic measures are to place the patient in the left lateral head down position, and aspirate air from the right ventricle. The latter is accomplished by introduction of a needle just below the xiphoid process, or in the fifth right intercostal space, and directing it cephalad so as to draw off the buoyant superficial air in the ventricle.

Amniotic fluid embolism should be suspected in the presence of tetantic uterine contractions. Treatment consists primarily of the administration of oxygen, delivery by the easiest manner possible, and replacement of blood loss and of specific blood elements determined to be deficient.

It is important to remember that hemorrhage from vascular rupture during pregnancy should be suspect in those individuals having previously demonstrated congenital aneurysms or atherosclerosis, and those who are sensitive to oxytocic agents.

One word should be said regarding the adequacy of obstetric knowledge on the part of the doctor caring for parturient patients. Perusal of the "Directory of Medical Specialists" shows that throughout Texas and the Southwest, one or more board certified obstetricians are within every 50 to 75 mile area, and in almost every situation they are as near as the telephone to provide help in obstetric emergencies. To help, however, they need adequate information before the patient's condition deteriorates beyond retrieval. An ounce of prevention is worth many pounds of cure.

Summary

The management of essentially all obstetric emergencies revolves not around the size of the hospital, but the alertness of the obstetrician to the fact that specific emergencies can arise as a result of pregnancy, extraneous causes, or injudicious use of medication. Constant suspicion of these problems should be ever present, even in cases that appear normal.

The only hospital facilities essential for the care of obstetric emergencies are a laboratory basically equipped to do present day standard procedures, adequate availability of blood facilities, nursing personnel obstetrically oriented to obtain adequate information (such as food eaten prior to admission, accurate observation of blood loss, routine observation of blood pressure and pulse and other vital signs), and simple safety facilities in the delivery room and the operating room. Evaluation of the blood centers and blood banks throughout Texas and the Southwest reveals that there is virtually no area that cannot avail itself of blood facilities. According to the American Association of Blood Banks, Texas has nine blood centers, and 312 hospitals with blood bank facilities.

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Incus Replacement Surgery To Restore Hearing

REVIEW ARTICLES

ABSENCE OR MALFUNCTION of the incus continues to plague the otolaryngologist in his daily struggle to rebuild the ossicular chain and restore hearing to the deafened patient. Prior to the general use of prosthetics in the middle ear, Wullstein of Germany believed he had the problem solved with his type 3 tympanoplasty. This gave good hearing in some cases but as a general rule a significant air-bone gap remained on the audiogram postoperatively. Guilford reported that 45 percent of his type 3 cases exhibit a 20 db. air-bone deficit. Especially in the patient with an intact healthy tympanic membrane hiding a deficient incus, the recession of the eardrum to the stapedial head often unnecessarily narrowed the already cramped middle ear space.

The feasibility of plastics and other foreign materials as aids in rebuilding the deficient chain became apparent when Shea published his initial report of the successful stapedectomy utilizing a polyethylene strut. Shea later adapted his original stapedectomy procedure to replacement of the absent incus. He used an unbeveled strut of polyethylene between the tympanic membrane and a vein graft over the oval window (Fig. 1). The chorda tympani nerve was impacted into the outer opening of the strut to stabilize it and to help prevent ear drum perforation in the postoperative period. He reported approximately a 50 percent success rate with this method.

As new techniques for primary stapedectomy were evolved, the authors adapted each one in an attempt to find a satisfactory incus replacement procedure. Kos used a wire vein plug attaching it to the malleus handle; Shea attached his polyethylene tube to this bone also. Schuknecht used the fat plug in these cases. In 1962, Sheehy reported his cases using this wire fat plug method, slightly modified from Schuknecht. This was the largest published series at that time, and showed that with careful surgical technique and proper preoperative patient selection a high degree of success could be expected from incus replacement surgery.

Repositioning of the deficient incus or the handle of the malleus has been popularized in Europe and has some advocates in this country. Guilford believes that transposition offers advantages in chronic cases. Of his cases, 89 percent showed an air-bone gap of only 20 db. Austin uses the Shea Teflon piston against the intact footplate with "good results" in chronic ear conditions. He feels the inertness of the Teflon is important to prevent scar tissue fixation of the strut and subsequent poor hearing. The Tefwire modification of this piston is also being used by Guilford for incus replacement with satisfactory results.

Surgical Technique

Since November, 1960, the author has performed incus replacement surgery on 30 patients. The majority of the cases were done using the notched polyethylene strut and vein graft. More recently the side opening Teflon piston has been substituted for the polyethylene tube with satisfactory results.

It is important to forbid the patient to wear his hearing aid in the ear to be operated on for at least two weeks preoperatively. This simple request will vastly decrease the bacterial flora of the canal or cavity, which may be an important factor contributing to postoperative cochlear loss.

Local Xylocaine anesthesia is quite satis-

Incus replacement with polyethylene strut and vein and with Teflon piston is discussed, and results of surgery in 30 cases are given.

H. E. MADDOX, III

factory with premedication of Seconal (0.1 gm.) and Demerol (100 mg.) given one to two hours before surgery. In the occasional restless patient, intramuscular Phenergan may be used during the procedure.

Following anesthesia the cavity or ear canal is meticulously cleansed of debris and irrigated thoroughly with Tis-U-Sol or balanced salt solution.

The incision (Fig. 2) is similar to the one routinely used for stapedectomy or tympanotomy except for some posterior extension. Also the superior limb is extended. It is important, however, to avoid overextension of this superior incision—frequently this skin is the only solid attachment of the malleus handle since the head has been removed at a previous fenestration operation. A hypermobile manubrium is a difficult resting spot for the polyethylene strut or Teflon piston. The horizontal canal fistula is usually uncovered with the incision, and its pathology is studied.

Next, the superstructure of the stapes is removed (Fig. 2) and the oval window niche is denuded of all mucosa. The distance from the malleus handle to the footplate is then carefully measured (Fig. 2) and a polyethylene strut beveled and notched to the proper size. The author prefers 160 gauge tubing to the usual polyethylene 90 used routinely in stapedectomy.

A vein taken from the dorsum of the opposite hand is next prepared and placed on the promontory. The footplate is removed using hooks. The vein, with the adventitia down, is moved over the oval window. It is essential that a rather large vein be used to support the extra weight of the malleolar strut. The average strut is 5.25 mm. long. The prosthesis is next inserted between the malleus and vein.

More recently this technique has been modified with the advent of the Teflon piston. After the superstructure of the stapes is removed, a small area of the footplate is extracted, usually the posterior one-third, to accommodate the .8 mm. in diameter piston (Fig. 2). Following this a pocket between the manubrium and tympanic membrane must be created for the Teflon piston loop. This can generally be done with a sickle knife and small footplate picks. With this method there is a more secure fixation between the prosthesis and the malleus (Fig. 2).

Selection of Patient

Incus replacement provides varying degrees of success in four general types of cases: (1) the fenestrated ear, (2) the chronically infected ear, (3) the post-traumatic ear, and (4) the congenitally malformed ear.

The fenestration failure provides the greatest number of patients for this procedure. There should be an air-bone gap of at least 25-30 db. or more before this type of proce-

TABLE 1.—*Selection of Patient (Fenestrated Group).*

1. Little or no hearing gain following fenestration operation.
2. Successful fenestration with residual 25-30 db. bone-air gap.
3. Initial successful fenestration with subsequent bony or fibrous closure of fenestra.

TABLE 2.—*Causes of Malfunctioning Incus in This Series (30 Patients).*

	No. Patients
Fenestration	14
Non-Fenestration	
Infection	12
Congenital	1
Trauma	3

Fig. 1. Shea's palyethylene incus replacement.

dure is justified. More specifically the patient should not have serviceable hearing resulting from his previous fenestration operation. Sheehy divides these cases into three groups according to their post-fenestration course. The first group consists of those patients who got little or no significant hearing improvement following fenestration. Unfortunately, these patients usually will not gain significant hearing improvement from stapedectomy revision of their fenestrated ear. Sheehy had eight cases of this type in his series. Only two of these gained as much as 11 db. as a result of surgery. He no longer advises surgery in this type of case.

The second group includes those who have a satisfactory fenestration result (bone-air gap of 25-30 db.). Such persons have been further improved by stapedectomy in 55 per cent of the cases.

The third group, and most satisfactory patients for the incus replacement procedure, are patients who obtained a good initial gain from their fenestration operation only to lose it two to three months later as a result of fibrotic or bony closure. In Sheehy's series, 75 percent of this group obtained a hearing improvement to within 20 db. of the preoperative bone conduction level.

In summary, the incus replacement prosthesis should generally be recommended in a fenestrated ear when: (1) the bone-air gap is 25 db. or more, (2) there is a loss of serviceability of hearing, (3) there is the possibility of gaining serviceable hearing, or (4) in extreme mixed losses to provide the patient with better benefit from a hearing aid.

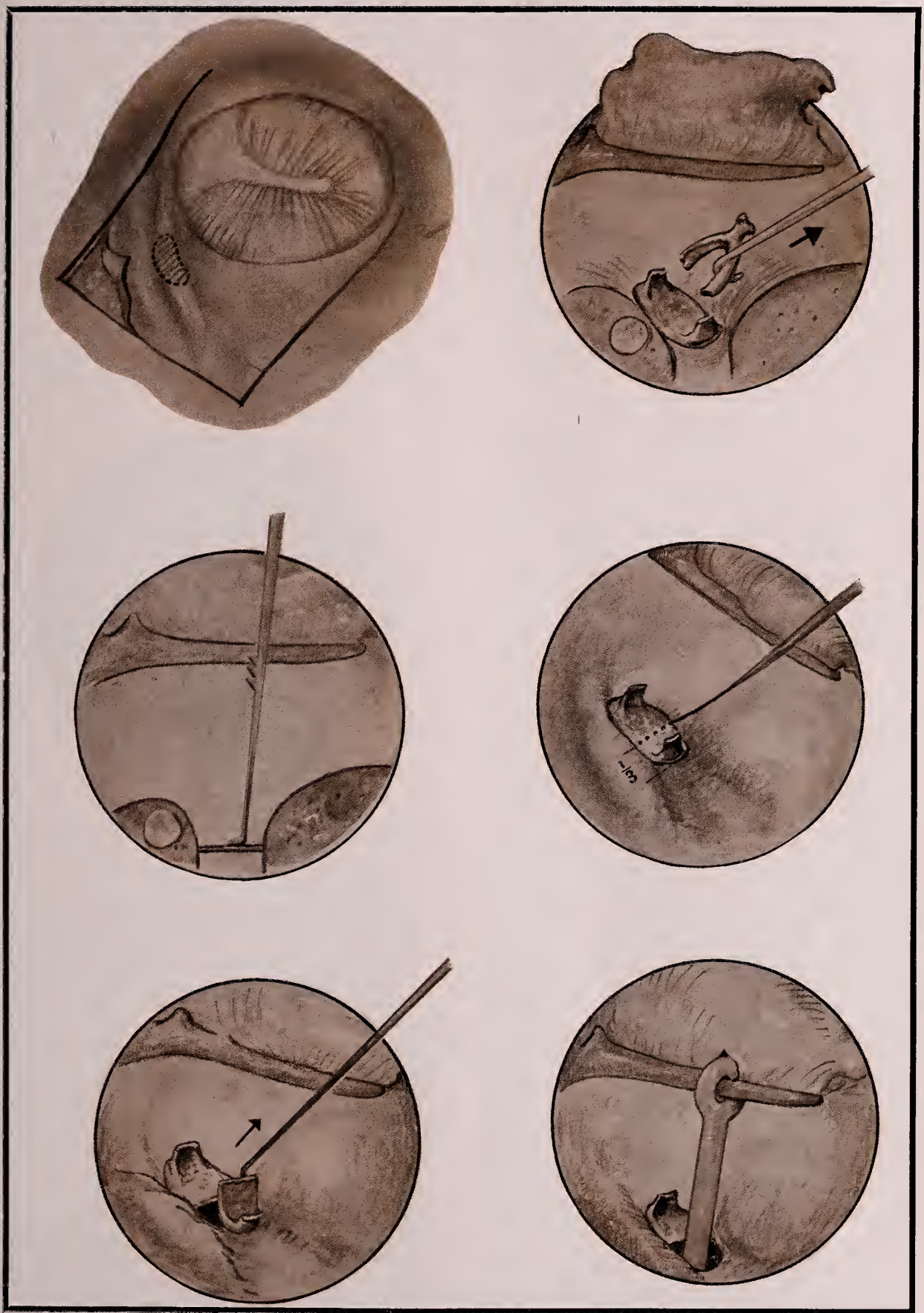
Selection of the patient to receive an incus replacement in the nonfenestrated ear contains more pitfalls. In addition to surgical removal of the incus to facilitate the creation of the fenestra ovalis, infection, trauma, and congenital abnormalities can obliterate this ossicle and interrupt the continuity of the chain. The most common is acute or chronic infection causing a necrosis of the bone, usually in the long process. The cholesteatoma would have to be included here, although an occasional epidermoid cyst may occur in the absence of infection.

The incus may also be absent or nonfunctioning secondary to trauma. Head trauma with or without demonstrable skull fractures is being seen more often. Blows to the temporoparietal region with resultant longitudinal fractures of the temporal bone are especially prone to cause injury to the middle ear ossicles. Many authorities believe the middle ear is always damaged in longitudinal fractures of the temporal bone. The tympanic membrane is frequently torn, with the discharge of blood or cerebral spinal fluid from the ear. The ossicular ligaments and articular capsules may be torn and the incus may be partly or completely disarticulated from the malleus or stapes, or both. If this dislocation of the incus is more than slight, a replacement prosthesis is often necessary.

A second type of trauma causing irreparable damage to the incus may result from accidental damage during stapes surgery. Unfortunately these cases seem to be increasing at an alarming rate. Most often the lenticular process and a portion of the long process of the incus are missing. Dislocation rather than actual bone destruction may be the problem. An incus replacement prosthesis may be needed here unless enough of the long process remains to receive a prosthesis directly.

A third class of patients often requiring the incus replacement procedure is the result of misguided embryologic development. Perhaps one of the most common congenital anomalies that involves the middle ear where

Fig. 2. Surgical technique is illustrated. The pictures at the top shaw the incisian in fenestratian cavity for secondary stapedectamy (left) and remaval af the stapes superstructure (right). Center, the distance between the malleus and oval windaw is carefully measured (left) and the faatplate is perfarated (right). The lower set shows remaval of the posteriar ane-third af faatplate (left) and the Teflan piston in pasitian between the malleus and oval windaw (right).



a normal tympanic membrane is present is partial or total absence of the incus.

Results

Fenestration Cases.—Fourteen patients who previously had undergone fenestration surgery were operated on by this method. Following the incus replacement procedure, ten of them obtained a hearing improvement to within 20 db. of the preoperative bone conduction level. Four achieved complete air-bone gap closure. Nine attained the 30 db. level.

Postinfection Cases.—In this group of 12 people there was a remarkable and significant difference in the hearing results obtained according to whether the tympanic membrane was intact at the time of surgery or was absent, requiring a graft as well as the malleolar strut. (This has been mentioned by previous authors; Sheehy lists the perforated ear drum as a contraindication to incus replacement.) Eight patients obtained postoperative hearing to within 20 db. of their preoperative bone conduction level, and five attained a complete air-bone gap closure. However, only one patient who had a perforation before surgery achieved any significant gain afterward. All others with deficient tympanic membranes either failed to gain or developed further conductive losses.

The often quoted high rate of cochlear loss associated with incus replacement surgery was not seen in this small series. In time, this ominous statistic will develop as it has in other groups of patients. The most satisfactory postoperative results in this postinfectious class were obtained behind the intact ear drum. There were eight patients in this group who had no sign of a tympanic deficit at the time of surgery. They often had bilateral conductive losses which except for the occasional scarring on the drum, would have surely been mistaken as otosclerotic. Two of them exhibited small cholesteatoma eroding the stapes and incus. All of these eight patients closed their air-bone gap to within 20 db. of their preoperative bone level, and five had complete closures.

Traumatic Cases.—Three patients in this series had conductive deafness as the result of trauma. One had an actual head injury while the other two had received ear damage during previous attempts at stapedectomy. The first patient fell from the back of a truck, striking his head. The roentgenogram showed a linear fracture in the left parietal bone and a linear fracture of the left squamosal portion of the temporal bone extending into the mastoid process. There was a slight haziness of the left mastoid air cells. The preoperative audiogram revealed a 40 db. conductive loss in the left ear. The tympanic membrane was intact. An exploratory tympanotomy was done. As the posterior canal wall flap was elevated, a fracture line could be seen in the posterior bone canal (Fig. 3). It entered the middle ear at

Fig. 3. Fracture of temporal bone with dislocation of incus.

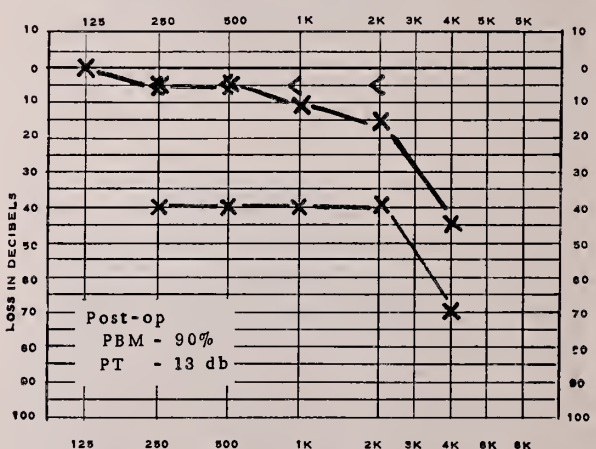


Fig. 4. The audiogram was made following head trauma with fracture through the middle ear and dislocation of the incus. The lower line on the graph shows preoperative air conduction (40 db.), and the upper line shows normal hearing obtained after incus replacement by polyethylene tube and vein graft.

about the attachment of the posterior malleolar ligament. The incus was dislocated from the stapedial head, and its long process displaced anteriorly. The anterior end of the stapes footplate was dislocated and riding above the oval window. It was not possible to reposition the misplaced ossicle, and a stapedectomy using a vein graft and polyethylene tubing no. 160 was performed. The postoperative audiogram showed an air-bone gap closure with hearing at a 13 db. level (Fig. 4).

In both of the other trauma cases, the entire long process of the incus was gone. Following surgery, the air conduction was within 10 db. of the preoperative bone conduction level in one case; in the other there was no appreciable gain.

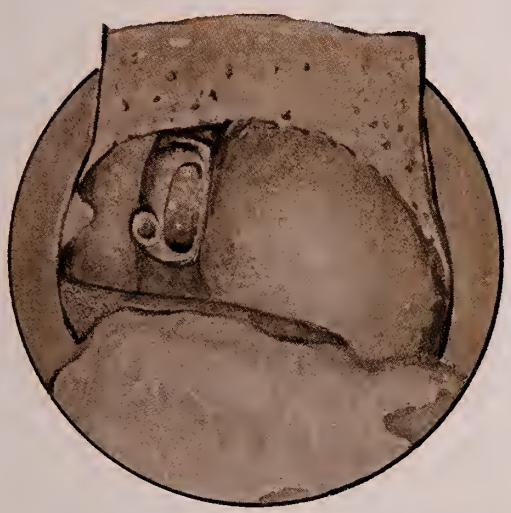


Fig. 5. Congenital middle ear deformity with absence of long process of incus and stapedial tendon.

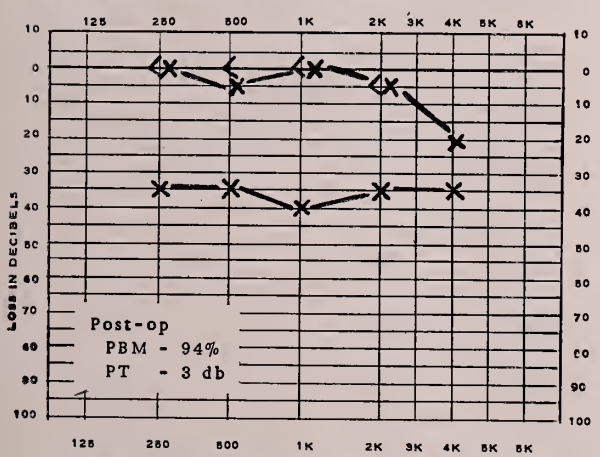


Fig. 6. The audiogram shows a conductive hearing loss of 40 db. in a 12-year-old boy (lower line). The postoperative audiogram was normal (upper line). At surgery, congenital absence of the incus was found, and reconstruction was with polyethylene and vein graft.

Congenital Case.—Developmental anomalies continue to be one of the most interesting problems encountered. Severely retarded development often precludes any form of oval window surgery, but when a relatively normal window is present gratifying results usually can be obtained. In this series, there was one case of this type where a malleolar strut could be utilized. This child was 12 years old when first seen. The pure tone audiogram showed a 40 db. conductive hearing loss. Discrimination was excellent (94 percent). The external auditory canal and auricle as well as the tympanic membrane failed to reveal any abnormalities. The opposite ear had normal hearing. There was no history of otitis media or of myringotomies. One year after the initial visit, an exploratory tympanotomy was done under general anesthesia. The middle ear space revealed an anomalous stapes with an absent anterior crus. The malleus appeared normal but the incus had no long process. The stapedial tendon was also missing (Fig. 5). Reconstruction of the chain was carried out using no. 160 polyethylene tubing and a vein graft. The postoperative pure tone audiogram done one month after surgery showed a 3 db. level with a discrimination score of 94 percent (Fig. 6).

Conclusions

From these results, and from similar observations by other authors, it is obvious that incus replacement procedures can restore hearing in a high percentage of cases. These techniques do not yet approach the 90 percent mark generally attainable with the conventional primary stapedectomy operation for otosclerosis. But with proper selection of patients this figure may be matched. The literature on this subject together with the results of this series point out several important factors to be emphasized if this type of surgery is to be done successfully:

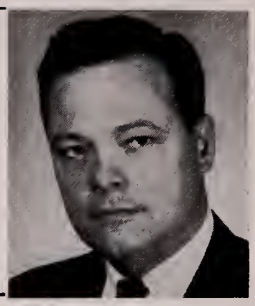
1. Unless the malleus, or at least the handle of the malleus, is preserved in doing modified and radical mastoidectomies these techniques are unsuitable. Conservation of this important ossicle should not supersede the primary eradication of disease, especially where cholesteatoma is present. But often with removal of the malleus head alone to expose the attic, and with careful microsurgical dissection under sufficient magnifica-

tion, all vestiges of matrix and infection can be removed leaving the important manubrium for secondary ossicular reconstruction at a later date.

2. The absence of infection in fenestration cavities and post-mastoidectomy ears is absolutely essential before any opening is made into the perilymphatic space. There were no severe cochlear losses in this series, but this may be because of the small size of the series. Operating in the presence of infection is an invitation to cochlear loss. As stressed by Sheehy, in fenestration cases the intact tympanic membrane appears to be another important prerequisite to this surgery. This seems even more essential in ears where the incus is absent because of infection. The results of this group of patients certainly bear this out. The total infectious group closed the air-bone gap to within 15 db. of the preoperative bone level in only eight cases, while all the patients with intact membranes closed to a similar degree.

3. Proper selection of the patient is another essential point. Lack of sufficient middle ear air space, areas of adherence of the tympanic membrane to the promontory, and even small perforations and cholesteatomas are easily missed in the routine otoscopic office examination. During the evaluation of patients in the latter half of this series, the Zeiss operating microscope was used routinely in the office preoperatively in every case. There is no guess work about the candidacy of a patient for this operation after a careful examination under 16 power, binocular vision. Recently, several previously acceptable patients have been rejected because of middle ear inadequacies viewed under this magnification.

4. The literature on incus replacement is largely directed at solving the problem of the poorly functioning fenestrated ear. Only casual reference is made to the use of these techniques in the ear which has lost its long crus from other causes. It is believed that equally good, if not better, results can be obtained in the postinfectious ear *provided* the tympanic membrane is intact. This may often necessitate staging of operations. This surgery seems even better suited to the traumatic or congenital ear problems, since the middle ear space usually is normal in these cases. The wide use of the incus replacement in all four of these groups of patients should be kept constantly in mind by the otologist.



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Congenital Sensorineural Deafness

BOB R. ALFORD, M.D., AND J. R. DICKEY, M.D.

CASE REPORTS

ACCORDING to reports based on incidence, there are approximately 180,000 people in the United States who are totally deaf. Sank and Kallmann[10] found that 94 percent of these people became afflicted before the age of eight years. It was their impression that 55 percent of these patients can be assumed to have a nonhereditary cause for their deafness, and 40 percent to have inherited deafness.

This paper has two purposes: one is to present the author's observations from patients with total or almost total hearing loss; the other is to review and crystallize a working classification for deafness that begins at birth or is acquired shortly thereafter.

Presentation of Cases

Seven patients with severe sensorineural deafness have been admitted to the Methodist Hospital for studies. All but one of these patients have had a hearing loss since birth. One patient, who has dental malformations and saddle nose, developed a progressive sensorineural hearing loss in the second decade of life.

Each patient has had an electronystagmography examination (ENG) of his vestibular reaction to caloric stimulation. Six of the patients have had electroencephalographic (EEG) studies. All seven patients have been subjected to general anesthesia and recordings made of the electrical activity of the cochlea.

At the time of surgery, the ear drum is reflected and a Teflon-coated platinum ball tip electrode is placed into the round window niche. No malformations of the round window have been observed. In most instances a round window reflex on palpation of the stapes has resulted in motion of the electrode.

Sound is delivered into the ear speculum from a driver unit (speaker) by means of a flexible brass tube. The ear is stimulated with click sounds and with pure tones of 250 cycles per second, 500 cps., 1 kc., 2 kc., 4 kc., 8 kc., and 12 kc. The stimulation is controlled by an electronic programmer. For each stimulus there are two intensities, a high and a moderate one (Table 1). The responses are simultaneously observed on the face of the oscilloscope and recorded on tape. The tape records are permanently preserved in the files and referred to for study. They are photographed by playing from the tape deck to the oscilloscope face.

The normal response to pure tone stimulation as observed at the round window is a sine wave reproduction of the stimulus, which is referred to as the cochlear potential (coch-

TABLE 1.—*Stimulus Sound Pressures 2.5 Cm. from Tip of Sound Tube.*

Frequency	Intensity (db)	
	Low Stimulus	High Stimulus
250 cps.	77	88
500 cps.	92	106
1000 cps.	96	111
2000 cps.	103	118
4000 cps.	108	123
8000 cps.	98	112
12000 cps.	72	87
click	84 (approximate)	93

Findings in seven patients studied by cochlear recordings are presented and compared with other reports. Classification of congenital deafness is reviewed, and the limitations of usual classifications of inner ear malformations are discussed.

lear microphonic). From click stimulation one sees three characteristic phenomena: the cochlear potential, followed by the eighth nerve action potential, which has two components referred to as the N_1 and N_2 (Fig. 1).

The authors have observed essentially three types of patients so far as their cochlear potential activity is concerned (Table 2). One is the patient in which all of the potentials are present. Another is the patient in which only partial or incomplete responses are observed with absent eighth nerve action potentials. The other type has no electrical activity, that we can detect at the round window, to any kind of sound stimulation.

TABLE 2.—*Summary Physiologic Information; Patients With Congenital Deafness.*

	Normal	Abnormal	Absent
Cochlear Potential Responses	1	3	3
ENG	3	2	1
EEG	2	4	

The patient who has all potentials present has a severe sensorineural hearing loss and at audiologic testing responded only to sound at the 250 cps., 500 cps., and 1 kc. frequencies. This patient has a nearly normal EEG and does demonstrate a "K" complex (a characteristic wave form in the EEG that results from sound stimulation) on auditory stimulation. His vestibular response as measured by the electronystagmography study is normal. On stimulation of his ear by pure tone and click stimuli during cochlear potential recording, he is observed to have a faint 1 kc. response, a good 2, 4, and 8 kc. response, and a complete click response with an eighth nerve action potential (Fig. 1). We may have been unable to record the cochlear potential (cochlear microphonic) from the 250 cps. and 500 cps. stimuli because the intensity of the stimulus at this

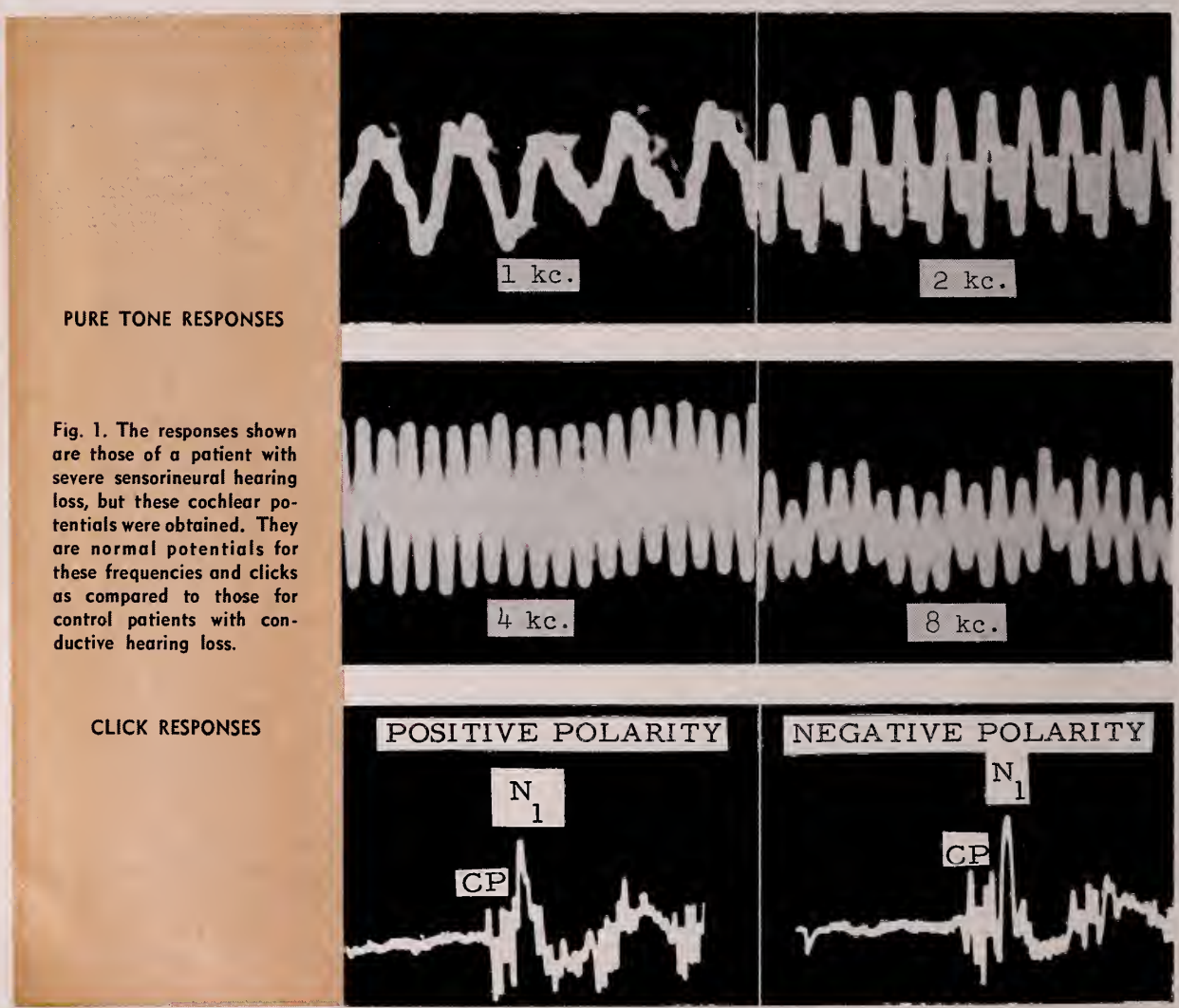

Fig. 1. The responses shown are those of a patient with severe sensorineural hearing loss, but these cochlear potentials were obtained. They are normal potentials for these frequencies and clicks as compared to those for control patients with conductive hearing loss.

frequency is reduced over that of other frequencies.

Three patients have incomplete cochlear potentials to sound stimulation. Two of the patients have only the cochlear potential part of the click response and an island of pure tone cochlear microphonic (Fig. 2). The other patient has congenital stapes fixation and had prior exploratory surgery in this ear. The surgeon was unable to complete the stapedectomy after perforation of the footplate. This patient has an abnormal EEG with slow activity and absent focal signs. The ENG is inconclusive. In this patient there is a complement of pure tone cochlear microphonics but there is no action potential to the click. A good click cochlear potential response is seen, but no N_1 or N_2. This patient has a moderate conductive component to her hearing loss. One of the patients with incomplete cochlear responses has a normal EEG and normal vestibular reactions to caloric stimulation. The other patient is the one with the saddle nose deformity, notching of the teeth, and in the ear explored there is total deafness audiologically. There is a hypoactive caloric response as measured by the ENG. The EEG in this patient is poorly organized with slow activity. The serology is nonreactive. Her hearing loss began in the second decade of life.

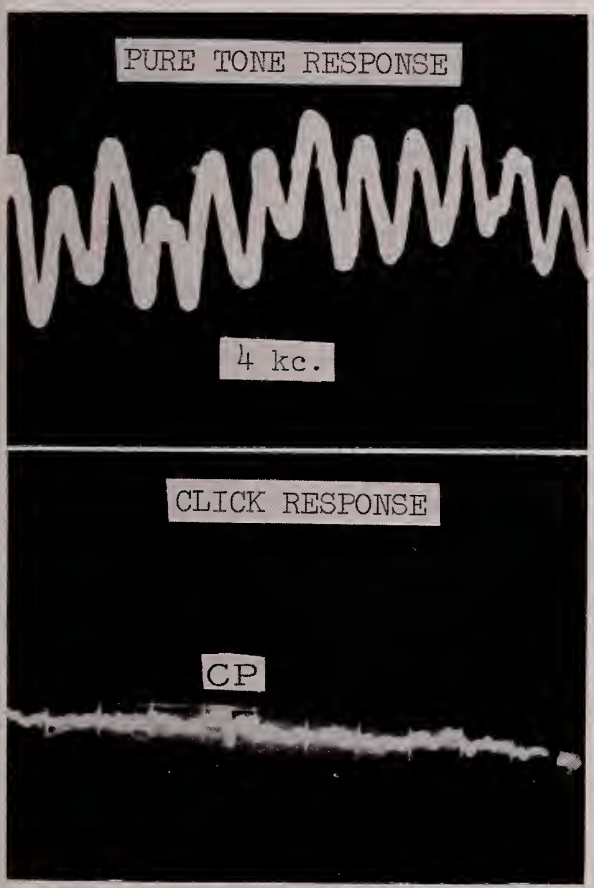

Fig. 2. These responses demonstrate a pure tone cochlear potential at 4000 cps., and only the cochlear potential part of the eighth nerve action response to click stimulation. The latter phenomenon has been proven to be an artifact in subsequent cases.

Three patients have completely absent cochlear potentials even on intense stimulation. One of these patients has a conductive component to the hearing loss as suggested in the audiogram in that a bone conduction response on audiometric testing is observed at the 250 cps. and 500 cps. level, at 25 and 50 db respectively. The pure tone air response at those frequencies is 80 and 95 db respectively. There are no responses to other pure tone stimulation on the audiogram. The patient's EEG is abnormal with slow activity and asymmetry, and there is a hypoactive caloric response on ENG. At the time of surgery the stapes was questionably fixed and was removed after a preliminary recording was made. A prefabricated stainless steel wire was inserted into the oval window and attached to the incus, but there was no cochlear potential response. The postoperative audiogram shows no improvement in hearing. Histologic section of the stapes demonstrates no abnormality of the stapes footplate. Another patient with absent cochlear potentials has a measurable audiogram for all frequencies. The average pure tone loss in the speech frequencies is 75 db in the left ear and 82 db in the right ear. No caloric response is observed on ENG study. An EEG was not obtained prior to surgery in this patient. No cochlear potential can be measured at any frequency. There is no response to the click. Because the audiometric study was done one year before the surgical procedure, a follow-up audiogram is being obtained to determine if there has been progression in the hearing loss. Since the authors have not failed to record the potentials from people with conductive hearing loss, they believe that the recording reliability is satisfactory. There are other congenital anomalies in this patient, among them is a cardiac malformation for which cardiac surgery has been performed. The third patient with absent cochlear responses has no hearing that could be discerned by pure tone audiometry and has a questionable response to free field pure tone of 500 cps. at 95 db. The EEG demonstrates fusing in the occip-

ital area with positive spike discharges, 14 and 6 per second. Normal ENG studies are obtained in this patient.

Discussion

Most classifications of congenital sensorineural hearing loss are based upon retrospective reasoning. Numerous classifications have been proposed, and these are constantly revised when additional anatomical and physiological information is obtained. The continued studies of Altmann,[1] Kelemen,[3,4,5] Lindsay,[6] and Wolff[11] have given information concerning the histopathologic findings in patients with deafness from birth.

There are two broad categories into which these patients may be grouped (Table 3).

TABLE 3.—*Classification of Congenital Deafness.*

1.	Genetically determined
	a. Embryonic malformation
	b. Embryonic arrest and degeneration
2.	Acquired
	a. Prenatal
	b. Perinatal
	c. Postnatal

Deafness from birth may be the result of genetically determined factors or may be acquired as the result of prenatal, perinatal, or postnatal events. In genetically determined deafness two types have been documented. One is characterized by defects in the inner ear which occur during embryonic development. The other is characterized by degenerative changes which develop after birth or after complete embryonic development of the inner ear. In animals, representatives of each of these types of genetically determined deafness have been described. Human cases presented by Altmann[1] confirm this categorization.

The acquired sensorineural deafness that manifests itself before speech has developed may be caused by prenatal infections (rubella, syphilis), metabolic diseases, or by drug intoxications during pregnancy. In these instances, normal embryonic development of the ear has progressed up to the point of insult. Disease of the inner ear ensues and degenerative changes are manifest. In the perinatal period, complications of pregnancy, birth injuries, and blood incompatibilities with kernicterus and erythroblastosis fetalis are prominent causes of deafness. In the postnatal period hypoxia, meningitis, syphilis, ototoxic drugs, and numerous childhood diseases are responsible for the induction of sensorineural hearing loss.

Dependent upon reports, wide variations in the incidence of the two main groups of deafness have been observed. Sank and Kallmann[10] state that 40 percent of people with deafness from birth have congenital deafness on a hereditary basis. Other reports (Zonderman[12]) place this incidence much lower. Certainly an important aspect of any study must include genetic investigation of each family history.

Classically, four histologic malformations have been described. The most severe is the Michel malformation, which involves the petrous bone as well as the inner ear. Often in these malformations no structure is recognizable as the inner ear. The petrous bone itself may not form at all. The Mondini malformation (Mondini-Alexander type) is characterized by development of a single curved tube representing the cochlea with similar immaturity of the vestibule and semicircular canals. In the Bing-Siebenmann malformation the bony labyrinth is well formed, but the vestibular and cochlear membranous part is underdeveloped. The Scheibe deformity is cochleo-saccular in location and the vestibular system (semicircular canals and utricle) is normal.

Classifications of this type, based on histologic deformity, leave much to be desired. In only the Michel or Mondini-Alexander type may genetically determined deafness be acceptable. However, the patient with familial hereditary deafness, in which the inner ear degenerates after complete embryonic development and perhaps after a few years of life, does not fit into the classical classification. By the same token, it is not proper to think of the Bing-Siebenmann and the Scheibe malformations as being entirely acquired, for certainly in the animal kingdom

Dr. Alford presented this paper before the Section on Otolaryngology, TMA Annual Session, April 25, 1964, in Houston. He is an assistant professor in the Department of Otolaryngology at Baylor University College of Medicine.

certain lesions in mice (Shaker I, DF/DF) and the dalmatian dog are hereditary. The child with Waardenburg's syndrome (characterized by deafness, white forelock, epicanthal fold, broad nasal bridge, and heterochromia iridis) has a genetically determined lesion.

Of paramount importance in a study of congenitally deaf children is the overall performance of the patient as studied by physiologic methods. Not only is it important to have their audiometric profile and their psychological evaluation, but it is extremely important that they have carefully done vestibular studies and electroencephalographic studies with emphasis on the presence or absence of the "K" complex. Oftentimes, although auditory arousal stimulation is a matter of routine in EEG studies, this is not an area about which remarks are made.

Cochlear potential studies permit a confirmation of other impressions. This is a research tool that gives information that otherwise is not available and permits us to learn more concerning the pathology. It should not be misconstrued, however, to imply a definitive diagnosis, nor should the information be used to influence therapy.

The physiologic findings in the seven patients studied at the Methodist Hospital are exemplary of those that have been observed by Ruben and associates[7,8,9] and Bordley and associates[2] at the Johns Hopkins Hospital in Baltimore. They have identified six possible types of deafness based on correlation of the vestibular test and cochlear physiologic responses. Six of our patients fit well into five of the six types that are alluded to by Ruben.[9] One of our patients has total labyrinthine deafness characterized by absent cochlear potentials, eighth nerve action potential, and vestibular response. Two patients have cochlear deafness as described by Ruben[9] in which the cochlear potential and eighth nerve action potential are absent but the vestibular response is present. One patient, however, has hypoactive vestibular responses on ENG. Another patient possibly represents total deafness of the eighth nerve. In this situation the cochlear potential response to click and to 4000 cps. stimulus is present, but the eighth nerve action potential is absent, and the vestibular response is almost absent. One patient probably has partial deafness of the eighth nerve. She has a cochlear potential to the click response and 4 kc. pure tone, but an absent eighth nerve action potential and normal vestibular response. We have observed patients with central deafness and absent vestibular responses. One patient probably has central deafness, as the cochlear potential and eighth nerve action potential are present and the vestibular response is normal.

These patients are presented to generate interest in the problems of those individuals who, because of their deafness, are unable to communicate in conventional ways. Also it is hoped that this paper may crystallize the thoughts of the reader with regard to children who have severe sensorineural deafness.

ACKNOWLEDGMENTS

Methodist Hospital, and particularly Dr. J. Charles Dickson, chief of otolaryngology, and Mr. Ted Bowen, the administrator, arranged for these studies without cost to the patient. M. D. Anderson Foundation was also a benefactor in supporting the instrumentation necessary in this study. Dr. A. C. Coats did the electronystagmography studies. Houston Speech and Hearing Center permitted the authors to review their audiograms for the cases presented. Mr. Ike Estes helped to get the apparatus functioning. This project is supported in part by USPHS Corollary Grant B-4099.

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Treatment of Hepatic Coma By Hemodialysis

CASE REPORTS

HEPATIC COMA occurs most frequently in chronic fibrotic liver disease.[6] The second most common cause is acute viral hepatitis in which coma has a far worse prognosis.[6] There is no specific therapy for fulminant hepatic necrosis. The role of the physician is to achieve a favorable physiologic environment in which spontaneous hepatocellular regeneration may occur. Bowel sterilization, protein denial, and adequate carbohydrate to minimize protein catabolism are the generally accepted modes of therapy. The role of steroids is yet to be defined.

In acute tubular necrosis a similar set of circumstances is found. In this entity and in various intoxications, extracorporeal dialysis offers a rapid and effective method for removing exogenous and endogenous substances from the body. This suggests a role for dialysis in acute hepatic necrosis. Sherlock[6] used hemodialysis in one case of acute hepatitis in coma without success. The authors are unaware of any other reported cases of its use in acute hepatitis. However, it has been used with fair temporary success in hepatic coma complicating chronic liver disease.

A young male moribund with relentlessly progressing hepatic coma offered the opportunity to evaluate the efficacy of hemodialysis when the usual therapy and large doses of steroids were unsuccessful.

Case Report

An 18-year-old white male serviceman with four weeks of active duty was admitted to the Wilford Hall USAF Hospital, Lackland Air Force Base, Tex., on Aug. 22, 1962, because of jaundice which appeared on the day of admission. He had weakness and fatigue of four days' duration; nausea, vomiting, anorexia, and right upper quadrant pain of three days' duration; and dark urine, light stools, fever, and headache of one day's duration.

He had not been exposed to hepatotoxins, transfusion, or injections. More than one year prior to admission, he had taken narcotics intravenously on one or two occasions. A friend involved in the use of narcotics died of hepatitis at that time.

The patient was an alert, extremely icteric, muscular, well nourished, moderately ill, young male with an oral temperature of 99.2 F. A sharp tender liver edge was palpable 5 cm. below the right subcostal margin. The span of liver dullness was 14 cm. He had minimal generalized adenopathy. Several tattoos, more than two years old, were seen on the arms and chest. The stool was clay colored and guaiac negative.

Laboratory data confirmed marked derangement of liver function compatible with acute infectious hepatitis. (See Table 1 for serial determinations.)

On the sixth hospital day, the patient became progressively confused, irrational, and combative. Despite the institution of parenteral alimentation with adequate glucose, 8 gm. of neomycin by nasogastric tube, enemas, and L-arginine 20 gm. intravenously every six to eight hours, the patient entered grade four coma on the seventh hospital day. (See Fig. 1.) At this time the blood ammonia was 360 μg. percent (normal less than 120 μg. percent) and the electroencephalogram was compatible with hepatic coma. The span of liver dullness had decreased to 8 cm. Dexamethasone was given in a dosage equivalent to 1,500 mg. of hydrocortisone a day parenterally. He continued to deteriorate clinically. The pupillary reflexes were sluggish, the deep tendon reflexes were hyperactive, and Babinski's sign was positive bilaterally. Multiple petechiae developed over the entire body. At this time his prothrombin time remained between 10 percent

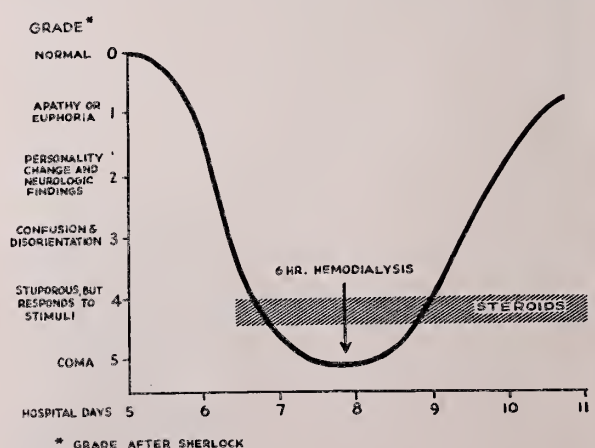

Fig. 1. Correlation of course of coma in relation to therapy.

Survival of a patient in hepatic coma from acute infectious hepatitis is credited to hemodialysis.

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and 20 percent of normal despite large intravenous doses of vitamin K. On the morning of the eighth hospital day, he was unresponsive to painful stimuli. At this time, a six-hour hemodialysis under regional heparinization was performed; 625 gm. of albumin were added to the dialysate to enhance bilirubin removal.[2] Approximately 12 hours after dialysis, he responded to pain and his reflexes were normo-active. Thirty hours after dialysis, spontaneous movements occurred and 60 hours after dialysis, he was talking and answering simple questions. Steroids were decreased progressively during the next several days. (See Fig. 1.)

On the 14th hospital day, massive upper gastrointestinal hemorrhage occurred. Esophagogastroscopy revealed a superficial ulcer on the posterior wall of the stomach at the junction of the upper and middle thirds of the fundus. Despite intensive medical management, on the 16th hospital day the ulcer was imbricated and a gastrostomy tube was inserted because of persistent bleeding. The patient required a total of 19 units of blood before, during, and after the laparotomy.

On the 27th hospital day, ascites as well as presacral and ankle edema appeared. Spontaneous diuresis ensued on the 31st hospital day, and the patient lost 14 pounds in seven days. His course thereafter was one of gradual improvement. On the 96th hospital day, the liver function studies had returned to normal, the liver was normal on physical examination, and there had been a dramatic return of the overall muscle mass of the body.

The liver biopsy performed on the 28th hospital day showed massive necrosis and collapse of the hepatic tissue. (See Figs. 2 and 3.) A liver biopsy on the 49th hospital day showed remarkable reconstruction of hepatic parenchyma with continued evidence of active inflammation. (See Fig. 4.) At this time, there were minimal abnormalities in liver functions. The third liver biopsy was obtained on the 96th hospital day. It showed evidence of continued regeneration with minimal fibrosis and continued inflammatory reaction. (See Figs. 5 and 6.)

Discussion

Hepatic coma as the result of infectious hepatitis has a mortality rate approaching 100 per cent in various series despite the use of the generally accepted measures. Katz and others,[4] who now have a ten-year experience, favor massive steroid therapy in the range of 1,500 mg. of hydrocortisone or its equivalent daily, and report a survival rate of approximately 40 per cent in 23 cases. Wildhirt,[7] reporting on the use of steroids in 42 cases (27 of precoma and 15 in coma), had

TABLE 1.—*Serial Laboratory Data.*

Hospital Day	Bilirubin (mg.) Total	Direct	SGPT (Sigma units per ml.)	Alkaline Phosphatase* (Sigma units per ml.)	Albumin (gm.)	Na	K	CO_2	Prothrombin Time (%)	Hemoglobin (gm. %)
						(mEq. per L)				
1									28	
2	20.3	14.0	770	4.09						
5	15.5	7.4	162	1.2						14.2
6	31.0	20.5		7.2		136	3.5	35.1	10	15.8
7	31.0	28.5	250			136	3.5	35.1	16	15.8
8–Pre-dialysis	37.0	25.5				133	3.6	21	16	12.4
8–Post-dialysis	24.5	17.1	27							
10	31.5	22.4	27			135	4.5	16.4	20	12.2
12	29.3	21.3		2.51		139	6.4	20.2	49	8.5
15	18.5		15	2.39		142	3.5	21.4	49	14.0
30	10.4	0.8	112	3.7	1.7	136	4.0	25	73	11.8
49	1.6	1.4	52	1.8	2.7				83	12.5
96†	0.5	0.13	10	2.6	3.3				100	13.6

* Normal Adult, 0.8-2.3 Sigma units per ml. (Bessey-Lowry)
† BSP 4.5%

a survival rate of 60 percent overall and 53 percent in coma. These encouraging reports are not, however, universally shared.

Extracorporeal dialysis is an accepted method for rapidly removing certain exogenous and endogenous substances from the body. A definite role for its use is recognized in the treatment of drug intoxication and acute renal failure. Hemodialysis is more rapid than peritoneal dialysis in restoring the normal fluid and electrolyte balance and in removing exogenous substances. However, it requires an experienced and proficient team of physicians and technicians with accurate laboratory support. With a high-pressure, high-flow system, such as in the Kolff twin coil apparatus, constant attendance of a physician is mandatory. Peritoneal dialysis, albeit less rapid, requires minimal training, considerably less laboratory support, and does not require constant physician monitoring. Hemodialysis requires heparinization. In patients with hepatic decompensation, the coagulation mechanism already shows significant derangement. The real danger of such bleeding may be essentially eliminated with regional heparinization, that is heparinizing the blood in the line to the artificial kidney and neutralizing the heparin with protamine in the line returning to the patient.

Kiley and others[5] successfully reversed hepatic coma occurring in chronic liver disease as a result of an acute and remediable insult with hemodialysis. He reported temporary success in four of the five cases and suggested a role for hemodialysis in cases where the insult was due to massive gastrointestinal hemorrhage or to uremia. Jones and others[3] noted improvement in three cases treated with peritoneal dialysis, and offered the same indications.

Grollman and Odell[2] treated a patient with acute infectious hepatitis complicated by hemolytic anemia and uremia with both peritoneal and extracorporeal hemodialysis. They obtained marked enhancement of bilirubin removal by the addition of 200 mg. of 25 percent human albumin to the peritoneal dialysate. Their patient apparently did not evidence hepatic coma but ultimately succumbed with uremia on the 18th hospital day. Etteldorf and others[1] had similar enhancement with salicylate intoxication in dogs and recommended the use of albumin to remove other anions that might have an affinity for

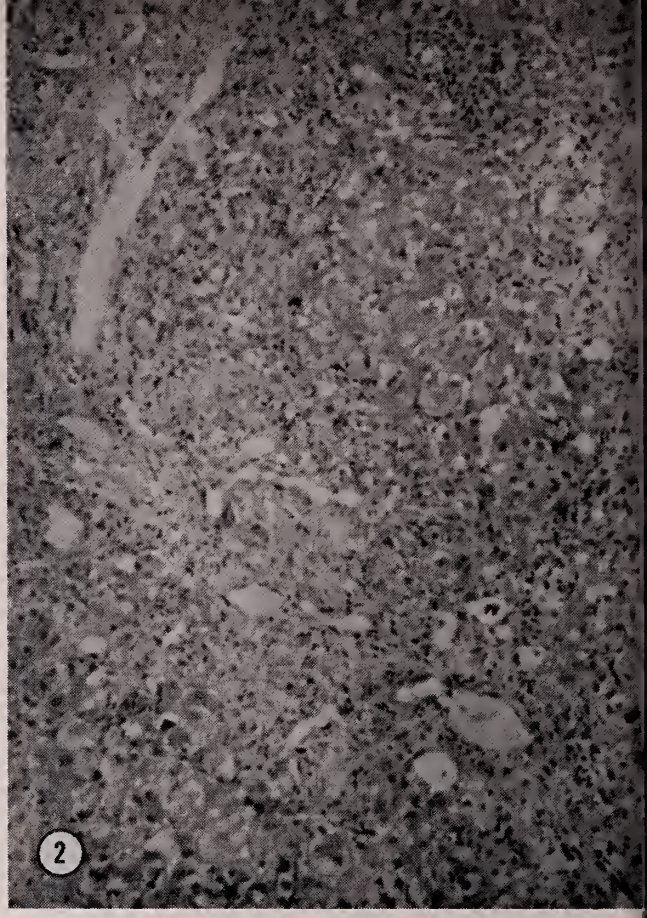
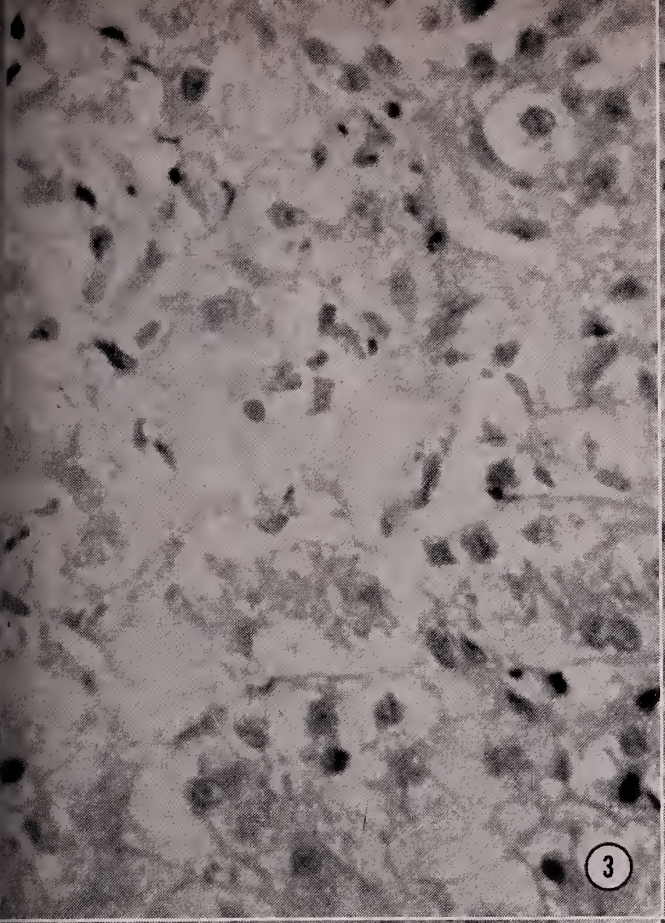


Fig. 2. Liver an 28th hospital day demonstrating extensive parenchymal collapse with severe hepatic cell degeneration. Nate marked diffuse infiltrate mast cancentrated abaut partal areas. Hematoxylin-easin. X 55. Fig. 3. Liver an 28th haspital day. Nate many of the cells as mere ghast autlines withaut cytaplasm or nuclei; considerable bile stasis intracellularly and in the canaliculi. Many af inflammatary cells are neutraphils. Reticulum stains (nat shawn) canfirm massive callapse. Hemataxylin-easin. X 236.5. Fig. 4. Liver an 49th hospital day with hepatic parenchymal cells shawing marked praliferatian and regeneration (binucleate cells). There is much less evidence of degeneratian (clearing of cytaplasm and ballaaning af cells). Inflammatary cells are decreased in number and appear ta be chranic. Hemataxylin-easin. X 236.5. Fig. 5. Liver on 96th hospital day demonstrating narmal labular pattern and occasional binucleate cells. Mild chronic inflammatory infiltrate in partal areas indicates same continuing activity. Hematoxylin-eos'n. X 55. Fig. 6. Liver on 96th hospital day. Minimal fibrasis in peripartal areas prahibits diagnasis af cirrhosis. Massan. X 55.



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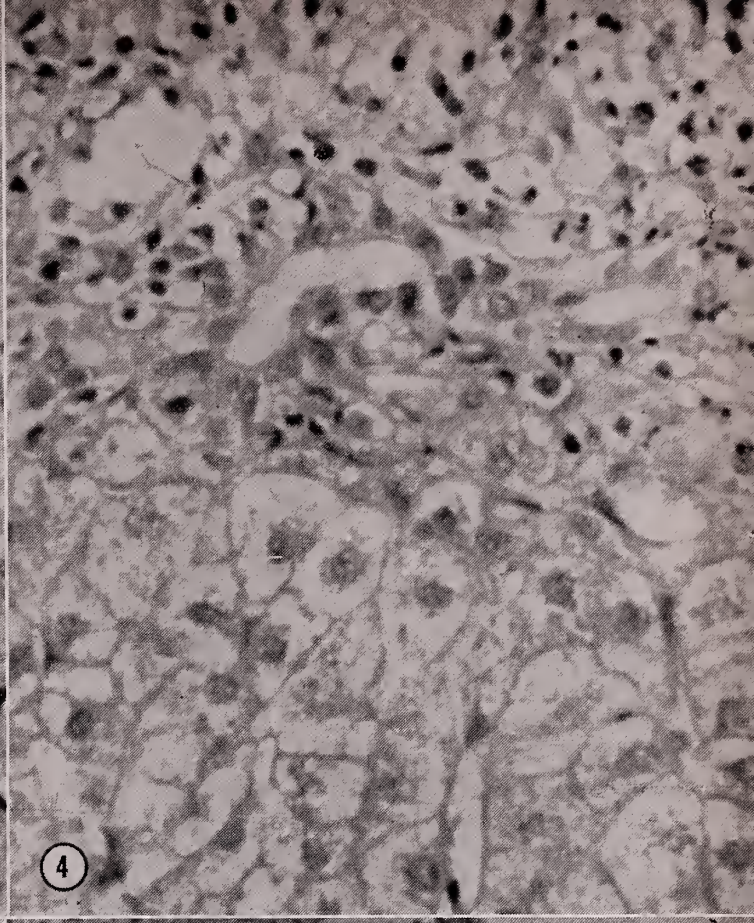

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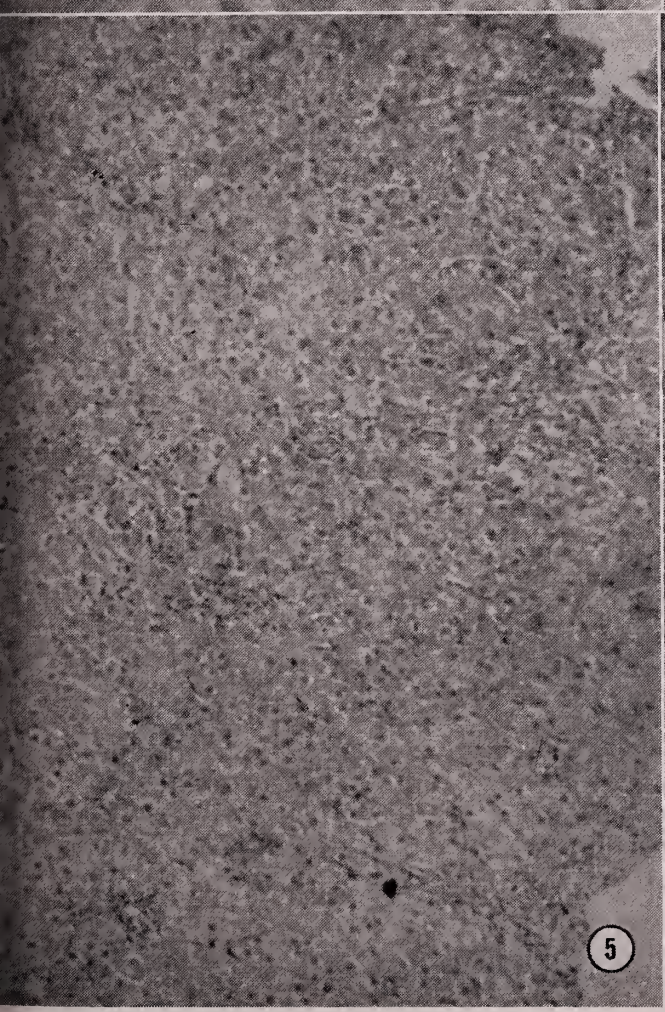

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albumin, such as barbiturates.

This patient was treated with the usually accepted measures to which L-arginine and steroids were added with little or no improvement. Katz's group found that an improvement in the prothrombin time in the initial 24 to 48 hours was the earliest reliable sign of a favorable prognosis while using steroids. This patient did not evidence any improvement in prothrombin time but displayed further deterioration in his coagulation mechanism, as evidenced by the appearance of petechiae. The depth of his coma continued to increase. When faced with the imminent demise of this teenage male, evaluation of the possible benefit of hemodialysis was undertaken.

The time relationship of return to consciousness was compatible with the effects of dialysis in other patients treated for hepatic coma and uremic encephalopathy. Whether or not this was casually related cannot be ascertained because of the multiple modes of therapy prior to and subsequent to dialysis. It is not inconceivable that the other therapy alone might have effected the same reversal. However, with the knowledge that ammonia and bilirubin are dialized, and the time relationship to clinical improvement, it is believed that hemodialysis played an integral role in an overall therapeutic approach that resulted in survival.

The hepatic coma of infectious hepatitis accounts for an overwhelming mortality in young and otherwise healthy people. It would seem justified to use extracorporeal dialysis as adjunctive therapy in those cases of fulminant hepatitis in coma that are unresponsive to all other measures. This single case supports the need for more information on the efficacy of this measure because even large medical centers will have only a rare case warranting such therapy.

When an attempt is made to suggest indications for extracorporeal dialysis in hepatic coma, it is believed that chronic liver disease and acute hepatitis should be considered separately. In the former, the indications of Kiley and others, namely, massive gastrointestinal bleeding and/or uremia are worthy of consideration. However, long-term results will necessarily be poor and the immediate efficacy will depend on the control of bleeding and/or the reversibility of any renal lesion. Extremely careful selection may establish a rare patient as a candidate for such therapy.

Dr. John G. Weg and the other authors are air force officers who treated this patient at Wilford Hall USAF Hospital, Lackland AFB.

In hepatic coma due to acute infectious hepatitis, the patient is usually young and otherwise healthy. If the immediate problem can be controlled, the expectancy for long-term survival is good. In such cases, the use of extracorporeal dialysis is suggested (1) when there is evidence of progressive deterioration or lack of response to the use of accepted modes of therapy and massive steroids for a reasonable period of time, namely, 24 to 72 hours, or (2) when uremia or anuria intervene.

Summary

Survival from hepatic coma in acute infectious hepatitis, the second most common cause of such coma, is rare. Therapy is directed toward elimination of any unnecessary stresses on the liver and toward meticulous supportive care in an effort to buy time for the liver to assume its vital functions. A case in which hemodialysis is believed to have served this end is reported. Indications for extracorporeal dialysis in hepatic coma are enumerated. Further assessment of extracorporeal dialysis in the coma of infectious hepatitis is suggested.

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The Migrant Labor Situation

DAVID M. COWGILL, M.D.

RESEARCH & INVESTIGATION

THE COUNTY of Lubbock has a population of 166,417 (est. 1962), approximately 80 percent of which live in the city of Lubbock. The county has an area of 892 sq. mi. The economy is largely agricultural using large numbers of seasonal migrant laborers.

During the first nine months of operation of the Migrant Health Program, which began at the end of the chopping season and continued throughout the harvest season, there were 15,000 migrants. The migrants began arriving in small numbers in April for planting, and increased as chopping season opened. The great influx for cotton harvest began Oct. 1 and continued until Dec. 31. The last migrants left Lubbock County about Feb. 15.

Nearly all the migrants were United States citizens of Mexican descent. They ranged in age from birth to 60 years. Most of the migrants were from South Texas.

Because these migrant laborers were known to be subjected to poor living quarters and poor sanitation and their disease rates were high, a federal grant was requested through the Public Health Service. The objectives of the program were to improve the public health of domestic migrants through establishing environmental health services for the domestic migrants that are not presently available. These services were to include sanitation at migratory labor camps and population centers, instituting tuberculosis case finding and follow-up programs, well child and immunization clinics, and routine visits by the public health nurses to the schools with migrant children, home visits in migratory labor camps and other concentration centers in Lubbock County, and establishing a record system especially for the migrants.

The grant provided two full-time nurses, one sanitarian, and one clerk with financing for the equipment and supplies to carry out the program. During the last quarter of the year additional funds were appropriated for the employment of a full-time public health educator.

The nurses work under the supervision of the health department nursing supervisor; the sanitarian works under the supervision of the health department public health engineer; and the public health educator works under the direction of the public health director.

When the nurses first got into the field they found that they had no clean place in the migrant camps in which to set up their clinics. The public health director and the engineer visited the surplus property depot where they found a panel truck which could be converted into a mobile clinic. This truck was purchased and put in running order for $185. Then it was equipped with cabinets, examining table, sink, water tank, and heating, cooling and lighting fixtures. The total cost was under $1,000.

The nurses took the mobile clinic to each of the migrant labor camps located at the cotton gins and to seven schools where mi-

Poor disease rates and poor sanitation of migrant workers in the Lubbock area led to a health education and health services program which has received the confidence of the migrants. Community attitudes toward migrants have also improved. Still, there are several barriers which the program must overcome to improve sanitation and educational levels.

grant children attended at least once during the program operating period. Four camps were visited every two weeks and 14 camps were visited monthly for four months. Four schools were visited monthly for three months; three schools were visited once.

The mobile clinic was designed to provide generalized services involving all aspects of public health. The clinic sessions began at 7 or 8 a.m. and averaged five hours per session. Eighty-eight sessions were held with approximately 46 individuals seen at each session. A total of 4,000 migrant laborers were seen in the four-month period during which the mobile clinic operated. This figure includes return visits to the mobile clinic. An undetermined number of follow-up visits were made without the mobile clinic.

Among the migrant laborers seen at the mobile clinic the greatest health problem was upper respiratory infection, especially among children. Diarrhea and skin conditions were prevalent among this group also. Other conditions noted among the migrants

TABLE 1.—*Major Conditions Requiring Care Oct. 1-Dec. 31, 1963, Lubbock County, Texas.*

Age	Upper Respiratory Infection			Diarrhea			Skin Conditions		
	M	F	T	M	F	T	M	F	T
0-15	46	70	116	17	27	44	11	24	35
16-49	13	23	36	2	4	6	1	1	2
50 & Over	4	4	8					3	3
Total	63	97	160	19	31	50	12	28	40

M=Male. F=Female. T=Total.

included tuberculosis, gastrointestinal disturbances, burns, and dental problems.

Referrals were made when necessary to various sources in the community. Most of the referrals were made to the clinics operated by the Lubbock City-County Health Department, in particular the maternity, x-ray and dental clinics. Additional referrals were made to private physicians and to the Parkdale Clinic, operated locally by the Baptist church. Personal or telephone follow-up of referrals was the responsibility of the nursing personnel. Most referrals were completed.

The personal health record (PhS-3652) is being used by the Lubbock Migrant Health Program. Five hundred forty-eight individuals were given the record with instructions to present it the next time they saw a doctor or nurse; 17 individuals already had some form of health record and presented it when requested. These records were all from the Lower Rio Grande Valley.

Nursing conferences were held in the mobile clinic. Maternal, child, and adult health were the major topics of discussion. Group teaching was conducted by the nursing staff. They provided information, verbal and written, about adult health practices, infant care, tuberculosis, and immunizations. On a group basis, 2,276 migrants, mostly women and children, were counseled.

Individual counseling was given to the migrants requiring such help. By specific problems the number of persons counseled included these categories: tuberculosis, 186; maternal health, 230; child health, 145; dental health, 20; immunizations of all types, 744.

Educational efforts of the nursing staff, in addition to those conducted with the migrants, included contacts with the ginners and interested community organizations. Personal conversations were held with 54 gin owners and workers. The Migrant Health Program was explained and permission was obtained to work in the camps. The services available to the migrant laborers were detailed and ginners were asked to encourage migrants to avail themselves of these services.

Sanitation Services

Sanitation services for the Migrant Health Program were provided by a full-time sanitarian under the supervision of the Lubbock City-County Health Department's public health engineer. The services of the regular department staff sanitarians were available for necessary consultation and assistance.

In Lubbock County there are no laws or regulations that protect the domestic farm laborer. In evaluating the existing housing and sanitation facilities the criteria established for application to the Mexican nationals under Public Law 78 were used as a guide. The law was not enforceable.

A location housing five or more workers is defined as a camp. The major camps located at the gin sites have been a primary focus of attention thus far. There were 22 such camps varying from 8 to 60 rooms in a camp. Families occupied single rooms. There were no single males or Mexican nationals in these camps. One hundred thirty-three visits were made to locate camps, 19

TABLE 2.—*Migrant Laborers Receiving Immunizations and Skin Tests, Oct. 1-Dec. 31, 1963, Lubbock County, Texas.*

Age	DPT			DT			Smallpox			Polio			Tuberculosis Skin Tests		
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T
0-15	105	104	209	38	52	90	48	44	92	138	159	297	400	549	949
16-49								1	1	3	48	51	2	44	46
50 & Over											4	4		3	3
Total	105	104	209	38	52	90	48	45	93	141	211	352	402	596	998

M=Male. F=Female. T=Total.

visits to obtain permission for conducting a survey, and 38 visits to build good public relations. Twenty-two camps were visited during the operating period. There have been no standards established for domestic farm labor housing in Lubbock County. A newly constructed camp did meet the standards as provided under Public Law 78.

None of these defects have been corrected. The sanitarian has discussed defects and possible corrections with the camp managers. Plans are being considered to bring defects to the attention of such agricultural interests as the Ginners' Association and the Farm Bureau.

A complete survey of migrant housing locations has not been completed. There are 2,570 cotton farms located in Lubbock County. Types of housing, other than camps, used by migrant laborers in Lubbock County included small houses and barracks of two rooms or less located on farms and in towns.

Visits were made to 66 locations with a total of 256 housing units. It was estimated that 8 to 15 individuals were housed in each of these units. Permission was obtained for conducting a survey of sanitation facilities in these locations. Survey work on farm facilities is incomplete and will continue until all locations have been charted.

Working relationships were established with the Migrant Ministry, the Lubbock County agent, the South Plains Ginners' Association, Plains Cotton Growers' Association, Texas Employment Commission, and the local Board of Health. These relationships were developed for the purpose of explaining the Migrant Health Program and to seek information that would be of help to the program staff.

TABLE 3.—*Defects in Sanitation Services of Migrant Labor Camps, Oct. 1, 1963-March 1, 1964, Lubbock County, Texas.*

Type of Defect	No. of Defects
Camp area	8
Water supply	14
Excreta and liquid waste	14
Shelter	35
Washrooms, bathrooms, laundry tubs	42
Cooking and eating facilities	60
Operation and maintenance	54

Health Education

Health education activities were initially carried on by the Lubbock City-County Health Department health educator who was engaged in part-time (25 percent) activities in the Migrant Health Program. Volunteers from the United Council of Churches and a paid church worker were also involved in health education activities. A full-time public health educator was employed by the program in March, 1964.

Educational activities were conducted among the migrants on an individual and a group basis. Approximately 60 migrants were individually counseled. Group counseling included mothers, children, adolescents, and mixed groups.

The purpose of educational activities among the migrants was to improve their existing health conditions. The importance of education for the children and its relation to an improved way of life and prevention of disease was stressed. Group sessions held with the migrants were concerned primarily with personal hygiene and child care. Audiovisual aids and demonstrations were used as educational methods.

The local health educator acted as a consultant to the volunteers from the United Council of Churches. Instruction was given concerning birth certificates and immunization records. Audiovisual materials were provided for use in the volunteers' educational programs. The health educator assisted in setting up a library for the migrants.

Effectiveness of Program

Certain problems hindered the effectiveness of services and educational efforts.

Nursing services were not readily available to the men, who were often at work when the mobile clinic was at the camp. The people were not in the area for a long enough time to obtain the full benefit of nursing services. Transportation to local medical facilities was a barrier to the effectiveness of the Migrant Health Program. The language barrier hindered the nursing personnel only slightly.

The major factors regarding improvements in sanitation have been a disregard and lack of responsibility for the care and maintenance of the existing facilities by the migrant laborers. The sanitarian has determined that camp operators would be more disposed to improve their facilities if a comprehensive education program could be established among the migrants.

Poverty and poor money management, lack of education, skills, training, and an understanding of citizenship rights are barriers to educational efforts among the migrants.

Local prejudice against this minority group was detrimental to total program effectiveness.

Few of these barriers were overcome in the first operating months, but considerable interest has been generated concerning the Migrant Health Program. Among the migrants, initial suspicion has been replaced by confidence and trust in the health department personnel. Community attitudes have improved with an increased awareness and interest in the migrant laborer.

Certain factors contributed to the effectiveness of the total Migrant Health Program. The mobile clinic was essential to the provision of the nursing services. Cooperation of the local health department personnel and the possibility of referral to specialized clinics in the department was helpful. The local medical society supported the program and some physicians were willing to accept referrals. Patients were charged according to ability to pay.

The interest, enthusiasm, and cooperation of the local growers and ginners also contributed to the program's effectiveness.

The acceptance of the program among the migrant laborers was the factor contributing most to the effectiveness of the Migrant Health Program. The group was in need of help and was appreciative when it was provided. If they understood instructions they appeared to follow them well. The migrants are proud, modest, hard-working people. They want to help themselves and among the group will help one another. Trust in the Migrant Health Program personnel has been established. This should contribute to future program effectiveness.

Dr. David Cowgill is director of the Lubbock City-County Health Department.*

Evaluation of Program

Nursing services were highly acceptable to the migrants who made use of the mobile clinic. Many of the laborers stated that they would like to stay in the Lubbock area. Expediency, however, demanded that they continue to migrate when the gins closed. The people often asked if similar services would be available elsewhere. Migrants increasingly have understood and accepted responsibility for improvement of their general and personal health. They took advantage of local health services when they could arrange means to get these services. Migrants seemed to realize their financial responsibility toward their families. They often paid willingly for their own doctor bills and medicines without asking for financial assistance. Migrant families made their own arrangements for maternity care.

Summary

The migrant labor program was established under a grant from the United States Public Health Service including nursing services, sanitation services, and public health education to the 6,000 to 20,000 migrant laborers who come to Lubbock County yearly. A mobile clinic was improvised for the use of the nurses. Visits were made to the migrant camps and schools regularly for con-

*Dr. Cowgill originally presented this paper as a talk before the Conference of City and County Health Officers held during the TMA Annual Session in April, 1964.

sultation, immunization, and referrals to clinics of the health department or local physicians. Approximately 4,000 migrants were seen by the nurses.

A survey of the migrant camps at the cotton gins and a partial survey of the camps on the farms showed that most of the camps had very poor sanitation. Since there are no laws regulating migrant housing, any improvements must be accomplished by education of both the migrants and camp operators.

A public health educator has been employed to study the migrant situation and devise methods for public health education of the migrant workers.

Each year mechanization of agricultural labor increases, but migrant labor will probably be needed in the Lubbock area for a long time to come, although in decreasing numbers. Then, too, each year laborers leave the migrant stream and remain in Lubbock where they have obtained permanent employment.

Acting on these considerations it is the opinion of the Migrant Health Program personnel that this program will be necessary for a number of years.

Dr. Cowgill, Director, Lubbock City-County Health Department, Box 998, Lubbock 79408.

One Out of 21 Is An Alcoholic

Statistically speaking, every 21st adult in the U.S. is an alcoholic by medical definition, says the director of the Alcohol Problems Association of Seattle.

Herbert H. Hill divides alcoholics into four classifications: (1) catastrophe drinkers, who react to some cataclysmic experience; (2) pressure drinkers, who have a pre-existent problem; (3) sensitivity drinkers, who react to increased pressures of life; and (4) those who have personality deficiencies and who are social misfits.

Pointing out the difference between the "social drinker" and the "reward drinker," Hill explains, "the 'social drinker' is one who drinks only very small amounts, seldom drinks except on social occasions . . . and tolerates well the mild euphoria and easing of tensions associated with his drinking.

"The 'reward drinker' tends to keep up with, or even a bit ahead of the crowd in his drinking, finds drinking heightens his pleasure at social events, prefers occasions where alcoholic refreshments are served, and enjoys a marked release from tension as a result of drinking," he said.

"I think too much is made of the 'escape value' of drinking in the life of the problem drinker and the alcoholic, and too little is made of the 'reward potential' of alcohol for the social drinker," says Hill. "I believe it would be more effective and more understandable to the average social drinker . . . if he were helped to understand that alcoholism does not occur because an individual seeks dramatic 'escape from reality' in drunkenness."

"Rather it is because the nature of alcohol in its effect upon the central nervous system is such as to create illusions of competence, security, and skill, which lead one in the face of insecurity and tension to return unwittingly to the illusion until the capacity for reality is diminished and the appeal of 'escape' is irresistible."

Prevention, Recognition, and Treatment of Heat Disorders

REVIEW ARTICLES

ILLNESS RESULTING from exposure to the various types of hot environments is a continuing and severe problem. Although heat disorders may result from inadequately conditioned heat dissipating mechanisms, they as often occur from a severe or acute heat load when environmental conditions do not permit maximum heat dissipation. A usable understanding of the mechanisms concerned with heat dissipation, and the metabolic consequences of heat loss, are essential to the prevention, prompt recognition, and appropriate therapy of the various heat disorders.

The following review summarizes practical, usable information concerning heat production and dissipation, furnishes a guide to preventive control of heat disorders in common "heat stress" situations, and outlines the physiology of heat disorders which formulates the foundation for recognition and therapy.

Mechanism of Heat Dissipation In Hot Environments

In temperate climates the balance of heat production with heat dissipation is easily achieved through radiation, conduction, convection, and sweating (insensible). Radiation of heat to cooler objects accounts for dissipation of over 60 percent of the daily total of heat Calories produced (Fig. 1). Invisible sweating accounts for approximately 30 percent.

As the ambient temperature approaches the normal skin temperature of 92 F., radiation of body heat to surrounding objects ceases and heat dissipation depends principally upon sweating. Sweating dissipates heat by the vaporization of water. The vaporization of 1 cc. of sweat requires .58 Calories (1.72 cc. of sweat liberates one Calorie). The optimal climatic conditions for vaporization are low relative humidity, and removal of moisture from the body surface by air current rapid enough to prevent saturation of the air by the moisture from the skin surface.

When the process of vaporization is impeded by high humidity, body insulation, or practical cessation of air movement, dripping sweat results. The caloric dissipation under these conditions is greatly reduced, yet the tremendous loss of body fluid continues and is even increased under these circumstances. Thus, the most highly developed heat dissipating mechanisms cannot function, and heat storage ensues.[27]

Sweat is more efficiently and readily produced after repeated exposures to a given hot environment. This process-acclimatization is complex and incompletely understood. It is characterized by an increased quantity of sweat production, a decrease in sodium content from 50-75 mEq. to between 10-25 mEq. per liter,[6] sweat beginning at a lower

NORMAL HEAT DISSIPATION

	HEAT CALORIES	PER CENT
RADIATION, CONDUCTION, CONVECTION	1950	65
SWEATING (evaporation)	900	30
WARMING AIR (and excretion of CO_2)	90	3
URINE AND FECES	60	2

Fig. 1.

When the body generates more heat than it can dissipate within a reasonable time, illness results. Accurate evaluation of physical condition relative to the proposed activity, and to weather conditions, are important factors in heat dissemination potentials. These mechanisms are outlined, along with preventive measures and treatment.

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skin temperature[37] (the gradient between internal and skin temperature is decreased),[4] and a significant reduction in the strain on the cardiovascular system to deliver heat to the skin surface. Although simple exposure increases heat tolerance,[12,25,26] complete acclimatization is attained only by work in a hot environment.[23,28,36] King and Barry[20] have demonstrated that maximum acclimatization is attained in workers when the internal body temperature rises to and is maintained at 101 F. during periods of exertion.

The cardiovascular system assumes a key role in acclimatization by increasing blood flow to the skin (12 percent increase). If heat production continues to exceed loss, excessive heat storage results and cardiac output progressively increases (60 percent above normal) until the heart fails.[22] The high-output cardiac failure results in a rise in venous pressure which can be directly correlated with the decreasing capacity to dissipate heat.[15] The maximum utilization of a rapid circulation to enhance heat dissipation is obviously impaired by relatively small deficits of circulating fluid volume.

The important relation between heat dissipation and sweat losses is shown in Fig. 2. A man walking in a 100 F. dry environment generates about 300 Calories per hour and absorbs 150 Calories per hour, which requires one liter of sweat to dissipate the heat under these environmental conditions. If relative humidity is increased to 60 percent, only 750 cc. can be evaporated in the allotted time, although the total sweat output doubles. Unless atmospheric conditions change or the subject is removed to a cooler environment, body temperature continues to rise.

The continued effectiveness of conditioned heat dissipation depends upon the maintenance of normal fluid volumes and the severity of heat stress per unit time. The work of Adolph[1] indicates that sweating efficiency decreases when fluid volume deficits of greater than one liter are incurred. Extremes of heat stress such as maximum physical activity not only produce fluid deficits of this magnitude in as little as 20 minutes, but are accompanied by acute rises in internal temperature above that predicted for maximal heat dissipation (102 F. rectal).[7,14]

The most devastating result in terms of body economy is the tremendous loss of fluid volume which accompanies the dissipation of heat. Since sweat losses are predominately water (sodium content 10-25 mEq. per liter) the initial losses from the extracellular fluid compartment result in an increase of serum sodium concentration above normal; water moves from the interstitial compartment to reduce the total osmolarity of the extracellular fluid toward that of the intercellular compartment. If the rate of water loss is slow enough to allow the movement of intercellular water into the extracellular space, then severe deficits of total body water occur prior to significant heat retention or to collapse from extracellular fluid volume deficit. Thus eight or more liters may be lost (16-18 lb.) before symptoms appear. Collapse may occur with a deficit of two liters if the rate of loss is so rapid that there is not time for transfusion of the extracellular fluid by intercellular water. The sweat rate of the running man in Fig. 2 would lead to collapse if continued for the full hour.

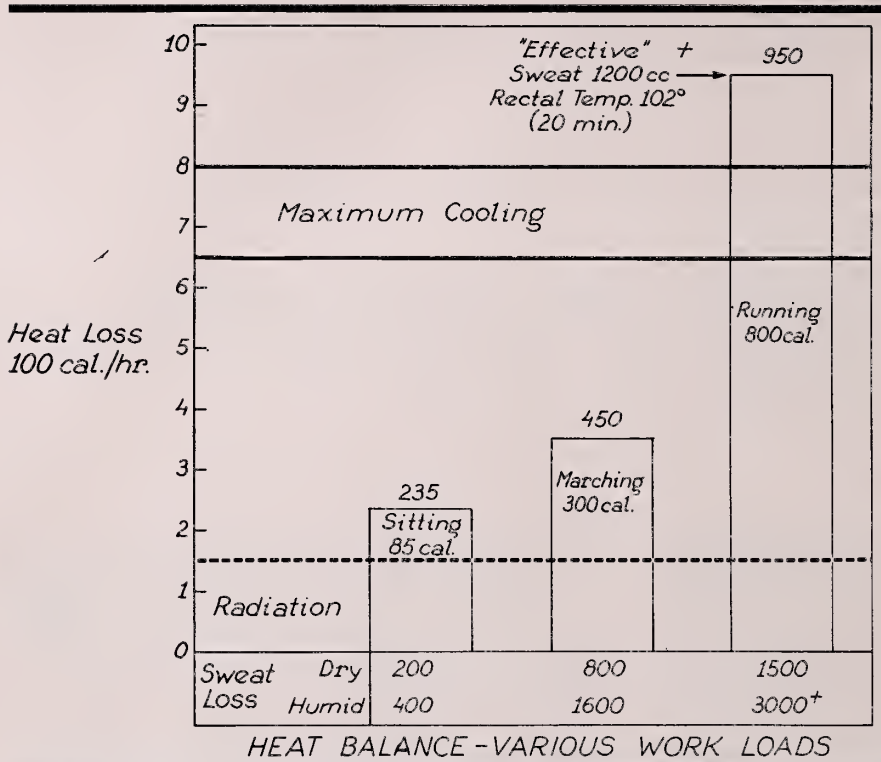

Fig. 2. Illustration shows the relation between work load-sweat rate-humidity on the effectiveness of sweating. Running exceeds "maximum cooling power" of the fully acclimatized man.

Prevention of Heat Disorders

The prevention of heat illness depends upon the practical application of the physical and physiologic principles governing heat dissipation in groups or in environmental situations where the incidence of heat disorders is increased. Control measures, specifically adopted to a particular situation, have successfully reduced the incidence of heat disorders in susceptible groups. These groups can be broadly categorized according to the type of heat loading.

Sustained Work, Normal Individuals.— Heat disorders are noted more commonly among normal individuals exposed to work in the heat under regimented conditions. The bulk of these disorders will occur during the early days or weeks of the exposure and are the result, in part, of lack of acclimatization. The average time required for complete acclimatization is between ten days and three weeks.[27] However, as many as 15 percent of normal individuals may require several weeks to acquire full heat tolerance.[10]

The frequency of heat disorders in military training camps is much greater than those in civilian work situations. Although there are many differences, the prime factor is the inability of the regimented person to stop work when symptoms occur.[24] Further, adequate salt and water replacement often is not possible under field conditions.

A very successful preventive program has been developed by the armed services[31, 35] for repetitive or prolonged exposures. It has resulted in a decrease in heat disorders from 12 percent to less than 4 percent, and in a reduction in the number of deaths when heat disorders do occur. The program defines the activity allowable based upon a "temperature index" which integrates the ability to dissipate heat under different atmospheric conditions. The temperature index is formulated from a simple addition of the readings of a dry bulb thermometer, a wet bulb thermometer (humidity), and one placed in a black copper sphere six inches in diameter (absorbed radiation). This wet bulb globe temperature index (WBGT) is .7 the wet bulb temperature, .2 the black globe temperature, and .1 the dry bulb temperature. An example of the calculation is shown in Fig. 3. When the temperature index reaches 88 F. all strenuous physical activity outdoors is curtailed. When the temperature index is above 92 F., all physical activity in the heat is stopped. This simple calculation can be utilized profitably in civilian situations as a warning guide to limit the work pace and to

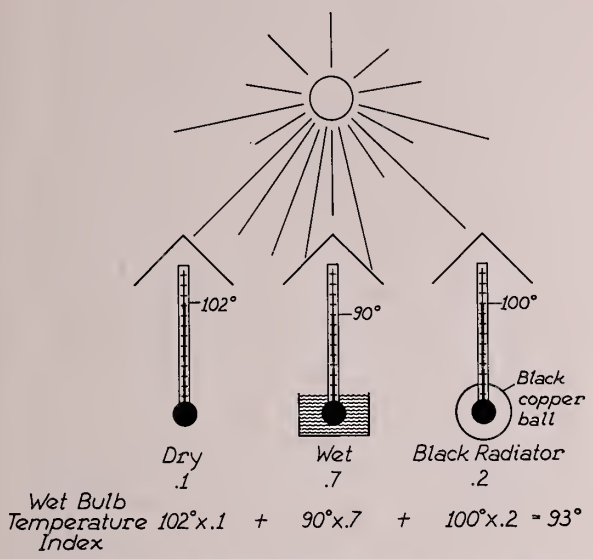

Fig. 3. Illustration shows how to calculate the Wet Bulb Globe Temperature Index. All measurements made in the shade.

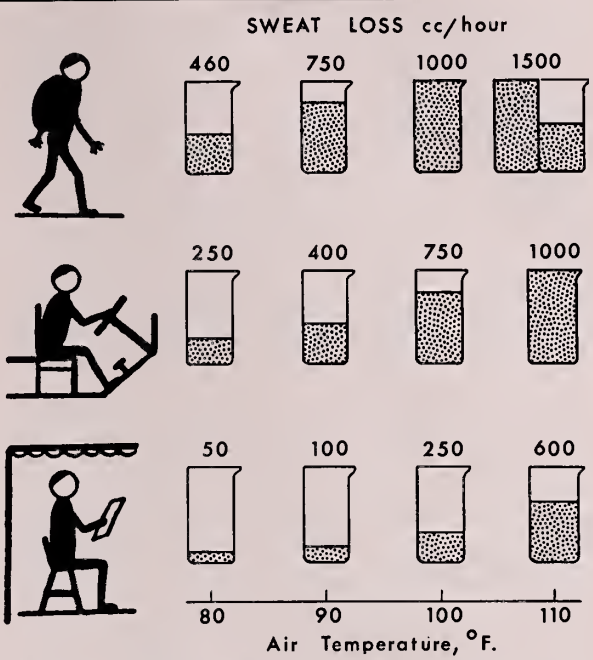

Fig. 4. Sweat losses at dry ambient temperatures for sitting in shade (bottom), light work in the sun (middle), and marching (top). [Adapted from Adolph.[1]]

punctuate it with frequent rest breaks (every 20 to 30 minutes).

Salt and water must be supplied in accordance with at least a rough estimation of losses. A mean estimate of sodium losses after acclimatization is 15 mEq. per liter of sweat.[24] The sweating rate can be determined roughly by the measurements of Adolph of losses at various dry temperatures commensurate with the activity involved (Fig. 4). The calculated amount of both salt* and water should be provided at break periods. Enforced salt tablet administration without water replacement may result in extreme hypernatremia[2, 3] (serum sodium above 160 mEq. per liter).

The basic rules of any program will not allow for individuals who are slow to acclimatize, have febrile illnesses, or are obese, nor for disability of physical dissipation between exposures to heat. The screening of these individuals and group education will further reduce heat disorders.

Acute Heat Loads, Athletics.—An extremely high rate of metabolic activity will temporarily exceed the capacity of well developed heat dissipating mechanisms even with ideal environmental conditions.[5, 26] Most heat disorders occur in the early spring and late summer when high humidity (above 60 percent) is combined with high temperature. This environment is often present during late track season and in spring and early fall football training periods. An adequate program to decrease heat disorders can be outlined as follows.

1. Preseason Conditioning. An athlete should become acclimated to heat by exposure for at least three weeks prior to the first practice session. The use of the six-minute mile adequately prepares most athletes for the season. During the three weeks of acclimatization, a gradual accomplishment of "the mile" is gained through running 220 yards, then walking 220, gradually increasing to 440, then to 880, and so forth, until a running mile is accomplished just prior to beginning practice. During acclimatization, two or three salt tablets and one quart of water should replace each two pounds lost during the exercise interval.

2. Instruction of the Athlete. The detrimental effect of fatigue, dissipation, obesity, intercurrent illness, and the use of drugs (see section on impaired heat loss) should be thoroughly explained.

*Salt tablets are marketed in 365, 500, and 650 mg. tablets (5.8, 9.5, and 12 mEq.), and 1.0 gm. and 1.5 gm. enteric coated tablets. All "salt tablets" referred to in this article are 500 mg. (9.5 mEq.) tablets.

3. Workout Procedure. The two-a-day workout should be scheduled for early morning and late afternoon. The weight and temperature are recorded prior to the morning workout and again before the afternoon workout. If a weight decrease greater than 2.5 lbs. is present, or the temperature is still elevated from morning practice, additional heat exposure should not be allowed. Incipient extracellular fluid volume deficits (often requiring two to four days to become serious) are prevented.

The practice sessions themselves should be broken by five- or ten-minute breaks every 30 minutes. During the breaks, jerseys should be elevated, helmets removed, and shade and wind sought to obtain maximum heat loss for each player. If enough evaporation is not obtained to dry the undershirt, it should be changed prior to continuation. Where these conditions are not possible to obtain, a helmet and jersey filled with crushed ice will rapidly dissipate accumulated heat.

Water should be allowed in small quantities (one or two cups) since the withholding of water has no physiologic basis. Large quantities may produce vomiting. The quantity of salt tablets consumed in a day will vary with individuals and will vary with dietary intake. The number recommended should be gauged from the weight losses incurred by the individual in a practice session, roughly two 500 mg. tablets per liter of sweat loss will suffice after acclimatization. A high salt intake without adequate water is a most dangerous situation.

A rough guide to the limitations imposed by evaluation of environmental conditions is as follows.[33] With temperatures under 90 F. and humidity above 60 percent the more susceptible individuals (for example the obese or poorly conditioned) will be predisposed to heat disorders. At a 90 F. temperature and high humidity, all available preventive precautions should be taken. At dry temperatures above 100 F. or between 90-100 F. with high humidity, practice sessions should be postponed until evening or conducted in shorts and T-shirts.

Since the spring and early fall are the periods when excessive heat with high humidity exist, heat disorders are prone to occur. They tend to occur late in the first week of the two-a-day practice schedule, since subclinical deficits of salt and water depletion may gradually develop. Seldom are heat disorders a problem after the first two to three weeks.

Impaired Heat Loss, Medical Illness.—Conditions which impose limitations on the expected physiologic adjustment to a hot environment are likely to be associated with heat disorders. Since the cardiovascular system is so importantly involved in the mechanism of heat dissipation, diseases of the heart or circulation are frequently associated with heat disorders. Inhibition of cutaneous vasodilatation and/or sweat glands activity by interference with sympathetic activity, whether disease or drug induced, may increase heat storage. Rarely, the absence of sweat glands or skin disease may be the cause.

The highest incidence is seen in cardiac patients who are unable to increase the cardiac output in response to heat. A history of cardiac failure, angina, or severe hypertension connote an inability to increase the cardiac output. Atherosclerosis and arteriosclerosis interfere with the necessary selective cutaneous vasodilatation, preventing an adequate compensatory increase in blood flow to the skin. This is frequently observed in acute alcoholism, with the use of vasodilators, and with large doses of anticholinergic drugs. Persons with severe diabetes are prone to heat retention, perhaps as a result of the destruction of the sympathetic nervous system as well as the accompanying vascular disease. Artane limits this response in Parkinson's disease. Vasomotor and cardiac stimulants such as Benzedrine, Dexedrine, and excessive quantities of coffee and tea may lead to difficulty in healthy individuals.

Endogenous heat loads, such as those occurring in thyrotoxicosis and infections accompanied by high fever, predispose to heat disorders particularly with increasing age. Since aging per se is associated with heat storage,[16,18] fever may be sufficient to precipitate heat stroke.

Recognition and Treatment

A satisfactory classification of heat illness is based upon observations of the clinical syndromes. The common denominators for all these syndromes are exposure to heat, fluid deficits, and heat storage. Malfunction of heat dissipation, though probably present to some extent, cannot be shown in all cases. The important cardiovascular response to

heated blood is pathologic only when venous pressure rises.[8,15]

Of greatest clinical importance are the deficiencies of extracellular fluid volume inevitably present in all forms of heat disorder. Variations of the classical syndromes usually can be traced to improper replacements of these deficits. The concept that various clinical syndromes are interrelated is supported by the fact that if minor disorders are not recognized and corrected, they may lead to heat stroke on re-exposure to heat.

Heat Cramps.—The least severe of the heat disorders is heat cramps. Prolonged or acute excessive losses of sweat replaced by water alone results in this disorder. The illness is characterized by slow, powerful, and spontaneous muscle contractions occurring at irregular intervals. The muscle groups principally affected are usually those involved in the work performance. Manual laborers are prone to have cramps in the upper arm and abdominal musculature, while the back and thigh muscles are more frequently involved in athletes. The patient appears acutely ill, hyperirritable, and mildly agitated. The body temperature is between 100 F. and 102 F., and is accompanied by a mild tachycardia and normal blood pressure. The skin is flushed, and sweating is usually present. Hemoconcentration and serum hyponatremia are characteristic laboratory findings.

The treatment of heat cramps is basically to replenish the extracellular fluid volume deficit with the rapid administration of saline. The serum hyponatremia (serum sodium of 120 mEq. per liter will rapidly correct toward normal when volume is adequately replaced by two to four liters of normal saline, Na=155 mEq. per liter). An occasional patient will not be relieved completely by volume replacement. These cases probably are complicated by a decrease in the ionic calcium of the blood resulting from an accompanying hyperventilation of the apprehensive patient.[19] Since unexplained elevated serum calcium levels occasionally have been observed,[2a] the intravenous administration of calcium gluconate should be limited to one or two grams, given with great caution. Sedation may be safely given only after fluid replacement.

Following the initial treatment, the individual should be removed from a hot environment for a minimum of three days. The completeness of volume replacement is determined by the return to previous normal body weight and a return of serum sodium to normal. Oral salt and water may be necessary to complete the resuscitation.

Heat Exhaustion.—A more serious disorder occurs when large volumes of relatively isotonic fluids are lost during heat exposure. This type of heat disorder is therefore most frequently seen during acclimatization when the sodium content of sweat is extremely high and the volume of sweat is excessive. The nearly isotonic loss from the extracellular fluid prohibits the movement of water from the intercellular space to partially replenish the losses, and hypovolemia rapidly results. Heat collapse is usually preceded by promontory signs of headache, giddiness, loss of mental acuity, weakness, and nausea (orthostatic hypotension). It occurs most often during work with a high motivation, such as athletics, where the individual does not stop when these symptoms appear. Impaired judgment may also prevent this vital limitation.

The usual signs are those of hypovolemia with tachycardia and hypotension. Sweating has usually stopped and the skin may be clammy. The body temperature is near normal since a cardinal sign of extracellular fluid volume deficit is hypothermia. Ordinarily the temperatures will range between 98 F. and 100 F. As volume is replenished with isotonic solutions, the temperature often rises as high as 103 F. transiently before beginning a rather rapid decline. Laboratory findings of hemoconcentration and a normal serum sodium are usually found.

Treatment should consist of sufficient quantity of isotonic balanced salt solutions to return not only the circulatory system but also the urine output to normal. The patient again should be removed from the hot environment which precipitated this deficit, and given supplementary oral salt and water daily until the previously normal weight is obtained. This disorder is of such severity that inactivity in a cool environment should be maintained for approximately one week.

Heatstroke.—The catastrophic event of heat disorders is heatstroke. It has been recognized for ten years that the classical criteria of diagnosis—namely a temperature greater than 106 F. and cessation of sweating—are not necessary for the diagnosis of heatstroke. The criteria of Austin and Berry[2] serve as excellent guides for the diagnosis:

(1) history of exposure to heat, (2) rectal temperature of 104 F. or above, (3) exhibition of varied signs of central nervous system involvement, and (4) a hot, usually dry, and red skin. These criteria recognize the facts that a rectal temperature may not reflect the core temperature of the body, that the cessation of sweating is not necessary for the diagnosis, and that immediate lowering of temperature is mandatory to prevent visceral damage. Brain damage has always been appropriately emphasized,[30] but other vital organs such as the kidneys and liver may frequently suffer irreparable damage.

Heatstroke seldom occurs without warning signs of inappropriate behavior and confusion.[13] An interesting variant has been observed in healthy individuals who develop heatstroke following one or more days of hypernatremia, usually induced by salt tablet administration without sufficient water.[3] Inappropriate behavior, periodic confusion, and odd complaints of knife-like pain behind the eyes or in the ear are typical. A heatstroke victim is usually lethargic, in coma, or may present with convulsions only. These central nervous system signs are generally associated with forceful expiratory excursions and a grunt simulating a Valsalva maneuver.

Evaluation of body temperature even as taken per rectum may not reflect stored internal heat. Internal temperature of the body may be considerably higher than rectal temperature because of changes in blood flow to this area.[34]

The skin of the heatstroke victim is usually dry, reddened, or purpuric.[40] Splotchy cyanosis may be present. When frank shock is present the color may be ashen (gray shock) or remain reddened (red shock). In general red shock may still be accompanied by distended veins and moderate hypotension. Gray shock is always accompanied by signs of severe shock, such as collapsed veins, Cheyne-Stokes respirations, disorientation, and severe hypotension.

The essence of treatment of heatstroke lies in rapid lowering of the body temperature. The most effective means is immersion in an ice bath. There is no effective substitute for this rapid method of cooling.[9] Cooling should be stopped when the temperature is between 102 and 103 F. If cooling is continued below this level, the temperature may drift to dangerously low levels, and death from ventricular fibrillation can occur.

Since the mechanism of heatstroke involves failure of ventricular function, rapid intravenous digitalization should be routine. This can be easily performed during the rapid administration of two liters of isotonic crystaloid fluid (Ringer's lactate). The latter is necessary because of the fluid deficits which are routinely present when heatstroke supervenes.[25] Fluid administration should begin while the ice bath is prepared since cooling causes intense vasoconstriction and a cut down then becomes necessary.

The immediate supportive care of the heatstroke victim after cooling, digitalization, and fluid volume replacement is critical. Arterial hypoxia from shock, high protein pulmonary edema, and hemorrhagic pulmonary infarcts are often present.[1,11,13] An open airway for oxygen administration under pressure should be secured. An endotracheal tube should be used if necessary, but tracheostomy avoided if possible because of the thrombocytopenia and fibrinogenopenia. Vomiting and aspiration from gastric dilatation can be prevented by the routine use of a Levin tube.

Less acutely, electrolyte abnormalities may be determined and controlled. Hypokalemia usually occurs and is easily controlled.[21] A low CO_2 combining power more often reflects respiratory alkalosis from central hyperventilation. When hypernatremia is present, rapid administration of 5 percent dextrose in water may produce sufficient glycosuria with the attendant water loss to prevent correction of the hypernatremia. A short period of explosive diarrhea may require a large isotonic fluid volume replacement.

Close observation for renal dysfunction, adrenal failure, liver damage, and clotting abnormalities,[13,17,38] as well as a search for precipitating causes will be rewarding.

Summary

The problem of heat illness is reviewed from the standpoint of normal heat-dissipating mechanisms and the associated limitations on heat dissipation. The role of the cardiovascular system and appreciation of the magnitude of fluid volume losses from excessive sweating are discussed.

The principles of preventive measures under different types of heat loads are reviewed.

The treatment of heat disorders empha-

sizes the vital role of isotonic fluid volume deficiencies present in all heat disorders, and outlines the principles of therapy in each.

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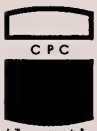
Clinicopathologic Conference

Robert C. Morrison, M.D.; Carl Peterson, Jr.;
Norman M. Kaplan, M.D.; Donald W. Seldin, M.D.; and C. T. Ashworth, M.D.

Clinical Summary



DR. MORRISON: This 59-year-old white male was first seen as an outpatient at Parkland Memorial Hospital in January of 1964. At that time he complained of paroxysmal nocturnal dyspnea of about two months duration and dyspnea on exertion for three weeks. His blood pressure was 200/100. In March, 1963, his blood pressure was found to be 200/64. In 1960 his physician had found his blood pressure to be normal. With a diagnosis of hypertensive cardiovascular disease and left ventricular failure, mercuhydrin and digitalis were administered. Shortly thereafter he was admitted into the hospital for evaluation of his hypertension. Significant in his past history was his feeling of "tiredness" in both hips and legs after walking one-half to one block, which would disappear with rest. He was evaluated elsewhere for this difficulty two years previously, and surgery for "arterial clots" was recommended which he refused. The only other significant complaint was that of intermittent episodes of vertigo during the past several months.

Physical examination revealed a blood pressure of 240/80 in the right arm and 250/70 in the left arm. It was 90/40 in the left leg and 0/0 in the right leg. Funduscopic examination revealed slight elevation of both discs, focal arteriolar narrowing, arteriovenous nicking, occasional small hemorrhages and fresh exudates. A systolic bruit was heard over both carotid arteries. There was venous distension. The lungs were clear. A well defined thrusting point of maximum impulse was felt just outside the midclavicular line in the sixth intercostal space. The aortic second sound was greater than the pulmonic second sound. There were no murmurs. A nontender liver was palpable one fingerbreadth below the costal margin. The spleen was not palpable. Bruits were heard beneath the umbilicus and over both femoral arteries. Both femoral pulses were palpable but the right was stronger than the left. Neither popliteal, dorsalis pedis, nor posterior tibial pulses were palpable.

Laboratory values are shown in Table 1. The electrocardiogram showed inverted T waves and slight ST segment depression in V_5 and V_6. An intravenous pyelogram demonstrated small kidneys bilaterally, with the right smaller than the left. There was delayed excretion of the dye on the right. The cortex of both kidneys was noted to be thin.

Angiotensin infusion was performed and the patient was found to have a 10 mm. diastolic pressor response to 40 micromilligrams per kg. per min. of angiotensin. A translumbar aortagram revealed complete occlusion of the aortic lumen at a point just below the origin of the superior mesenteric artery. It was noted that the left renal artery filled well without evidence of obstruc-

This is the sixth in the 1964-1965 series of clinicopathologic conferences to be provided by the University of Texas Southwestern Medical School at Dallas. Dr. C. T. Ashworth, professor and chairman of the Department of Pathology, is guest editor and also presents the pathologic findings.

Clinical summary is by Dr. Robert C. Morrison of the Department of Medicine; radiologic findings and discussion by Dr. Carl Peterson, Jr., Department of Radiology; and clinical discussions by Drs. Norman M. Kaplan and Donald W. Seldin, Department of Medicine.

A splenorenal anastomosis was performed in this case of a 59-year-old white man with hypertension and "tiredness" in the hips and legs after walking one-half to one block.

tion or post-stenotic dilatation. The orifice of the left renal artery was very near the point of aortic obstruction. It was only faintly opacified and there appeared to be some narrowing near its origin.

A surgical consultant felt that the aortagram showed diffuse narrowing of the left renal artery. It was felt that uremia might be improved by revascularization of the left kidney, and that it might then be possible at a later date to remove the occlusion of the aorta and revascularize the renal artery. For this reason, a left splenorenal shunt was performed. At the time of the operation, the kidney was found to be freely moveable and was smaller than normal, but was larger than the right. The aorta was occluded at about the level of L-2. The left renal artery was found to have a long thick posterior plaque occluding 40 to 50 percent of the vessel. Postoperatively, there was no change in blood pressure. He apparently did well following surgery, although at the time of discharge 11 days later, his blood urea nitrogen was 82 and creatinine 6.6.

He was readmitted three weeks later with complaints of progressive exertional shortness of breath, orthopnea, nervousness, and weakness. Physical examination revealed a pale, nervous white male with moderate respiratory distress. Marked venous distension in the neck was present, and dullness and decreased breath sounds were heard over the base of the left lung. The liver was questionably enlarged to 6 cm. below the right costal margin. One-plus pitting edema of both feet was noted. Admission hematologic studies showed a hemoglobin of 7.9, white blood count 9,000, and urinalysis revealed two-plus albumin and 15-30 white blood count per high-power field. Blood urea nitrogen was 125 and creatinine 10. Serial laboratory values are recorded in the chart. Venous pressure was 26 cm. of water and circulation time 13 seconds. Chest X-ray revealed a left pleural effusion and bilateral hilar infiltrate, more pronounced on the right, believed to be pulmonary congestion or uremic pneumonitis. A thoracentesis was productive of 250 cc. of straw-colored liquid containing 1.8

TABLE 1.—*Chart of Laboratory Findings.*

	1/25/64	2/4	2/17	2/21	3/17	3/19	2/20	3/21
Hemoglobin (gm./100 cc.)	10.3	8.9	10.3	9.0	7.9	7.2	8.2	7.2
WBC (1000s/cu. mm.)	5.7				9.0	8.1	11.2	14.1
BUN (mg./100 cc.)	58.0	54.0	76.0	82.0	125.0	143.0	116.0	82.0
Creatinine (mg./100 cc.)	4.8	4.8		6.6	10.0		9.6	8.4
CO_2 (mEq./L.)		26.0	25.0		22.0		20.0	25.0
Potassium (mEq./L.)	4.0				4.7	7.5	5.0	5.3
Phosphorus (mg./100 cc.)	5.1				10.1			
SGOT (Units)	17.0				10.0	10.0	155.0	
Venous Pressure (cm. water)					26.0	28.0	18.5	

1/25/64—Urinalysis—1+ Albumin, 0-2 WBC, no casts.
Creatinine clearance—87 ml./min.
PSP—15% excretion after one hour.

gm. of protein per 100 cc. The electrocardiogram was essentially the same as during the previous admission.

Initially, an effort was made to control his venous congestive state via assisted respiration and oral diuretics (Naqua). His dyspnea increased, however, and venous pressure continued to be elevated. Chest X-ray showed increasing pulmonary congestion. It was elected to begin peritoneal dialysis with hypertonic glucose in an attempt to reduce pulmonary congestion. The dialysis was complicated by the development of diffuse abdominal tenderness and cloudy return on the second day. Initially, this was thought to be due to nonbacterial peritoneal irritation, for Gram's stain of the centrifuged dialysis return showed many white blood cells but no bacteria. A culture of the second day's dialysate, however, grew a gram-negative organism. Colistin was added to each dialysis bottle. On the night preceding the beginning of peritoneal dialysis, the patient had an episode of substernal chest pain accompanied by nausea and nervousness which lasted 45 minutes. Subsequent electrocardiograms were unchanged, but the serum glutamic oxalopyruvic transaminase was found to be elevated to 155. In the meantime, although he responded chemically to the dialysis with a drop in blood urea nitrogen to 80, clinically he continued to deteriorate. During his last day, he became disoriented and began vomiting dark coffee-ground material. Finally, increasing dyspnea and hypotension ensued, and he expired.

Radiologic Findings

DR. PETERSON: The first available roentgen examination on this patient is from the time of his original admission to this institution on Jan. 21, 1964, and is comprised of posteroanterior and lateral films of the chest. At this time, the heart was borderline enlarged with a slightly left ventricular preponderant pattern and the hilar vascular structures on both sides were grossly engorged. Kerley B lines were present in both lung bases and there was a left-sided pleural effusion. Following digitalization and diuresis, a film obtained on Jan. 21, 1964, showed the heart to have decreased in size and the hilar vascular structures to have returned to a fairly normal appearance. The Kerley lines had disappeared and the left pleural effusion had nearly disappeared as well. On Jan. 21, 1964, an excretory urogram on this patient was rather strikingly abnormal. The right kidney was noted to have appeared fairly normal in size and contour. Excretory function was delayed and concentration of contrast material never approached normal on the right side. All of the findings mentioned were highly suggestive of unilateral renal arterial disease on the right side. Incidental findings on the excretory urogram were localized degenerative changes involving the L-3, L-4 disc interspace with rather marked narrowing at this level, and contrast material retained within the subarachnoid space from a previous myelogram. In the absence of other degenerative disease of the spine the possibility of trauma as an etiological factor for these localized changes would have to be considered.

On Feb. 4, 1964, a translumbar aortagram was performed by the vascular surgery team (Fig. 1). Complete obliteration of the aortic lumen below the level of the celiac axis was demonstrated on this examination. The occlusion did not include the left renal artery ostium and the left renal artery was seen to fill fairly well and its ramifications showed a normal distribution. Slight irregularity of the lumen was noted just distal to its point of origin from the aorta. Early films showed complete absence of filling of the right renal artery, but late films demonstrated filling of a fairly normal appearing artery, apparently by collateral circulation.

No roentgenograms were obtained on this patient, following the aortagram, until his readmission to the hospital on or about

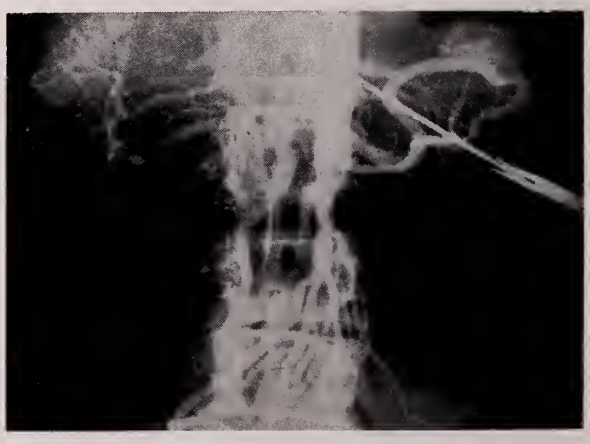


Fig. 1. Aortagram showing obliteration of aortic lumen at level of celiac axis. The renal arteries filled fairly well.

March 14, 1964, at which time chest films showed that his heart had enlarged and there were bilateral pleural effusions. Hilar vasculature was markedly engorged once again. The Kerley lines had returned, as best seen in the right base, and a patch of infiltrate in the right lower lung field was suspect. The pleural effusion on the left side was much larger than on the right. Serial films on March 17, 18, and 21, 1964, showed increasing vascular engorgement and the appearance of a hilar butterfly distribution of alveolar infiltration having the roentgen appearance of "uremic pneumonia" or "uremic pulmonary edema" (Fig. 2). The pleural effusion was larger on March 17 than on March 14, but was decreased on March 18, 1964, apparently following thoracentesis. The preterminal recumbent film on March 21, 1964, showed bilateral diffuse patchy infiltration primarily situated in the central portion of both lung zones. Bilateral pleural effusions were layered posteriorly and added density to both lung fields in this recumbent position.

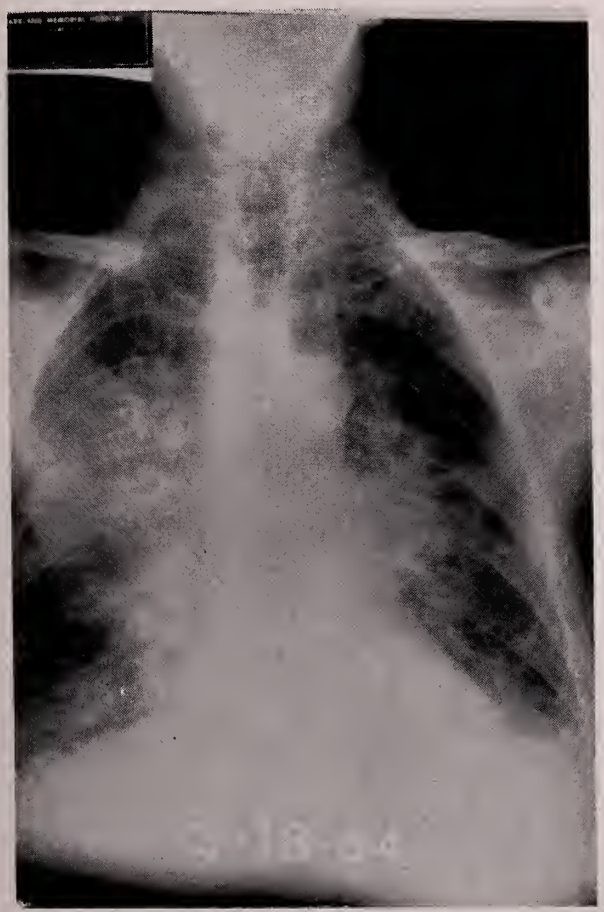


Fig. 2. Anteroposterior view of chest X-ray showing vascular enlargement and hilar butterfly distribution of pulmonary infiltrate suggestive of "uremic pneumonia."

The series of roentgenograms on this patient, as described between Jan. 21 and March 21, 1964, seem to indicate a rather straight-forward series of events related to this patient's severe arteriosclerotic disease and most particularly as it involved progressively severe generalized arteriosclerosis and accompanying hypertension (systolic only), finally involving the right kidney and leading into a rapidly deteriorating picture of "malignant hypertension" and accompanying progressive uremia and death. However, there is one finding that does not fit easily into the crossword puzzle and this is the predominantly left-sided pleural effusion throughout this patient's course. Purely congestive pleural effusions, if unilateral or if predominant on one side more than the other, almost always are on the right side. When faced with a situation such as this, with left-sided pleural effusions, other pathologic entities within the left lung must be entertained. These include (1) tumor, (2) collagen disease, (3) pulmonary infarction, (4) infection, that is, tuberculosis, (5) abdominal or retroperitoneal inflammatory processes such as pancreatitis. It certainly would not surprise me to find that this patient had one of these entities, as well as the previously discussed findings at the time of death.

Clinical Discussion

DR. KAPLAN: The most interesting feature of this patient's course was the evidence of very extensive hypertensive as well as arteriosclerotic disease in the absence of sustained elevation of the diastolic blood pressure. The evidence for severe hypertensive disease was most obvious in the eyegrounds, showing extensive hemorrhages and exudates and questionable papilledema on the initial admission. Other than for one blood pressure recorded as 200/100, all of the other readings taken for some years prior to his admission and throughout his hospitalizations did not show diastolic hypertension.

A number of possible alterations in hemodynamics could have prevented or abated hypertension which had been present previously. First, he may have been hypovolemic, but there was no evidence for this, and in fact there was some evidence of an over-

expanded extracellular fluid volume. Second, in the presence of severe congestive heart failure, the diastolic pressure might fall, but again there was no clinical evidence for this. Third, and most likely, the function of the myocardium as a pump may have been severely impaired by arteriosclerotic disease, leading to a fall in the diastolic pressure.

On the other hand, the patient had extensive arteriosclerotic disease, with carotid bruits, eyeground changes, evidence of renal insufficiency and greatly diminished pulses in the legs. In view of the history of previous trauma to the lower abdomen and the evidence of diminished blood flow to the extremities, the possibilities of occlusive disease of the renal arteries was investigated by an angiotensin infusion test, an intravenous pyelogram and later a translumbar arteriogram. Since the infusion test was done at a time that the patient did not have diastolic hypertension it could not be evaluated in the usual way, because the procedure has been used in the differential diagnosis of patients all of whom had diastolic pressures of 90 or above.[5, 6] Nonetheless, this patient was highly insensitive, more so than any normotensive individual or any patient with renal vascular disease yet studied. This great insensitivity to the pressor action of exogenous angiotensin, in the absence of obvious plasma volume depletion, certainly suggests the presence of high endogenous angiotensin which in turn could reflect renal artery occlusive disease. Since the patient was azotemic, any renal involvement must have been bilateral but the difference in size on the intravenous pyelogram suggested that renal ischemia might be more severe on one side. The arteriogram confirmed this.

The question then arose as to the advisability of surgery on the renal arteries, in hopes of improving renal function by increasing renal blood flow. The group in Houston have described their experience with a number of patients having renal insufficiency related to main renal artery occlusive disease.[7] After surgical repair, most of these patients have shown improved renal function. The degree of renal insufficiency in some of their patients was as severe as that of our patient. Therefore, the vascular surgeons were consulted with the hope that at the same time repair of both the lower aorta and one or both renal arteries could be performed. Since the patient was in such poor condition, they decided to do the quickest procedure which might improve blood flow to the left kidney, a splenorenal shunt, as suggested by Morris and others. If improvement occurred, more definitive repair could then be more safely attempted. However, the patient did not improve after surgery and soon began to deteriorate with accelerating renal insufficiency. No attempt was made to show that the splenorenal shunt was patent, but I doubt if further surgery would have been indicated.

Certainly the initial surgery was indicated in hopes of improving blood flow to one kidney and thereby stopping the progress of renal insufficiency. In retrospect, perhaps more aggressive surgery might have been attempted, such as a definitive repair of the lower aorta with shunts to both renal arteries. But, in view of the patient's precarious status at the time, the splenorenal shunt was probably the best procedure.

His terminal episode may have been related to the progression of his renal impairment possibly associated with thrombosis of the shunt, but the possibility of a myocardial infarction seems quite good, in view of the substernal pain and the elevation of the serum glutamic oxalopyruvic transaminase.

DR. SELDIN: It is an interesting and extraordinary phenomenon to see fundi, such as this patient displayed, in the absence of diastolic hypertension. You will recall that examination of the fundi revealed exudates, hemorrhages, and at least a suggestion of papilledema. There is no reason to suppose that there might be a primary intracerebral cause for this funduscopic picture. We have no neurologic signs nor other evidence pointing to a cerebral lesion. There is every reason to believe, therefore, that the funduscopic picture was a reflection of vascular disease.

It is the prevailing opinion of most investigators that the hypertensive process causes vascular disease in direct proportion to the magnitude of the elevation of blood pressure.[2, 10] The elevation of the blood pressure is regarded as the direct cause of the vascular disease. If this patient really did have vascular necrosis of the hypertensive type without elevation in diastolic pressure for a prolonged period before the vascular lesions developed—as seems to be the case—it would be a significant argument against

the point of view that Byrom and Pickering have propounded, and would argue that there is something in the hypertensive process in addition to elevation of blood pressure that can result in vascular necrosis. You may remember that Muirhead, Grollman, and their associates, in studies on experimental hypertension in the dog, were able to dissociate, in certain preparations, elevation of blood pressure from vascular necrosis.[8] Perera[9] reported six instances in which patients with the accelerated phase of hypertensive vascular disease developed retinopathy during a sustained period of normal blood pressure. These observations suggest that while elevation of diastolic pressure may aggravate vascular disease, there may well be some other factor in the hypertensive process, which can sometimes be dissociated from elevated diastolic blood pressure, that is necessary for the necrosis of the smooth muscle of the vascular wall. We certainly will await the pathologic findings with interest.

A second noteworthy point about etiology of this patient's illness concerns the extraordinarily widespread evidence of arteriosclerosis—in renal vessels, aorta, carotids, and so forth. It is well to remember that disorders amenable to treatment, such as hyperlipemia or hypercholesterolemia, can be responsible for extensive arteriosclerosis. I have seen at least two patients, one of whom was a woman on our service, with essential hypercholesterolemia, who had a long history of vascular disease manifested by coronary insufficiency, intermittent claudication, bilateral renal artery stenosis, and carotid artery stenosis. It seems likely that the arteriosclerosis was the direct consequence of hypercholesterolemia. In the present case plasma cholesterol was normal and the serum was clear, so that we have no reason to attribute the arteriosclerosis to hypercholesterolemia or hyperlipemia.

The final point I want to make concerning the clinical configuration of this patient is the question that was raised by the X-ray department concerning uremic pneumonia. By uremic pneumonia is meant an infiltration process in the lungs which is unique to the uremic process. Uremic pneumonia, according to this point of view, is not heart failure but rather an exudative process attributable to uremia per se. The X-ray picture is striking but by no means diagnostic. The infiltration assumes a butterfly distribution, leaving a peripheral clear zone of emphysema, with the infiltrative process bunched toward the hilum, giving a picture frame pattern. This X-ray picture can occur in any kind of pulmonary edema, but is more frequent and characteristic of the uremic state. In early pathologic descriptions, there were some reports of vascular disease in the pulmonary vessels, but almost all the recent studies deny the presence of a specific pulmonary vascular lesion. Characteristically, the exudate into the alveoli is extremely hemorrhagic. In addition, inflammatory cells, protein, fibrin, and dense hyaline masses are present in the alveoli. The alveolar septa may be infiltrated with edema containing mononuclear cells and fibrinous bands. This picture is not specific for, or unique to, the uremic state. It can be seen in a wide variety of different inflammatory or congestive processes. The key point about the cause of uremic pneumonia is this: will the infiltration recede if successful measures, such as digitalis, salt restriction, diuretics, perhaps dialysis, are taken to cause the delivery of salt and water? The largest series attempting to examine this issue is from Sweden. Alwall and his associates[1] studied 16 cases with uremic pneumonia and found that in all instances if edema is delivered, as evidenced by marked weight loss, the X-ray lesions clear. These investigators therefore argued that the cause of the congestion is essentially the retention of salt and water and blood in lungs as a consequence of left ventricular failure or noncardiac circulatory congestion. In a recent report,[4] clinical evidence on one patient is presented to suggest that uremia per se can cause uremic pneumonia. Dialysis uniformly resulted in clearing of the pulmonary infiltration, but on two of six occasions, the lung lesions disappeared despite a positive net fluid balance. This does not resolve the issue. It is possible, for example, that cardiac function improved as a result of the correction of the uremic state; in consequence, pulmonary congestion might improve despite the failure to deliver edema. The fact that the heart remained dilated after dialysis is strong evidence for an important cardiodynamic derangement. Nevertheless, the observation that the lung lesions clear following a dialysis which corrects the

uremic picture but does not remove fluid suggests that in addition to left ventricular failure and noncardiac circulatory congestion,[3] uremia per se may play a role. The important point to remember, however, is that from a clinical point of view uremic pneumonia should be treated as a form of circulatory congestion, since in most instances delivery of edema will correct the disturbance. Should uremic pneumonia persist or should diuresis not occur, dialysis is indicated.

Finally, I want to reemphasize one point that Dr. Kaplan made previously. Until recently, renal vascular disease has been attacked clinically in order to relieve hypertension. A second indication appears to be emerging: the correction of renal insufficiency or renal failure per se. I believe that if this patient were deteriorating as a consequence of renal failure secondary to vascular occlusion, surgery to relieve the vascular insufficiency must be considered, not so much from the point of view of hypertension as to improve renal function. Striking improvement in renal excretory and regulatory function has been reported following the correction of renal artery stenosis.

Pathologic Findings

DR. ASHWORTH: One group of significant pathologic findings in this case relates to the heart. The heart was moderately enlarged due to left ventricular hypertrophy and weighed 470 gm. In view of the demonstrated systolic hypertension of high degree, even in the absence of a continual elevation of diastolic pressure, and because no other causes for cardiac hypertrophy were discovered at autopsy, it was our view that the cardiac enlargement is the result of hypertensive cardiovascular disease. A subendocardial infarct was found in the posterior-lateral wall of the left ventricle, occupying a major portion of the posterior wall. The necrotic area was clay colored and was surrounded by hyperemic margins, indicating a duration of several days. Histologic study likewise showed coagulative necrosis of the usual type seen in acute myocardial infarction (Fig. 3). Viable but severely injured myocardial cells persisted in a well delineated zone just beneath the endocardium. Reparative changes were minimal, and it is concluded that this infarct was about four to five days in duration, thus corresponding in time to the onset of chest pain several days before death. The coronary arteries revealed extensive atherosclerosis and the right coronary and circumflex branch of the left were almost entirely occluded by atheromas.

The lungs were extremely congested and the combined lung weight was 1850 gm. This marked increase in weight was due to massive pulmonary edema. The lower lobes of both lungs were also the seat of confluent, patchy areas of consolidation. The gross and microscopic findings confirm that this was due to the usual type of hypostatic bronchopneumonia (Fig. 4). We do not find the usual hemorrhagic and interstitial changes that are characteristic of so-called uremic

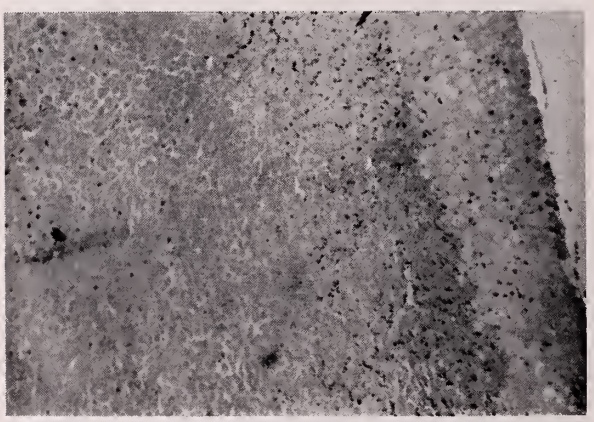


Fig. 3. A lower power photomicrograph of left ventricle of heart showing recent myocardial infarction of the subendocardial zone.

Fig. 4. Low power photomicrograph of lung showing terminal or hypostatic bronchopneumonia. The changes of "uremic pneumonitis" were not seen.

pneumonitis. Accordingly, this pneumonia was considered to be a terminal type rather than uremic pneumonitis.

Acute hemorrhagic erosive esophagitis and numerous acute mucosal erosions of stomach and duodenum were present. These areas of erosion were occupied by black deposits, indicating that hemorrhage from these lesions was active and recent.

Perhaps the most interesting and significant part of the anatomical findings pertain to the aorta, renal arteries, and kidneys. Severe aortic atherosclerosis was found, and it increased and progressed lower in the abdominal aorta and iliacs. Occlusive thrombosis was present in the abdominal aorta beginning at a point just below the superior mesenteric artery and extending as far down as the bifurcation of the aorta (Fig. 5). This thrombus was moderately adherent to the aortic lining. The central portion was friable and unorganized. The renal arteries were moderately involved by atherosclerosis, but they were patent throughout with the exception of a small area of the left renal artery near the site of the splenorenal anastomosis. At this site an atheromatous plaque almost completely occluded the left renal artery for a distance of about 1 cm. This atheromatous plaque was positioned in such a way that it must have interfered with the expected anastomotic flow of blood through the splenorenal anastomosis. The anastomosis itself was intact, but the splenic artery was completely occluded by a thrombus. This showed evidence of having been present for many days, and it probably occurred shortly after the operation. Obviously, this would have rendered ineffective the corrective surgical procedure. Probably related to this thrombosis of the splenic artery was the presence of a large infarct in the spleen which had not been surgically removed. The infarct occupied about three-fourths of the entire spleen and fibrin was present over the serosal surface.

The two kidneys were asymmetrical. The right weighed 80 gm. and the left weighed 110 gm. The right kidney appeared distinctly atrophic and had a very thin cortex. Both

Fig. 5. Gross photograph of portion of abdominal aorta, both kidneys, and the splenorenal anastomosis. Top two arrows reveal the splenic artery to be completely occluded by a thrombus. Severe aortic atherosclerosis and occlusion of the aorta by a thrombus (arrow near center of picture) are seen. The left arrow reveals significant narrowing of the right renal artery by atheroma. The right kidney (left side of photograph) is appreciably larger than the left.

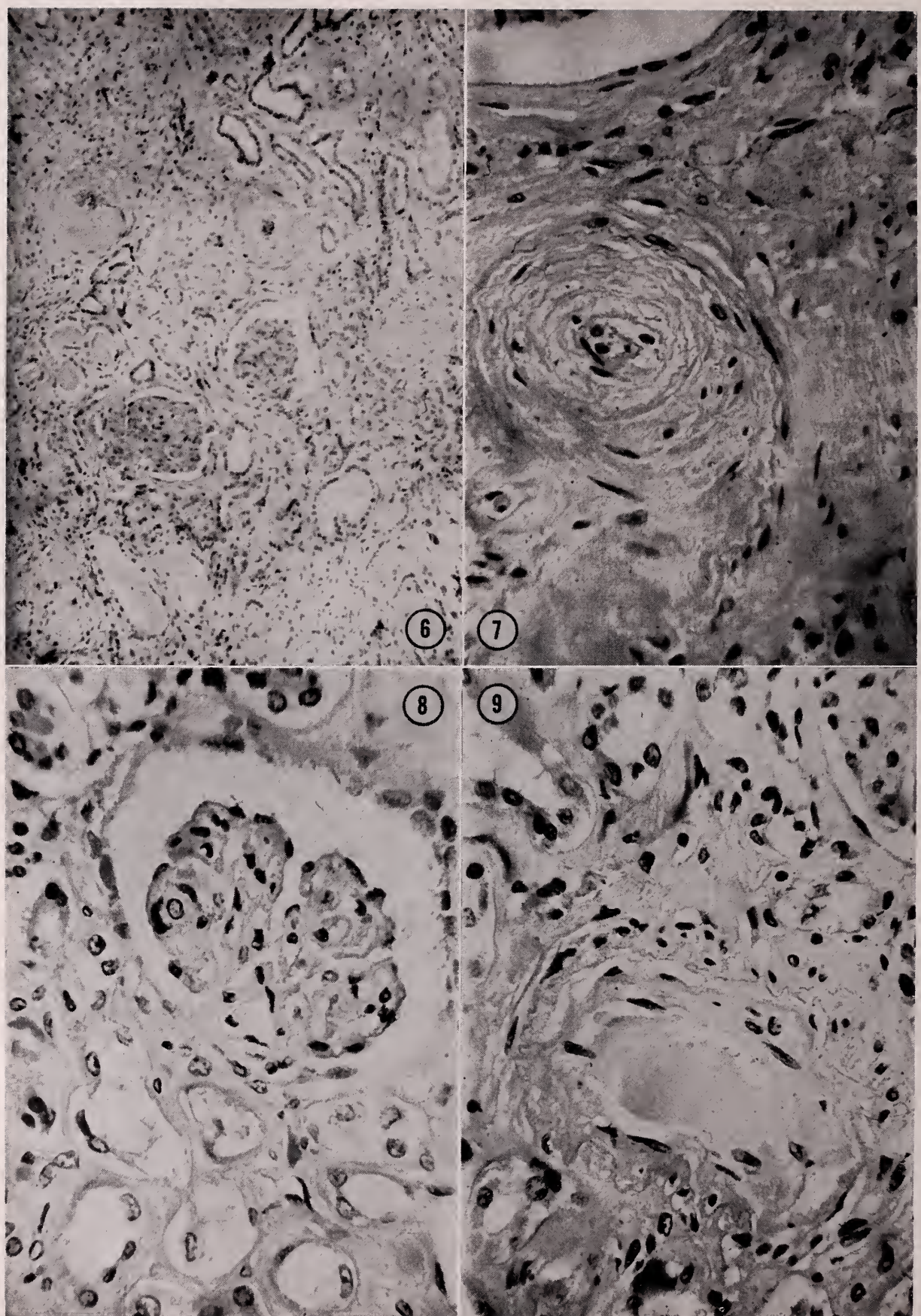

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Fig. 6. Low power photomicrograph of right ("unprotected") kidney which reveals marked arteriolar nephrosclerosis. Greatly thickened afferent arterioles and slight glomerular atrophy can be seen.

Fig. 7. A high power photomicrograph of the right kidney. Much concentric hyperplasia with thickening of an afferent arteriole as occurs in malignant nephrosclerosis can be seen.

Fig. 8. High power photomicrograph of left ("protected") kidney, showing ischemic glomerular sclerosis and peritubular fibrosis.

Fig. 9. A high power photomicrograph of an afferent arteriole in the left kidney. The almost normal thin wall (minimal arteriolar sclerosis) can be seen and is representative of the appearance of arterioles throughout this kidney.

kidneys presented slightly granular, mottled reddish brown external surfaces, and the capsules were adherent. The cortical atrophy was more distinct in the right kidney than in the left. Microscopic study of the kidney reveals some interesting differences also (Figs. 6, 7, 8, and 9). Interstitial fibrosis with tubular atrophy and glomerular fibrosis and atrophy were particularly prominent in the right kidney. Only focal patchy areas of nephrosclerotic fibrotic atrophy were seen in the left kidney. The arterioles of the right kidney showed only mild arteriolar sclerosis. On the other hand, in the left kidney, severe concentric cellular hyperplasia, with marked narrowing of the lumens, was observed in the afferent arterioles and in the interlobular arteries. A few areas of necrotizing arteriolar sclerosis also were found in the left kidney. The left kidney also revealed evidence of acute tubular degeneration of several days duration. In some of the tubules of the collecting duct system and distal convoluted tubules the lining cells were necrotic and had desquamated into the lumens. The lumens also contained red granular casts such as are seen in acute tubular degeneration of the kidney.

The gross and microscopic differences in the two kidneys are of interest in this case. On the basis of these changes it can be concluded that the right kidney was most severely affected by ischemia and over a longer period of time. The ischemia was probably due to the marked narrowing of the ostia of the right renal artery by severe aortic atherosclerosis. Quite possibly, this ischemia and atrophy of the right kidney may have been the initiating factor in the production of this patient's hypertension. On the other hand, preceding essential hypertension complicated later by aortic atherosclerosis cannot be excluded. Whatever the initiating cause of hypertension, as the systemic effects of the accompanying vasoconstriction increased, the left or unprotected kidney underwent the changes of arteriolar nephrosclerosis. The eventual development of malignant hypertension was indicated by the severe cellular hyperplasia of afferent arterioles and the occasional necrotizing changes in affected arterioles. As pointed out by Dr. Seldin, it is remarkable that this patient's diastolic pressure did not usually achieve hypertensive levels. In part this may be explained by rigidity and loss of elasticity in the atheromatous aorta. The findings in this case suggest that diastolic hypertension itself is not the only factor involved in the development of the anatomical and clinical changes of malignant hypertension.

In the posterior portion of the eyeball, vascular changes were relatively slight, but there was a mild degree of arteriolar sclerosis. More significant, retinal edema and small hemorrhages were present in this portion of the eye. These alterations are those of hypertensive retinopathy.

In summary of the anatomical findings and pathogenesis of lesions, this patient had severe generalized atherosclerosis, resulting in thrombosis of the abdominal aorta. Aortic atherosclerosis led to narrowing of the ostia of the renal arteries and renal ischemia. For a time, at least, the right kidney was more affected by the ischemic process. This unilateral renal ischemia might have been the initiating factor in the development of this patient's hypertension, but essential hypertension cannot be excluded. Later, changes of systemic hypertension and malignant nephrosclerosis were superimposed on the unprotected (left) kidney. At the same time, the changes of severe hypertension became manifest, resulting in retinal edema and focal hemorrhages. The terminal event appears to have been that of a myocardial infarction affecting the posterior wall of the left ventricle due to atherosclerosis. The pneumonia present terminally was a terminal hypostatic type and was not directly related to the uremic process.

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Help for Migrant Harvesters

Harvest time is synonymous with autumn in the minds of many people—particularly the migrant laborer.

Over 130,000 Texans will be harvesting the nation's crops from now until the cold bite of winter sends them to their homes in southernmost Texas.

They began their migration early last spring to labor in fields or orchards wherever they could find work. Whole families pack a few things and travel in pickups, trucks or buses following their "crew leader" across the country.

This is the migrant way of life, with its many inherent problems.

For example, the typical migrant farm laborer is functionally illiterate—that is, he never attained more than a third grade education. This problem is perpetuated by the younger generation as the children seldom attend school for more than a six-month period.

His home, where he lives less than six months a year, is normally a small structure with little or no plumbing. On the road, following the crops, he and his family live wherever they can.

The migrant's income averages about $900 a year. His diet, more often than not, consists of beans and tortillas. Milk is an infrequent treat.

The average migrant never sees a doctor during his entire lifetime. *His life expectancy is about 30 years.*

Obviously, health problems are a major concern of the migrant worker and the communities where he goes. Tuberculosis and other diseases may be prevalent and go undetected. Immunization schedules—if any—are erratic, and infant mortality soars.

The Texas State Department of Health has under way a statewide project aimed at bettering health service for the migrants. Separate projects are being conducted by Laredo-Webb County, Southwest Texas Region, and Lubbock City-County and Plainview-Hale County health departments.

NEWS

Texas Surgical Society, Texas Academy Of General Practice To Meet Next Month

TEXAS SURGEONS and Texas general practitioners will hold statewide meetings during October.

The Texas Surgical Society will meet in San Antonio Oct. 5 and 6 at the Granada Hotel, and Corpus Christi will be host to the Texas Academy of General Practice for its 15th annual scientific assembly Oct. 11-14.

San Antonio To Host Texas Surgical Society

On the Texas Surgical Society program for Monday, Oct. 5, are the following subjects and speakers: "Experiences with Massive Rectal Prolapse," Dr. Ernest J. Gregory, Jr., San Antonio; "How to Treat Fractures Without Really Trying," Dr. John H. Sewell, Fort Worth; "The Surgeon and His Investments," Dr. Sam G. Dunn, Lubbock; "The Surgical Techniques of Temporal Lobectomy for Epilepsy," Dr. A. Earl Walker, Johns Hopkins Hospital, Baltimore, Md.; "Use of the Electrosurgical Unit in Radical Mastectomy," Dr. Paul L. Gorsuch, San Antonio; "The Implantation of Low Intensity Radium Needles in the Supra Clavicular and Intercostal Spaces as an Adjunct in the Treatment of Carcinoma of the Breast," Drs. Joseph P. McNeill, John V. Goode, and James A. Martin, all of Dallas; "Vena Cava Interruption: Ligation or Plication?," Drs. Jesse E. Thompson, Dale J. Austin, R. Don Patman, and C. G. Wheeler, all of Dallas; "Surgical Treatment of Complications of Esophageal Hiatal Hernia," Dr. G. Valter Brindley, Jr., Temple; "Gastrointestinal Bleeding of Appendiceal Origin," Dr. R. Wilson Crosthwait, Waco; "Pseudoatresia of the Sigmoid in the Newborn," Dr. N. J. Bellegie, Waco; "Complete One Stage Colostomy in the Presence of Obstruction," Drs. J. Peyton Barnes, Houston, and Nihat Tasdermiroglu; and "Biliary Tract Endoscopy," Dr. Henry G. Glass, Houston.

On Tuesday, Oct. 6, the program includes the following: "Diverticulitis and Carcinoma of the Colon," Dr. Robert J. Rowe, Dallas; "Irradiation Injuries of the Colon," Dr. Augustus F. Herff, Jr., San Antonio; "The Historical Development of the Neurosurgical Treatment of Pain," Dr. Walker; "Broadening Indications for Total Thyroidectomy," Dr. Joe T. Gilbert, Austin; "Ephebiatric Oncology," Drs. J. W. Humphreys, Jr., Lackland Air Force Base, and L. J. Fontennelle; and "Parathyroid Cyst," Dr. Edwin M. Sykes, Jr., San Antonio.

TAGP Assembly Plans Complete

For early arrivals at the Texas Academy of General Practice meeting, on Sunday, Oct. 11, the TAGP Education Committee has planned a Pre-Assembly Seminar on Physical Medicine in General Office Practice. Guest speakers, from the Department of Physical Medicine, Texas Institute for Rehabilitation and Research, Houston, will include Drs. Charles R. Peterson, John S. Tennant, Max A. Stoner, and Lewis A. Leavitt. On the evening schedule is a cruise of Corpus Christi Bay.

The scientific program proper will begin Monday morning, Oct. 11, in Memorial Coliseum, and end at noon Wednesday, Oct. 14. The guest speakers will include Drs. Donald A. Sutherland, Dallas; William E. Barfield, Augusta, Ga.; Charles T. Stewart, San Mateo, Calif.; Philip Thorek, Chicago; Dr. Petter A. Lindstrom, San Francisco; and F. James McCutchon, Corpus Christi.

Tuesday's slate of speakers will be Dr. Joseph G. Klotz, Corpus Christi; Dr. Thorek; Dr. Lindstrom; Dr. W. E. McKittredge, New Orleans; Dr. Stewart; Dr. B. C. Sinclair-Smith, Nashville, Tenn.; and Dr. John McGivney, Galveston.

Wednesday morning's program will feature Drs. McGivney, Chinn, Kittredge, and Sinclair-Smith.

The TAGP Committee on Annual Scientific Assembly, responsible for planning the assembly program, is under the chairmanship of Dr. Hunter Scales, Jr., of San Benito.

Dr. Haden E. McKay, president of the 1,650-man Academy in Texas, will deliver a message of welcome during the opening ceremonies preceding the first lecture Monday morning.

An extensive social program will include a beer-and-oyster party, Monday evening, hosted by Marion Laboratories; a "night in Las Vegas" party and midnight buffet, Tuesday evening; and luncheons Monday and Tuesday.

Mrs. Dwight S. Ewing heads the wives' committee which has completed plans for wives' entertain-

ment. Mrs. Ewing reports that activities for the ladies will begin Monday and extend until noon Wednesday, affording "every-minute-of-the-day" wives' entertainment.

All members of the Texas Medical Association are welcome to register for the Assembly's Scientific Program; the fee will be $10.

Panhandle District To Meet Oct. 3

"What's New in Medicine" is the theme that has been chosen for the annual meeting of the Panhandle District Medical Society which will be held at the Holiday Inn West in Amarillo on Oct. 3.

Physicians from throughout the Panhandle of Texas, Western Oklahoma, Eastern New Mexico, and Southern Kansas are expected to attend the meeting which will have an intercontinental flavor this year.

Highlighting the program will be the appearance of Sir Stanford Cade, Knight of the British Empire, C. B. Lord Cade is a fellow of the Royal College of Surgeons of England and of Edinburgh, an honorary fellow of the Royal College of Obstetrics and Gynecology and is the Hunterian Professor of the Royal College of Surgeons.

Featured as the speaker in the surgical section of the meeting, Lord Cade will discuss the latest trends in surgical treatment of cancer of the breast. Another paper will discuss the latest trends in surgical treatment of cancer of the mouth, tongue, and larynx.

Equally distinguished in their own fields in America as Lord Cade is in England will be the other scientific speakers on the program for the District meeting.

Heading up the medical section will be Dr. Raymond D. Pruitt, professor and chairman of the Department of Internal Medicine, Baylor University College of Medicine, Houston.

Dr. Pruitt will discuss "Medical Education — 1964 — A Clinician's Appraisal" and "An Appraisal of Developments in Cardiovascular Diagnosis and Treatment."

The chairman of the Department of Obstetrics and Gynecology at Vanderbilt University School of Medicine, Dr. Robert W. Noyes, will lead the discussions in the obstetrics and gynecology section of the meeting.

Dr. Noyes will speak on "The Endometrium and Fertility" and "Maternal Impression Resurrected."

Dr. John H. Githens, professor of pediatrics and associate dean of the University of Colorado School of Medicine, will be featured in the pediatric section. Dr. Githens will speak on "What's New in the Management of Jaundice in the Newborn" and "New Developments in Pediatric Hematology."

In addition to hearing about "What's New in Medicine," physicians atttending the meeting will have an opportunity to learn more about the "Business Side of Practicing Medicine" with the appearance of Horace E. Cotton of Southern Pines, N. C., a management consultant to the medical profession. Mr. Cotton is a widely read contributing editor to *Medical Economics*.

Other guests for the meeting include Dr. Travis Smith of Abilene, Vice President of the Texas Medical Association, and C. Lincoln Williston, Executive Secretary of the Texas Medical Association.

Activities for the ladies also are being planned for the District meeting. Their guest of honor will be Mrs. W. D. Nicholson of Lake Jackson, president of the Woman's Auxiliary to the Texas Medical Association. The wives of attending physicians will also be entertained at a luncheon and style show at the Amarillo Country Club.

The Amarillo Country Club will also be the scene of the evening festivities when physicians and their wives will be guests of E. R. Squibb and Co. for a hospitality hour.. This will be followed by dinner and dancing to the music of Johnny Long's Orchestra.

A post-meeting activity will be the golf tournament sponsored by Pfizer Laboratories at the Amarillo Country Club on Sunday morning. Participants will be furnished balls, awards, cocktails, and lunch by Pfizer. Nonmembers of the Country Club will be expected to pay their own green fees.

Plans are now being formulated to take visiting speakers and other honored guests on a sightseeing tour on the Friday afternoon before the meeting.

The tour will include luncheon and a visit to Cal Farley's famous Boy's Ranch and a stop at Amarillo's famed helium plant which is doing considerable research in the use of helium in medicine, particularly space medicine.

Over 200 physicians and their wives are expected to attend the meeting.

Oklahoma Clinical Opens Conference on Oct. 26

The Oklahoma City Clinical Society will open its 34th annual three-day conference at the Sheraton-Oklahoma Hotel on Oct. 26.

A program of postgraduate teaching has been arranged to include lectures and discussions by 15 distinguished speakers selected from various medical and teaching centers throughout the United States, among them Dr. Robert R. Franklin of Houston. In addition to the general assemblies there will be specialty lectures, breakfast meetings, roundtable luncheon meetings, and a clinical pathologic conference.

On Monday evening, there will be a social hour followed by specialty group dinners. Tuesday morning, breakfast meetings will be held, and Tuesday evening will be the annual Clinical Society banquet with outstanding musical entertainment.

All physicians who are members of their county medical societies may attend this meeting from Oct. 26 through 28. Registration fee is $25.

For further information, physicians may write: Executive Secretary, Oklahoma City Clinical Society, 2809 Northwest Expressway, Oklahoma City.

Dr. Wade Goes On Tour of Peru

Dr. David Wade, President-Elect of the Texas Medical Association, recently spent three weeks touring Peru with a group of eight Texans who represent the newly formed "Partners of the Alliance of Progress," a program created by the State Department's Agency for International Development. Under this program Texas has "adopted" Peru and is to find ways and means of helping this sister nation.

The purpose of the tour was to survey grass roots needs in Peru where Texas may help. Help may be in the form of a plan of assistance designed to supplement existing alliance programs and projects now under way. Lights have been provided for night adult literacy classes in Indian villages through the efforts of Texans, and a cement mixer and highway equipment have been sent to other villages.

Alvar Nunez Cabeza de Vaca

Soldier, Explorer, Amateur Medical Man

For physicians interested in
tory, and particularly the
growing number fascinated by
no, here is a complete
account of the Spanish explorer
beza de Vaca's wanderings
and captivity among the Indians.
za de Vaca, it seems,
performed some surgery and
gained a certain reputation
the Indians as a physician.
He was without training in
medicine, his methods were
de, and yet he may have
been successful at times.

Álvar Núñez Cabeza de Vaca

S. R. SNODGRASS, M.D.

IN SEPTEMBER, 1962, a bulldozer uncovered several skeletons near the lower end of Galveston Island. In fear that these might be remains of prohibition day gangsters, or less likely, victims of Jean Lafitte who for a time made his headquarters at Galveston and presumably buried treasure down the Island, the bones were promptly taken to the Sheriff's Office. Later that day, anatomists at the University of Texas Medical Branch identified them as bones of Indians. Further digging uncovered 19 skeletons buried in the fetal position facing west, while a 20th was found in a more extended position facing east. Nineteen skulls were rounded and their teeth showed no overbite while the 20th was more elongated and had an overbite. Carbon dating studies indicate that burial took place approximately 400 years ago. Since the first white men reached Texas about that time, the 20th skeleton, differing so much from its fellows, may not be that of an Indian.

Álvar Núñez Cabeza de Vaca* was born in Jerez de la Frontera, Spain, probably in 1490. During the wars against the Moors, a maternal ancestor led the Christians through an unguarded mountain pass enabling them to fall upon the unsuspecting Moors and win the decisive victory of Las Navas de Tolosa in 1212. As the pass had been marked with a cow's skull, King Sancho of Navarre awarded the leader the name of Cabeza de Vaca, or cow's head, as a title of honor. Pedro de Vera, Cabeza de Vaca's paternal grandfather, conquered the Canary Islands in a campaign notable, even in those days, for treachery and cruelty to the natives.

In 1511 Cabeza de Vaca went to Italy with a Spanish force to aid Pope Julius II and fought in several battles during a two-year tour of duty. Upon his return he helped overcome local uprisings and later fought against the French in Navarre.

The odyssey of Cabeza de Vaca began in June, 1527, when, as treasurer and second in command of an expedition to explore and colonize Florida, he sailed for the New World. The organizer and commander, Pánfilo de Narváez, had served as lieutenant governor of Cuba and had lost an eye fighting in Mexico. Six hundred men in five small vessels reached Santo Domingo early in August and remained there 45 days collecting horses and provisions. One hundred forty men deserted before the expedition sailed for Santiago de Cuba to take on more men, arms, and horses. Two vessels under Cabeza de Vaca's command proceeded on to Trinidad, a town on the south coast of Cuba, to load provisions, but while there a hurricane struck causing the loss of both vessels, 60 men and 20 horses. After this disaster, the expedition went into winter quarters. Cabeza de Vaca's description of the storm with the wind blowing from one direction followed by a calm and then the return of the wind from the opposite direction was the first given for a hurricane.

On Feb. 22, 1528, the fleet sailed for Havana but ran aground on shoals off the Isle of Pines and remained there 15 days. Later when they attempted to enter Havana harbor, a south wind drove them north towards Florida. Land was sighted on April 12 and after coasting along the shore for three days searching for the entrance to Tampa Bay where a base had been planned, on Good Friday, April 15, a landing was made near the present site of St. Petersburg. Exploration nearby showed villages with growing corn. A few bits of gold were also found; the Indians indicated these came from a town called Apalachee farther north.

By May 1, as food was running short, 300 men with 40 horses pushed into the interior following the coast northward, hoping to find a harbor but most of all to find gold. There were but two pounds of hardtack and a half pound of bacon per man. Cabeza de Vaca recommended that the expedition remain upon their ships until they reached a harbor but was charged with lack of courage. Except for occasional villages with corn there was little food and the Indians were unfriendly. Apalachee, reached on June 25, was a miserable huddle of 40 thatched huts with no sign of gold or precious stones. Three exploring parties returned with reports of a difficult impassable country and hostile natives. There was abundant game including the opossum; Cabeza de Vaca was the first to describe this animal. The Spaniards decided to return to the coast but progress was difficult on account of the terrain and harassment by the Indians. Illness, probably malaria, affected many of the men before they reached the coast at Apalachee Bay, Fla.

Although there was but one carpenter among them, the Spaniards built a forge and melted their stirrups, spurs, and other metal objects to form nails, saws, axes, and other tools. Every three days a horse was killed for food; the skin, flayed from the legs, was used for water bottles. In six weeks five barge-like boats, each 33 feet long, were built. Ropes and rigging were made from the tails and manes of the horses and the men's shirts were used to make sails. More than 40 men died of disease while the boats were being built and others were killed by the Indians.

On Sept. 22, 1528, 247 men embarked in the five boats sailing westward as they believed Mexico, actually 1,200 miles away, close at hand. When the boats were loaded, there remained but six inches of freeboard. The boats floundered along the coast, sometimes landing to search for food and fresh water and at times encountering Indians. The freeboard of the boats was raised 16 inches by constructing waistboards from captured Indian canoes. Continuing along the coast, they reached Pensacola Bay, Mobile Bay, and then the mouth of the Mississippi. So great was the river's flow that they dipped fresh water from the sea. When they attempted to cross the river, the current and a rising north wind carried them beyond sight of land. Land was not sighted until the third day when they were again driven offshore by a strong current and the boats became separated. Food was limited to half a handful of raw corn per day. After four more days, only a few of those in Cabeza de Vaca's boat were rational and only five could stand. When night came, Cabeza de Vaca steered the boat; no one else was able. Near dawn, the Spanish explorer thought he heard the roar of surf and after sunrise, the boat

*The drawing of Cabeza de Vaca on the preceding page is the work of Tom Lea and is reproduced with his permission from the book, "Twelve Travelers Through the Pass of the North."

was cast upon Galveston beach. The Spaniards began to revive, a fire was built, rainwater found, and some of their corn parched. The day was Nov. 6, 1528.

After they had recovered somewhat, Cabeza de Vaca chose Lope de Oviedo, the most vigorous of the men, to climb a nearby tree to reconnoiter. When Oviedo reported that they were on an island, Cabeza de Vaca sent him back to look for a path. Oviedo followed a trail more than a mile to some unoccupied huts. Searching for something edible, he found only a little dog and a few mullets. Taking these, he started to return, but before he had gone far was halted by shouts. Turning he saw three Indians armed with bows and arrows. When two more Spaniards appeared because of Oviedo's long absence the Indians allowed them to depart unmolested.

Soon the Spaniards were surrounded by a hundred bowmen who hesitatingly drew near at signs of invitation. They were tall, well-formed brown naked men and each wore in his pierced lower lip a piece of cane a quarter-inch in diameter. One or both of their nipples were perforated from side to side and a reed an inch thick and a foot or more long was thrust through them. The Indian leaders were given some beads and little bells. Each Indian gave Cabeza de Vaca an arrow as a sign of friendship, and indicated they would return in the morning bringing food.

At sunrise, the Indians returned with many fish and edible roots. In the evening they brought more food and were accompanied by their wives, naked except for girdles of Spanish moss, their deerskin skirted daughters, and their naked children.

For several days the Indians returned with their gifts of food. The Spaniards, wishing to journey on, dug their boat from the sand, placed all their clothes in the boat, and attempted to launch the boat in the surf. A short distance from the shore, waves overturned the boat, throwing out all but three who were caught beneath the boat and drowned. The remainder reaching shore were huddled naked about a fire when the Indians returned with their nightly gift of food. By signs, Cabeza de Vaca explained what had happened. The Indians sat down and wept for half an hour; these lamentations were not spontaneous but were a savage ritual. Before presenting a complaint or request, these tribes would weep for half an hour or more. With this concluded, the one visited would present all he had to the other, who after sitting for a while carried it away, often without a word.

It was apparent that their naked-

Three wanderers in elegant dress stand before a reproduction of the final page of Cabeza de Vaca's manuscript and the family crest. (Reproduced with permission from the Sheraton Corporation and the artist, Lumen Martin Winter.)

Drawing of a buffalo as described by Cabeza de Vaca. (Reproduced with permission from the Sheraton Corporation and the artist, Lumen Martin Winter.)

ness and the rising cold north wind doomed the Spaniards unless they could take shelter in the Indian village; the Indians indicated they would be honored by such a visit. The braves half carried and half dragged the weakened white men to another fire, where after resting and warming themselves, they were taken farther. There were four or five fires on the route to the village where a house was prepared with many fires. After the Spaniards arrived, the Indians danced and rejoiced all night. The Spaniards were uneasy, fearing these festivities might be a preliminary to their sacrifice.

The following day survivors from another of the boats, this one captained by Dorantes, came to camp. They had been cast ashore northeasterly on the same island a day earlier than Cabeza de Vaca. The strongest decided to continue to Mexico but when a launching was attempted the boat foundered, one man was killed, and all of their possessions were lost.

Because of the cold, it was decided to remain upon the island for the winter. Four of the strongest and best swimmers were sent forward on foot to make their way to Mexico to send back help. The task of finding food for the 80 hungry helpless men was an almost overwhelming one. As soon as the Spaniards' strength permitted, they were quartered in the various settlements upon the island.

The culture of the Indians living on Galveston Island was of the late Stone Age period. Their huts were made of reed mats, but they had no permanent homes, no domesticated animals, and planted no crops. They lived upon the island from October until February, placing their settlements along the bay, but as close as possible to the beach where they secured driftwood. The struggle for food was a constant one and often they went several days without eating. The children were nursed until the age of 12 to provide a more certain food supply. They fished by driving the fish into traps or weirs, but also by standing hip deep in water and shooting fish with arrows. They ate the submerged roots—a walnut size tuber—of a plant growing in the shallow water along the shore. After the plants began to sprout in February, the tubers were no longer edible and the Indians moved to the mainland. Usually they traveled in dugout canoes, although it was possible at some points to wade the two miles to the mainland. Oysters were abundant, and for three months this was the Indians' chief food. Later dewberries were plentiful for a month or so. These Indians were extremely generous and affectionate, and each brave took but a single wife. If there were children, the couple remained together for life, but if not they changed partners freely. They were extremely devoted to their children, and when a child died the whole village wept for them at dawn, noon, and evening for a year. When a son or brother died, all of the occupants of his house remained in seclusion for a month choosing to die of hunger rather than to go in search of food; others of the tribe brought them any surplus above their own bare needs.

The winter was severe, and disease came to complicate hunger and cold. A severe gastroenteritis appeared, and soon the Spaniards were reduced from 80 to 15 in number, and many of the Indians also died. Some of the Spaniards cannibalized their dead which was not at that time a custom of these Indians. When Indians came to where five Spaniards had lived and found five bodies with the flesh of four eaten, they were furious and their goodwill changed to threats and blows. They indicated they would have killed all the Spaniards at once had they expected such behavior.

The Indians believed that these strange white men must possess magic powers, and finally by threats and by withholding food made them undertake healing. Because the tribes on the island had no chief or priest, the medicine man was the most honored member of the tribe. He enjoyed special privileges and was allowed two or three wives who lived together in harmony. In case of a cure, the patient gave the healer all his possessions along with all those he could command from his relatives. The Spaniards learned the methods of the island medicine men; these included making cuts in the skin where pain was felt, sucking the skin about the incision, often discharging the blood into a bowl as a tonic for the women. They also cauterized by fire, afterward blowing on the location of the pain and giving the patient to understand that his illness was cured. In addition to these methods, Cabeza de Vaca blessed the sick, breathed upon them, recited the Lord's Prayer and an Ave Maria, meanwhile praying with all earnestness to God that he would give help.

By the end of the winter, Cabeza de Vaca's failures lost him his status as a medicine man. He was set to pulling the submerged roots in the salt marshes and his hands became so tender that they bled at a touch. When the tribe moved to the mainland, he helped carry and set up the mat houses and gather shellfish.

In April, 1529, the Spanish leader became seriously ill, probably with malaria. Twelve survivors from the island on their way southward to Mexico visited the camp, but did not see Cabeza de Vaca and continued on their way. During the summer, there were certain roots, which resembled onions, to eat and at times fish. Occasionally a hunter would bring down a deer. When luck was bad, the tribe ate large spiders, lizards, snakes, and rats. Mosquitoes were a constant torment. When Cabeza de Vaca was well enough, he was put back to work digging roots, bringing driftwood, and when the tribe moved, as it must every few days to look for food, he helped carry the mat lodges.

In the autumn the tribe returned to the island where Oviedo had remained. He took no interest in Cabeza de Vaca's plans for escape.

When the tribe returned to the mainland in February, 1530, the noble slave to the Indians fled farther inland and established a trading enterprise. From the coast he brought shells and cockles for beads and large conchs for knives. He bartered these for deerskins, red ochre for painting faces and dyeing hair, hard canes for arrow shafts, and flint for arrowheads. He ranged over a large area of East Texas, probably reaching the Red River, and was welcomed everywhere. He delayed venturing toward Mexico hoping that Oviedo, whom he visited each year, would join him. Finally, late in the summer of 1532, Oviedo agreed to go and they traveled down the coast to the Colorado River. After they ferried across, the Indians told them there were two other white men and a black faced man not far away to the south. When Cabeza de Vaca inquired as to how these men were, the Indians said

ROUTE OF CABEZA DE VACA

they were in poor condition as the Indian men and boys kicked them, slapped their faces, and beat them with sticks all day long for fun. With their bows, the Indians gave Cabeza de Vaca and Oviedo a demonstration of this punishment. When this was repeated upon several successive days, Oviedo escaped to return to the island. Several days later, Cabeza de Vaca met Dorantes at a camp along the river. At this time of year many tribes gathered along the Colorado River to eat pecans. Great groves of pecan trees extended up the river for many miles.

Another winter and spring passed while Cabeza de Vaca and Dorantes were captives of this tribe. Cabeza de Vaca reported seeing buffalo three times during this period and gave the first description of these animals which he called "hunch-backed cows." In the summer of 1533, the tribe journeyed to the prickly pear country where, when their fruits, or tunas, ripened, there was for once plenty of food and the Indians became fat. As many tribes gathered there, cautious questioning located the whereabouts of Castillo and his Moorish slave, Estebanico. Cabeza de Vaca and Dorantes decided to steal away and try to join their friends. Unfortunately, a great uproar arose in the tuna fields one night when two braves began to fight over a woman. Friends and relatives joined, many were beaten, and when all were exhausted, the factions packed their lodges and set out prematurely for home, taking the Spaniards with them.

After another dreadful year, the Indians returned to the tuna fields in the summer of 1534. Cabeza de Vaca and Dorantes sent word to Castillo and Estebanico to meet them at a certain place at the September full moon. After this meeting, the four fled northward as fast as possible to avoid recapture. After a few days, they joined a tribe, makers of bows, with whom Cabeza de Vaca had dealings when a trader. The Indian spokesman asked Castillo, perhaps the most striking in appearance of the group, if he could cure a great pain in his head which the medicine man had been unable to drive away. Castillo was a timid practitioner but upon strong urging from Cabeza de Vaca, he knelt, commended his spirit to God, said the Lord's Prayer, made the sign of the cross upon the aching head and said an Ave Maria. The Indian's pain was at once gone, and he soon returned with a gift of prickly pears and a piece of venison. Thereafter, all that night the sick of the village visited the miracle worker and each went away cured.

In their new role as healers, the Spaniards journeyed triumphantly farther north and west. Emissaries arrived from another tribe bringing five companions with a kind of paralysis which Castillo cured, receiving their bows and arrows as pay. The Spaniards remained in this area throughout the winter, again experiencing periods of great hunger. In the spring of 1535, they resumed their journey westward, guided by Indians who wished them to remain on account of their cures but were fearful of detaining them on account of their presumed magic powers. Near the present site of Pecos, an Indian wounded much earlier by an arrowhead, which was lodged above his heart causing him much pain and keeping him sick most of the time, was brought to Cabeza de Vaca. After proper prayers, the Spaniard palpated the area and found the arrowhead had passed through the cartilage. Using a knife, he opened the wound, found the point aslant but continued to

The author, Dr. Snodgrass, is professor of neurological surgery at the University of Texas Medical Branch, Galveston. The paper was first presented to the Texas Surgical Society in April, 1964.

cut. Finally, putting in the point of the knife, he drew forth a very long arrowhead. With a deer bone he made two stitches and stopped the flow of blood with the hair of a skin. The next day the sutures were removed and the Indian was well. This, the first recorded surgical operation in North America, greatly heightened the Spaniards' fame.

On the next stage of their journey, 2,000 Indians accompanied them. The Spaniards, barefoot and naked except for loin cloths, ate but one meal daily. As night drew nigh, the Indians made camp, setting up a mat lodge for each of the Spaniards. Food was cooked, and the best was set before them. The Indians then knelt in turn for the sign of the cross to sanctify their food. Frequently the Spaniards were accompanied by 3,000 or 4,000 persons, and as the food and drink of each had to be blessed before they would eat, their numbers were a great hindrance. When the Spaniards reached another tribe, they received many gifts. The Indians who had accompanied them then returned to their homes taking with them all the gifts presented to the newly arrived Spaniards thus replacing the gifts taken by the Indians who brought the Spaniards to them.

Permanent settlements where the Indian grew corn, beans, and squash were found along the Rio Grande and westward although the wanderers passed south of the great pueblos. Stretches of desert were crossed and finally they reached the valley of the Sonora River. When they reached the coastal plain, they turned southward to the Yaqui River which was flooded to such a stage that they had to wait two weeks before fording it. Here Cabeza de Vaca saw an Indian wearing a necklace with the buckle of a sword belt from which dangled a horseshoe nail. He learned that white men, some of whom rode horses which the Indians regarded as fearsome dragons, had visited there in the past.

The wanderers continued southward, and early in March, 1536, met Spanish soldiers burning villages and taking slaves along the river Sinaloa. Cabeza de Vaca was outraged at this treatment of the Indians and helped stop such activities as soon as he was able. Thereafter the journey continued southward with facilities provided by the Spaniards. It took a long time for Cabeza de Vaca to become accustomed to wearing clothes and shoes and to sleeping in a bed. Mexico City was reached on July 23, 1536, where the travelers were handsomely received by the viceroy and by Cortez. Their journey of almost 3,000 miles changed the ideas as to the geography of North America, led to further exploration to the north and west, and showed the value of cooperation with the natives. A report and map of their travels was prepared for the viceroy to forward to the king, but no trace of these is known. The Spaniards prepared another report which Cabeza de Vaca took to Santo Domingo, headquarters of the Spanish colonies. Although this report has disappeared, Oviedo, the contemporary historian, consulted and summarized it in his "History of the Indies." In 1543, Cabeza de Vaca prepared his own story of the journey calling it "The Shipwrecked Men"; a true copy of this exists in the New York Public Library.

The story of Cabeza de Vaca's survival in spite of incredible hardships and dangers makes fascinating reading and arouses speculation as to what forces allowed him to persist in his efforts and furthermore how his character and sense of values may have been altered by his experiences.

To return to Galveston Island—may not the 20th skeleton be that of Oviedo or some other member of the Spanish band?

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Community Health Week To Be Observed Oct. 18-24

The second annual Community Health Week has been set for observation Oct. 18-24.

The more than 2,000 medical societies and more than 20,000 public and private health agencies will commemorate the occasion by holding special community health programs, according to Dr. F. J. L. Blasingame, Executive Vice President of the American Medical Association.

The spotlight will be on health progress, medical advances, and the health services and facilities available in each community.

A packet of materials for promoting Community Health Week has been mailed from the AMA to every county society public relations chairman, and a small supply of these packets are available for others who may be interested. These may be obtained by writing to Jon R. Hornaday, Public Relations Director, Texas Medical Association, 1801 N. Lamar, Austin.

Chest Physicians Plan Mexico Congress

Physicians from more than 50 countries will present papers and discuss the recent advances in cardiovascular and pulmonary diseases at the eighth International Congress on Diseases of the Chest to be held in Mexico City, Oct. 11-15.

The Hon. Adolfo Lopez Mateos, President of the Republic of Mexico, will officially open the Congress on Sunday, Oct. 11.

All physicians are invited to attend this important Congress. For further information, physicians may write to the American College of Chest Physicians, 112 East Chestnut St., Chicago.

Sleeping Sickness—A Case Of Mistaken Identity

Viral encephalitis, an epidemic threat in Houston in late August, may also stage late-summer outbreaks elsewhere in the nation, public health officials have warned.

Confirmed cases have been reported in Florida as well as Texas; and suspected cases have been found in Illinois and Arizona.

In addition, the virus has been discovered in certain Florida mosquitoes and horses, important because the disease is often spread from animal to man by insect bites and stings.

In fact, insect-borne encephalitis is strictly speaking a disease of birds and small mammals rather than man, according to Raymond L. White, M.D., director of Environmental Medicine and Medical Services for the American Medical Association.

"Its transmission to man is, in a sense, an accident," he said.

The insects known to transport viral encephalitis generally disdain humans as hosts. But in their short-sighted world they mistake identities and man sometimes gets stung.

Accident or not, encephalitis is often severe and the percentage of fatalities can range from 2 to 75 percent. In addition, some victims are left paralyzed, enfeebled, or mentally affected.

Insect-borne encephalitis can be found in one form or another in many sections of the world. Three types are known in the United States—Western equine, Eastern equine and St. Louis.

The St. Louis type, identified with the Texas outbreak, gets its name from the epidemic it spread in and around that city in 1933 and again in 1937. It is much more likely to attack older people and the mortality rate increases steadily with age. Those over 40, and persons with high blood pressure and arteriosclerosis appear to be particularly endangered by the disease.

Western equine, as the name implies, was first thought to be spread by horses. But it later developed that horses, like men, are accidental victims. This form of encephalitis is most prevalent in the Western two-thirds of the nation, and although considered the mildest form in the United States, it kills about one victim in ten.

Eastern equine predominates among children. It is the most serious of the three types and is often fatal. Among young survivors the after-effects are often severe. Fortunately, the disease is infrequent, but the highest number of outbreaks is usually in September.

The viruses of encephalitis disrupt or destroy nerve cells and frequently damage small blood vessels supplying the brain. Susceptibility to encephalitis may have something to do with heredity; it may also be related to a weakness or the presence of other disease germs in a particular person's nervous system.

There is much about encephalitis that is still unknown. For instance, while mosquitoes get most of the blame for spreading the disease, it may well be that other insects such as mites, lice, and ticks are also implicated.

Just as important as determining what insects can transport encephalitis is finding the disease reservoir. Birds, ranging from song sparrows to turkeys, may be the chief source. Evidence of the virus has also been found in domestic farm animals, rodents, and other forms of wild life.

From what is known about encephalitis, it appears that it cannot be stamped out by eradicating one form of insect. Encephalitis is basically a disease of small animals. In this sub-human world it thrives and is probably passed from one species to another by a variety of infesting insects. Once in a while an insect comes along that will bite both animal and man. Then given the right circumstances—a large reservoir of infected birds, a large mosquito population and a dense human population, for instance—and you have an epidemic.

The only sure way of eliminating encephalitis would be to kill off much of the animal population, including valuable domestic breeds, or to wipe out insect life—an impossible task.

"More realistic," said Dr. White, "would be a nationwide program of surveillance. By keeping close watch on encephalitis in animals, by taking into account the density of insect populations as well as the predominant variety in any one area, it may be possible to initiate a program of encephalitis prediction, as influenza epidemics are now predicted.

"Once it is possible to predict where and when encephalitis might strike, then it would also become possible to take strong, concentrated control measures to make sure that the disease doesn't 'accidentally' get transmitted to man."

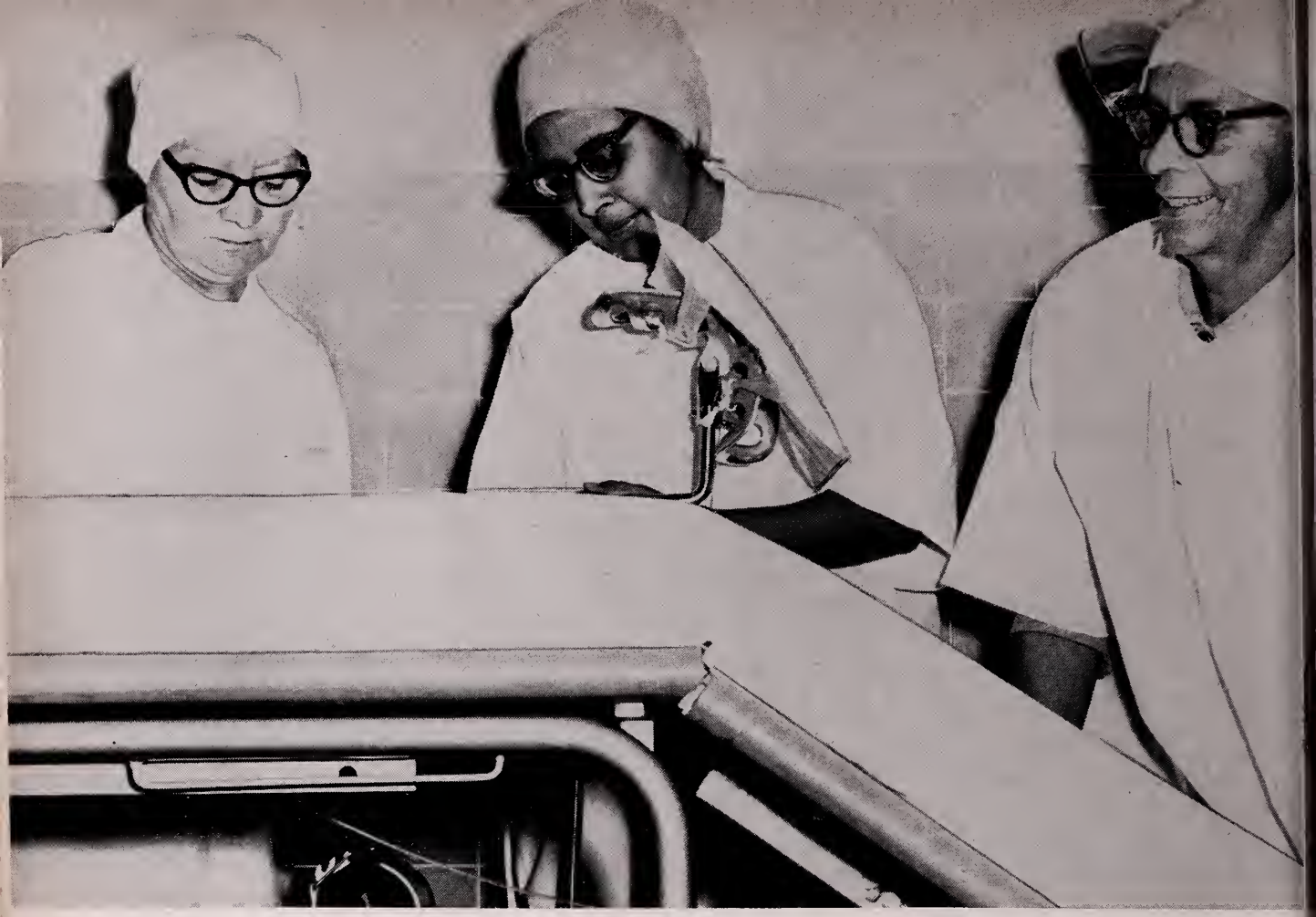
Doctors Urged To Write Congressmen Opposing Medicare

The President of Texas Medical Association, Dr. Max E. Johnson of San Antonio, issued an urgent appeal, following the Senate's 49-44 approval of the Administration's medicare amendment on Sept. 2, for doctors, their friends, and their patients to write their Congressmen opposing such legislation. The House of Representatives must approve the amendment if it is to become law.

Dr. Johnson's letter to members of Texas Medical Association noted that the medicare tax program is extremely expensive. The letter further noted, "It is not needed in Texas. Great progress has been made in our state in just a few short years. As you know, Texas has implemented an effective, economical program for 229,000 needy aged. Blue Cross-Blue Shield and the commercial insurance companies have introduced special plans, and Texas now leads the nation in the percentage of the aged who have one or more health insurance policies. The Belden study has shown that almost all aged who need and want help are receiving it. Texans will vote on a Constitutional Amendment on November 3 which will make possible the establishment of the second portion of the Kerr-Mills Act for those remaining few who still need help."

Doctors and patients alike are being asked to continue their telegram and letter writing campaign, asking their Congressmen to oppose the amendment passed by the Senate, or any compromise proposal embracing hospital care for the aged financed through the Social Security System.

"Please make an all-out effort to defeat the Administration's medicare program," Dr. Johnson said in his plea. "If we lose this legislative fight, we have lost forever."



Dr. Lena Edwards, right, prepares the delivery room at her hospital, Our Lady of Guadalupe Maternity Clinic, with the assistance of two registered nurses, Marie O'Conner, left, and Stella Gonzales, center. (Photo courtesy *The Hereford Brand.)*

Winner of Freedom Medal

Negro Woman Doctor Serves Migrants

A member of the Texas Medical Association has been named a recipient of the Medal of Freedom, the highest civilian honor the President of the United States can bestow in peacetime.

Dr. Lena Edwards of Hereford is one of 30 men and women in the nation who are to receive the award in 1964. Established in 1963, the Medal of Freedom has gone to those who have contributed significantly to security or national interests, world peace, or cultural or other public or private endeavors.

Dr. Edwards' presidential recognition came as a result of her work in establishing a hospital near Hereford for Latin American migrant laborers.

"This slightly built Negro woman gave up what it took a lifetime to accumulate to fight for and help migrant workers in this area," reads an article in the Fort Worth *Star-Telegram* by Jon McConal. In a story dated July 11, McConal tells how Dr. Edwards, now 64, left her 11-room Frank Lloyd Wright-style home in the East to come to the Hereford Latin American labor camp, composed of workers and families living in squalid barracks which once housed prisoners of war.

A former professor at Howard University, Washington, D. C., where she received her degree in 1925, Dr. Edwards is a gynecologist and obstetrician. In 1946 she was named one of the 13 outstanding women doctors in the nation. She was chosen medical woman of the year in 1955 by the New Jersey American Women's Association. She is a fellow of the International College of Surgeons and a diplomate of the American Board of Obstetrics and Gynecology.

Dr. Edwards left Washington in 1960 and came to Hereford where she set up housekeeping in a small un-air-conditioned room, cooking her own meals and making many of her clothes. She had learned of the need for some one to help the Hereford migrant workers from a friend of her son while he was studying to become a Catholic priest. She found the labor camp, situated near the Graymoor Mission of St. Joseph, inhabited by about 200 families. Infectious diseases such as diarrhea abounded, and as many as five children a month were dying. The only place these people could afford to go when they were sick was to a small clinic operated by a nurse.

Through Dr. Edwards' efforts a 25-bed $60,000 hospital, licensed for maternity and surgical prac-

tice, now stands near the workers' homes.

"It has taken four years of my life and $30,000 of my savings," said Dr. Edwards, "but each time I see a healthy child playing, it makes it worth it."

The care the Latin American workers receive from Dr. Edwards and the hospital is by no means free—but it is within the means of the laborer.

"I don't believe in giving something for nothing," she said. "I never got anything for nothing, and these people should not either."

She charges a flat fee of $100 for prenatal care, delivery, postnatal care of each baby.

Dr. Edwards became aware of the need for good maternity care for people in the lower socio-economic strata in 1944 when she prepared a paper on mortality of childbirth in relationship to environmental factors. She wanted to do some missionary work then, but decided to wait until after she had educated her six children. Dr. Albert Schweitzer's continuing ability to work into his old age inspired her to enter mission work when she reached 60.

She was shocked by the condition of the labor camp in Hereford.

"I thought I was in a foreign country," she said.

Dr. Edwards found that women had three choices regarding delivery, according to the Star-Telegram article. One, they could bear their babies in the barracks with just another woman—generally untrained—in attendance. Two, they could go to a "certain" office where they were made to remain in the car until just before delivery. After delivery, the mother was allowed to recover on a stretcher for an hour after which she and the baby were sent home. Three, they could go to a hospital, pay $150 for the delivery, plus medicine, laboratory, and hospital fees.

The third choice was financially impossible for most of these women, Dr. Edwards found.

Dr. Edwards immediately started to work for her hospital. She used her own money and whatever came in from her friends and from other individuals in the community.

As a Negro, Dr. Edwards has known prejudice and recognizes the plight of the Latin American workers who also face prejudice. She believes that the Latin American can better himself, however.

"I was born a Negro," she said. "I've never cried about it. I believe a person can accomplish anything regardless of color, if they try hard enough."

In a letter to *Texas Medicine* telling of her hospital, Dr. Edwards concludes, "With God's help and changing hearts, I think we can make this a pilot project for low income medical care which does not exist in our area."

San Antonio Luncheon To Honor Dr. Johnson

The Bexar County Medical Society and the San Antonio Chamber of Commerce will honor the President of the Texas Medical Association, Dr. Max E. Johnson of San Antonio, at a luncheon Tuesday, Sept. 29, at the St. Anthony Hotel in San Antonio.

Master of ceremonies for the luncheon will be Dr. Walter Walthall, Chairman of the Board of Councilors of the Texas Medical Association. Short testimonials will be given by the mayor of the City of San Antonio, the president of the Bexar County Medical Society, the president of the San Antonio Chamber of Commerce, the President-Elect of the Texas Medical Association, a Bexar County physician who is a lifelong associate of Dr. Johnson, the commander of the Brooke Army Medical Center speaking on behalf of the military physicians of San Antonio, the Bexar County Congressman, the president of the American Dental Association, the Executive Vice-President of the American Medical Association, and the president of the Alamo Chapter of the Boy Scouts of America.

The honoree has practiced medicine in San Antonio for 40 years and has been very active in medical organizations and in civic activities in the city, state, and nation. He is the past president of the Bexar County Medical Society, the International Medical Assembly of Southwest Texas, and other medical organizations. He has been a member of the City Council of San Antonio and interested in many other activities.

Physicians of the Texas Medical Association and their wives are invited to be present for this luncheon. Further information may be obtained by writing to Mr. S. E. Cockrell, Jr., Executive Secretary, Bexar County Medical Society, 202 West French Place, San Antonio, Texas 78212.

National Award Goes To Parkland Hospital

Dallas' Parkland Memorial Hospital has received special recognition from *Hospital Management Magazine.*

Judges in the MacEachern Public Relations Competition, sponsored by the magazine, made the award to the Dallas institution "for the general excellence of its total public relations program, demonstrated by the completely professional manner in which the complex events following President Kennedy's assassination were handled."

Hospital Management is the professional journal for hospital administrators and department heads, published by the Clissold Publishing Co., Chicago.

The awards were presented at a special breakfast given by the magazine during the American Hospital Association convention in Chicago on Aug. 24.

Dr. McKaig Ranks First On Licensing Examinations

Dr. Calvin Newton McKaig of Gladewater and now of Houston, recently licensed to practice medicine by the Texas State Board of Medical Examiners, made the highest grade on the examinations conducted by the Board June 22-24.

Dr. McKaig's average was 92.5. A certificate recognizing this accomplishment has been sent to him by the Board, according to Dr. M. H. Crabb, secretary.

The honor roll for the June examinations includes 36 names.

"Who's dissatisfied?"

Placing Mental Health Services Under One Agency Advocated

The Statewide and Community Mental Health Planning Committee has recommended putting mental health services under a single state agency.

This recommendation from the 112-member committee was made by a near-unanimous voice vote on Aug. 8 at the Texas Medical Association auditorium, as the panel wound up its fifth work session.

First meeting of the committee was held last December, followed by two-day work sessions in February, April, and June.

Judge C. L. Abernethy of Hale County, chairman, said that one more meeting will be necessary to put the Texas Plan for Mental Health Services into a complete form that will represent the group consensus. The 17-man Steering Committee and the chairmen of the 13 Task Forces for the planning will meet again during September, in advance of the full committee meeting.

Recommendation by the general planning committee of a single state agency for mental health services brought its thinking into focus on what has been considered one of the most difficult questions involved in drafting the new state plan. State mental health services now are divided among a number of agencies.

The committee did not attempt to say which of the present state agencies should become the central authority for mental health services in Texas, or if those services should be consolidated in a new agency. The Legislation Task Force, one of the 13 subcommittees within the planning group, has considered four possible alternatives:

(1) Placing the total program under the Board for Texas State Hospitals and Special Schools, which now administers the state mental hospitals and clinics.

(2) Placing the program under the Texas State Department of Health, which now performs some mental health services through its Division of Mental Health, and is now the mental health authority for Texas.

(3) Creating a new State Department of Mental Health, with its own governing board.

(4) Combining health and welfare activities under a single state agency.

Dr. Francis J. Gerty, Chicago psychiatrist and formerly director of the Illinois Mental Health Department, was a speaker and consultant to the committee. Dr. Gerty, who made a study of Texas mental health services and needs in 1956-1957 as a special consultant of the Texas Medical Association, told the committee he favored the principle of a single state office for administration of these services.

Such a central office has been established by the Illinois legislature, taking mental health services out from under the department of public welfare, Dr. Gerty said.

Now in psychiatric practice in a private clinic in suburban Chicago, Dr. Gerty credited his experience in Texas as a contributing factor for turning the concept of community mental health services into reality in Illinois.

Dr. Gerty said the Texas Plan now being drafted would be similar in principle to that in Illinois —a plan for community-centered comprehensive facilities having the full range of services needed for the diagnosis and treatment of all forms of psychiatric illnesses.

He said the "zone plan" for locating eight clinic-hospitals now being implemented in Illinois, however, would not be suitable for Texas because of the latter's size and population characteristics.

"Personnel is still a grave problem," Dr. Gerty said. "The Illinois Psychiatric Institute is gearing its program to our clinic-hospital manpower needs. Before 1960-1961 we were losing trained personnel to other states. Now these people are coming back to Illinois, attracted by better salaries.

At the close of the committee meeting, Judge Abernethy called for reports from each of the 13 Task Force chairmen. Some reported their work completed while that of others was nearing final form.

Judge Abernethy said that after the full committee puts its stamp of consensus on the tentative Texas Plan at the fall meeting, the next ingredient to be added to the plan will be public opinion.

"We will hand this Texas Plan to the Texas people—everyone from the medical profession and public officials to the man on the street—to see if they approve of it, if they want it, and if they will give it the moral and financial support necessary to put it into practice."

Working with Judge Abernethy on the Executive Committee for the statewide planning effort are Dr. James E. Peavy, State Commissioner of Health, chairman; Dr. Cyril J. Ruilmann, Director of Mental Health and Hospitals for the Board for Texas State Hospitals and Special Schools, cochairman; and Robert L. Sutherland, Ph.D., director of the Hogg Foundation for Mental Health at the University of Texas. Dr. Sutherland is vice chairman of the General Planning Committee.

Mental Health Group Sets Houston Meet

The Texas Association for Mental Health will hold its 32nd annual conference, Oct. 22-23, at the Sheraton-Lincoln Hotel in Houston. Focus of the meeting will be on volunteer leadership for mental health services.

Speakers will include Frank E. Proctor, St. Louis, president of the National Association for Mental Health; Mrs. Winthrop Rockefeller, Arkansas, chairman of the NAMH committee on volunteers; Arnold Maremont, Chicago businessman and member of the NAMH board; Mrs. Joseph Mandelbaum, New York, NAMH board member and former president of the National Organization for Mentally Ill Children; Waggoner Carr, Attorney General of Texas, and Mrs. Curtis Bok, Philadelphia civic leader.

Reports will be heard on programs and services presently going on in local mental health associations throughout the state. A session on communications will include a realistic examination of what it takes to arouse interest and motivate action in behalf of mental health. An examination of the plan as proposed by the State Mental Health Planning Committee will be made.

A gala benefit for children's services and research, the second annual Galaxy Ball will be held at 8:30 p.m. on Oct. 23 in the Sheraton-Lincoln Ballroom. A number of prominent motion picture and television personalities are expected to attend. Price of tickets is $25.

Three members of the Executive Committee for the current statewide and community mental health planning program conferred with Dr. Moody C. Bettis (right), psychiatrist-coordinator for the work, at an August planning session. They are (from left) Dr. James E. Peavy, Texas State Commissioner of Health, chairman; Dr. Cyril J. Ruilmann, Director of Mental Health and Hospitals for the Board for Texas State Hospitals and Special Schools, co-chairman; and County Judge C. L. Abernethy of Hale County and Plainview, chairman of the 112-member General Planning Committee. Fourth member of the Executive Committee, not pictured, is Robert L. Sutherland, Ph.D., director of the Hogg Foundation for Mental Health at the University of Texas and vice chairman of the General Planning Committee.

Postgraduate Courses

Cardiovascular Diseases, Houston, Oct. 31, 1964.—The Division of Continuing Education of the University of Texas Graduate School of Biomedical Sciences at Houston and the Frederick R. Lummis Medical Foundation will present a one-day symposium, "Current Practical Aspects of Cardiovascular Diseases," in the Texas Medical Center. This course is intended to give to practicing physicians a useful and authoritative review of current medical and surgical approaches used in managing common cardiovascular problems in adults. Credit for attendance will be given by the American Academy of General Practice. The following topics will be discussed: Changing Concepts in Ischemic Heart Disease; Hypertensive Vascular Disease, Diagnosis and Chronic Medical Management; Clinical Approach to Strokes; Management of Hypertensive Emergencies and Other Special Situations; Acute and Chronic Management of Strokes; Rheumatic Heart Disease in Adults, Current Concepts; Management of Cardiac Arrhythmias by Cardioversion; Indications for Surgery in Arteriosclerotic Diseases; and The Surgical Treatment of Mitral and Aortic Valve Lesions. Guest speakers are Dr. E. Harvey Estes, Jr., Department of Medicine, Duke University Medical Center; Dr. James Conway, Hypertension Unit, University of Michigan Medical Center; Dr. Clark H. Millikan, Department of Neurology, Mayo Clinic; Dr. John M. Lewis, Department of Medicine, Baylor University College of Medicine; Dr. Stanley Crawford, Department of Surgery, Baylor University College of Medicine; Dr. Denton A. Cooley, Department of Surgery, Baylor University College of Medicine.

For further information, physicians may write: Division of Continuing Education, The University of Texas Graduate School of Biomedical Sciences at Houston, 102 Jesse Jones Library Building, Texas Medical Center, Houston, Texas 77025.

Pediatrics, Houston, Nov. 12-14.—The Division of Maternal and Child Health of the Texas State Department of Health and the Department of Pediatrics, Baylor University College of Medicine, will hold their annual Pediatric Postgraduate Symposium at Texas Children's Hospital.

Guest speakers for the symposium will be Dr. Harry Bakwin, professor of clinical pediatrics, New York University of Medicine, New York; Dr. Thomas E. Cone, Jr., associate clinical professor of pediatrics, Harvard Medical School, Boston; Dr. Robert E. Cooke, Given Fund professor and chairman, Department of Pediatrics, Johns Hopkins University School of Medicine, Baltimore; Dr. Heinz F. Eichenwald, professor and chairman of the Department of Pediatrics, Southwestern Medical School, Dallas; Dr. Richard T. Smith, professor and chairman, Department of Pediatrics, University of Florida College of Medicine, Gainesville, Fla.

Interested physicians should contact Dr. Russell J. Blattner, chairman, Department of Pediatrics, Baylor University College of Medicine, 1200 Moursund, Houston, Texas 77025.

Senate Subcommittee Hears Pharmacy Secretary

The Senate Antitrust and Monopoly Subcommittee in August heard testimony from Joe H. Arnette of Austin, Secretary of the Texas State Board of Pharmacy, to the effect that 4.05 percent of Texas-licensed pharmacies are owned or partially owned by doctors or have doctors as officers and/or directors.

The Subcommittee is investigating the possibly monopolistic aspects of ownership of drug plants by physicians.

Arnette said the Texas State Board of Pharmacy has issued 2,886 pharmacy permits for the period June 1, 1964, to June 1, 1965. Of these, 117 reflect doctors as officers and/or directors and/or partial owners and owners.

He continued to say that 259 doctors or 1.6 percent of 16,034 practitioners licensed by the Texas Board of Medical Examiners have an interest in Texas pharmacies.

Twenty-six manufacturing pharmaceutical permits out of 126 issued by the Texas State Board of Pharmacy show doctors' interest, a percentage of approximately 20.6 percent.

Pharmaceutical manufacturing establishments are shown to have 74 doctors as officers and/or directors and/or partial owners or owners. This represents 0.46 percent of all doctors licensed in Texas.

Personals

Luther R. Parker has been appointed executive secretary of the Texas Pharmaceutical Association, to succeed C. J. M. Roesch who resigned in May. Parker is the former director of the pharmacy extension service and assistant to the dean of pharmacy at the University of Texas.

Texas physicians to appear on the program of the American Academy of Pediatrics Oct. 24-29 in New York are *Capt. Stanley N. Graven,* Lackland Air Force Base, and *Dr. John P. McGovern* of Houston.

A number of Texas physicians are scheduled to take part on the program of the meeting of the American College of Gastroenterology Oct. 19-21 in New York. These include Drs. *N. C. Hightower* of Temple, *Marcel Patterson* and *Arnold Drake* of Galveston, *Robert S. Nelson* and *Michael H. Siegelman* of Houston, *H. B. Eisenstadt* of Port Arthur, *Earle U. Scharff, Jr.,* of Fort Worth, and *Fred G. Conrad* of Lackland Air Force Base.

Dr. M. O. Rouse of Dallas will be one of the featured speakers at the meeting of the Association of American Physicians and Surgeons in New Orleans on Oct. 8-10.

Dr. Rouse will speak also to the members of the American Association of Medical Assistants Oct. 17 in Oklahoma City on the subject of "Medical Democracy in Action."

The preliminary program of the American Roentgen Ray Society annual meeting to be Sept. 29-Oct. 2 in Minneapolis lists the following Texas physicians as speakers: *Laird Jackson,* Houston; and *R. E. Collier, G. A. Balla, A. D'Errico, J. W. Tomme, J. E. Miller, J. T. Mallams, Charles L. Martin, James A. Martin,* and *Robert A. Wilson,* Dallas.

Dr. M. T. Jenkins of Dallas will give a refresher course on "Electrolyte Changes During Anesthesia and Surgery" at the meeting of the American Society of Anesthesiologists in Bal Harbour, Fla., Oct. 10-11. *Dr. Oliver F. Bush* of Dallas is president of the ASA this year. Others listed on the program who are from Texas include *Dr. Charles Barry, Dr. Etham Y. Kazucu, Dr. Stewart A. Wilber,* and *Dr. William S. Derrick,* Houston; *Dr. Billy Welch,* San Antonio; and *Dr. Edward Lowenstein* and *Lawrence I. Zaroff,* Fort Sam Houston.

Dr. Zoltan Petrany of Houston placed second in a recent sports-car competition at Opelousas, La. He has been interested in racing for several years.

Dr. Lawrence E. Lamb of the U. S. Air Force School of Aero-Space Medicine, Brooks Air Force Base, will give one of the basic science lectures at the forthcoming meeting of the American College of Physicians Oct. 8-10 in Los Angeles.

Texas physicians whose names appear on the program of the American College of Surgeons meeting include *John A. Moncrief,* Fort Sam Houston; *Harry M. Spence, Tom Shires, M. T. Jenkins,* and *Jack G. Kerr,* Dallas; *R. Lee Clark, Murray M. Copeland, Michael E. DeBakey, John S. Stehlin, Jr., George C. Morris, Jr., Denton A. Cooley, Walter S. Henly, Arthur C. Beall, Jr., John A. Wall, Herman L. Gardner, William S. MacComb, Richard H. Jesse, William O. Russell,* and *Edgar C. White,* Houston; and *Truman G. Blocker, Jr., Curtis P. Artz,* and *John R. Derrick,* Galveston. The meeting will be in Chicago, Oct. 5-9.

Dr. G. V. Brindley, Temple, was awarded an honorary degree at the summer commencement of Howard Payne College, Brownwood, on Aug. 21. His son, *Dr. G. V. Brindley, Jr.,* Temple, delivered the commencement address.

Dr. Leon Bromberg has been named "Boss of the Year" by members of the Galveston County Medical Assistants Society. Dr. Bromberg is a Galveston internist.

C. Lincoln Williston, Executive Secretary of TMA, and *Jon Hornaday,* Public Relations Director of TMA, attended the Medical Society Executives Association Institute and the AMA Public Relations Institute in Chicago in August.

Dr. Carlton E. Wertz of Buffalo, N. Y., Vice President of the AMA, is the owner of a ranch and other real estate in Raymondville.

Dr. Ray W. Ware has been named research manager of medical instrumentation of the Department of Electronics and Electrical Engineering of Southwestern Research Institute, San Antonio.

Dr. C. James Krafft of Dallas was designated as the official American Medical Association representative to the First Latin American Congress on Christian Medicine to be held in Quito, Ecuador, in July.

Dr. Michael E. DeBakey of Houston will be a member of a panel to discuss the epidemiology, diagnosis and prevention of strokes at the First National Congress on Strokes Oct. 29-31 in Chicago. Sponsoring agencies are the American Medical Association, American Heart Association, Heart Disease Control Program of the U. S. Public Health Service, and Vocational Rehabilitation Administration of the Department of Health Education and Welfare.

"The internal medicine boys are up to their old tricks."

Two Baylor University College of Medicine professors reported their scientific studies at the 42nd annual American Congress of Physical Medicine and Rehabilitation held in Boston in August. *Dr. Lewis A. Leavitt,* chairman, Department of Physical Medicine, reported on treatment of muscle weakness, and Willis C. Beasley, Ph.D., associate professor in kinesiology, reported on the electromyographic potentials of rested and fatigued muscles.

Dr. D. F. Ward Succeeds Dr. Welch as AMA President

Dr. Norman A. Welch of Boston, President of the American Medical Association, died Sept. 3 in Jackson, Wyoming. Shortly before he was to address the Wyoming Medical Society, Dr. Welch suffered a massive cerebral hemorrhage and died several hours thereafter. He was 62.

AMA President-Elect Dr. Donovan F. Ward, Dubuque, Iowa, assumes the duties of the presidency for the coming year.

Next Exam Announced By Basic Sciences Board

The next examination of the Texas State Board of Examiners in the Basic Sciences has been set for Monday and Tuesday, Oct. 19-20, in Austin.

Applications for the October examinations must be complete and in the office by Oct. 1, and all necessary information and documents required by examinees by the Board must be completed and in the applicant's file by that date.

Those interested in details of time and place should write to the executive of the Texas State Board of Examiners in the Basic Sciences at 1012 Sam Houston State Office Bldg., 201 East 14th St., Austin.

Las Vegas, Nev., To Host Southwestern Medical Meet

Southwestern Medical Association will meet in Las Vegas, Nev., Oct. 22-24. The faculty at the meeting is from the School of Medicine, University of California, San Francisco Medical Center.

Headquarters for the meeting will be the Flamingo Hotel. This is an open meeting with a registration fee of $25.

The three sessions of the scientific meeting are entitled "Systemic and Local Aspects of Urolithiasis," "The Hypertensive Patient," and "Diabetes and Renal Disease."

Members of the faculty are Drs. Robert C. Combs, James S. Elliot, Leon Goldman, Frank A. Gotch, Felix O. Kolb, Mary B. Olney, and Chauncey D. Leake, Ph.D. Dr. Leake will be the luncheon speaker on Oct. 23 on the subject of "The Treatment of Anxiety."

Additional information may be obtained by writing to Bob Reid, 310 North Stanton Street, El Paso.

Corsicana Physicians Undertake Mission Work

Two Corsicana physicians have recently gone to foreign countries as a part of the AMDOC program.

The AMDOC program, which has been undertaken as a community project of the Navarro County Medical Society, has headquarters in Wheaton, Ill., and was organized and incorporated in March of 1964. It has since been instrumental in sending a number of physicians overseas. Its purpose is to arrange for American physicians to work in needy places all over the world where they can bring American knowledge and experience to bear on local medical problems.

Dr. Robert D. Bone and his family went to Peru, and Dr. David Hansford, Mrs. Hansford, and two of their children went to Haiti. Dr. Bone was to work for several weeks with the Wycliffe Bible Translaters who are near Pucallpa, 300 miles inland over the Andes. He was to work in the Amazon River jungles near the site where five missionaries were slain in 1959.

Dr. Hansford was to serve as a gynecologist for a month at a missionary hospital at Port-de-Paix, Haiti.

Texan Named To Receive Tobacco Research Grant

Dr. Watts R. Webb, chairman of the Division of Cardiovascular Surgery at the University of Texas Southwestern Medical School, Dallas, has been named to receive a $44,868 grant to study the effects of tobacco smoke on blood circulation.

The grant is one of seven approved by the American Medical Association Education and Research Foundation at this time for tobacco research. In June, approval was announced of ten project grants—the first under the Foundation's research program on tobacco and health authorized last December by the AMA House of Delegates.

Blue Shield Benefits Over Billion Dollars

Benefits paid by the 77 Blue Shield plans in the United States, Canada, and Jamaica topped the billion dollar mark for the first time in 1963.

A total of $1,066,734,309 in benefits was paid to doctors for their services to a record number of subscribers and dependents. Blue Shield 1963 membership increased 2,545,635, up 5 per cent from 1962, to an all-time high of 53,450,349.

Blue Shield plans covered 26.41 percent of the United States population and 21.07 percent of the Canadian population in 1963.

The percentage of those covered in the United States increased in 1963, with Canadian Blue Shield coverage up over one-third that of 1962.

Blue Shield plans had 1963 enrollment gains totaling 1,627,798 while new plans in Memphis and Montreal added 917,837 additional members.

New Books In TMA Library

American Medical Association: **Report of the Commission on the Cost of Medical Care**, 4 vols., Chicago, American Medical Association, 1964.

Argosino, A. Y., Sr.: **Philippine Index Medicus**, vol. 2, 1917-1944, Manila, National Science Development Board, 1963.

Blair, E.: **Clinical Hypothermia**, New York, McGraw-Hill Book Co., 1964.

Child, C. G., III: **Liver and Portal Hypertension**, Philadelphia, W. B. Saunders Co., 1964.

Ciba Foundation Study Group no. 17, Wolstenholme, G. E. W., ed.: **Diet and Bodily Constitution**, Boston, Little, Brown and Co., 1964.

Cross, L. M.: **Preparation of Medical Literature**, Philadelphia, J. B. Lippincott Co., 1959.

Davis, L., ed.: **Christopher's Textbook of Surgery**, 8th ed., Philadelphia, W. B. Saunders Co., 1964.

Dunphy, J. E., and Botsford, T. W.: **Physical Examination of the Surgical Patient**, 3rd ed., Philadelphia, W. B. Saunders Co., 1964.

Garlington, W. K., and Shimota, H. E.: **Statistically Speaking**, Springfield, Charles C Thomas, 1964.

Jocobi, C. A., and Paris, D. Q.: **X-Ray Technology**, 3rd ed., St. Louis, C. V. Mosby Co., 1964.

Moore, F. D.: **Give and Take; The Development of Tissue Transplantation**, Philadelphia, W. B. Saunders Co., 1964.

Proctor, D. F.: **Tonsils and Adenoids in Childhood**, Springfield, Charles C Thomas, 1960.

Stephenson, H. E., Jr.: **Cardiac Arrest and Resuscitation**, 2nd ed., St. Louis, C. V. Mosby Co., 1964.

Swenson, O.: **Pediatric Surgery**,

2nd ed., New York, Appleton-Century-Crofts, 1962.

Transactions of Association of Life Insurance Medical Directors of America, vol. 47, New York City, Press of Recording & Statistical Co., 1964.

Wilder, J. R.: **Atlas of General Surgery**, 2nd ed., St. Louis, C. V. Mosby Co., 1964.

Young, H. H., and Owens, N., eds.: **Year Book of Orthopedics, Traumatic and Plastic Surgery**, 1963-1964 ed., Chicago, Year Book Medical Publishers, 1964.

Zollinger, R. M.: **Practical Outline for Preparing Medical Talks and Papers**, New York, Macmillan Co., 1961.

NEW JOURNALS

Biochemical Clinics, vol. 1, The Heart, New York, Reuben H. Donnelly Corp., 1963.

Biochemical Clinics, vol. 2, The Kidney, N. Y., Reuben H. Donnelly Corp., 1963.

Bulletins of the Joint Commission on Accreditation of Hospitals, Chicago, 1964.

Cleft Palate Journal, vol. 1, July, 1964, Baltimore, Md., American Cleft Palate Association, 1964.

International Anesthesiology Clinics, vol. 2, no. 1, November, 1963, Cardiovascular Problems, Boston, Little, Brown and Co., 1963.

International Ophthalmology Clinics, vol. 3, no. 3, September, 1963, "Keratoplasty and the Eye and Diabetes," Boston, Little Brown and Co., 1963.

Modern Treatment, vol. 1, no. 1, January, 1964, Treatment of Renal Disease, Treatment of Thyroid Disease, New York, Hoeber Medical Division of Harper and Row, 1964.

Modern Treatment, vol. 1, no. 2, March, 1964, Treatment of Pulmonary Diseases, Treatment of Liver Disease, New York, Hoeber Medical Division, Harper and Row, 1964.

Modern Treatment, vol. 1, no. 3, May, 1964, Treatment of the Anemias, Treatment of the Cardiac Arrhythmias, New York, Hoeber Medical Division, Harper and Row, 1964.

The Radiologic Clinics of North America, vol. 1, no. 1, April, 1963, Recent Clinical Advances in Radiology, Philadelphia, W. B. Saunders Co., 1963.

New Film In TMA Library

Fundamental Aseptic Techniques—1963. Sound, color, 21 minutes.

THIS film illustrates in detail the basic principles which must be taught to inexperienced personnel in preparation for work in an operating room. The purpose of the film is not to teach the skilled or instructor, supervisor, or scrub nurse, but rather to provide a visual reinforcement for lectures sometimes given in a classroom far removed from the actual operating room.

Book Notes

Books reviewed in this column are available from the TMA Library.

The Book of Health. R. L. Clark, M.D., and R. W. Cumley, Ph. D. 888 pp. Princeton, N. J., D. Van Nostrand Co., Inc., 1953.

THIS 888-page book with an index of 63 pages shows the vast amount of documentation that went into its preparation. It covers in understandable lay language very fine descriptions of how life begins, the birth of a baby, the new mother and a 32-page discussion of the child from infancy through teenage. Behaviour problems and common childhood diseases are dealt with. Childhood diseases are presented with colored photographs and apt captions with such clarity of description by Dr. Cumley that the parent can often recognize the condition present. The text is timely in that it depicts those symptoms and signs that help the parent to know when medical help by the family physician is indicated and needed. It is of particular interest to the reviewer because it presents historically the physician or scientist that contributed to the discovery and cure of the disease in question.

Other sections take up conditions seen in the central nervous system, the teeth, digestive system, urinary tract, reproductive system, and breast. Nutrition, management of sickness in the home, physical injuries, and first aid are among the many practical aspects of this most valuable contribution. This book will lead to intelligent self aid under both ordinary living conditions and in disasters of great magnitude.

Interesting aspects of tropical diseases are illustrated and discussed for those who would travel in tropical climates. Sanitation, insect and rodent control with a view toward eliminating vectors of disease are very effectively dealt with. (Mosquitoes, flies, rats and other disease carriers are specially stressed.)

New developments in medicine and law and their many ramifications are intelligently presented with the medical profession getting for once a good representation. This is followed by a section on medical history with a summary of its development through the ages. Then one encounters a refreshing account of your doctor, nurse, technologist, pharmacist, dentist, and finally your hospital, and health organizations. In conclusion and in keeping with the dynamic spirit of Texas in this modern age of space and Houston in particular there is a chapter on space medicine.

—Joe C. Rude, M.D., Austin.

Clinical Examinations in Neurology. Members of the Sections of Neurology and Section of Physiology, Mayo Clinic and Mayo Foundation for Medical Education and Research, Graduate School, University of Minnesota, Rochester. 396 pp. Philadelphia, W. B. Saunders Co., 1963. $8.50.

THE SECOND edition of "Clinical Examinations in Neurology" (W. B. Saunders, 1963) is a welcomed addition to other publications in this field. It affords the intern, resident, and generalist an outline of the rationale of the clinical neurological examination and should be available in hospital, and perhaps personal, libraries. The review of specific areas of the examination in which the examiner may wish some help, is simplified by the reference to the well divided chapters.

The first chapter, concerning the history-taking, contains many suggestions regarding common expressions, their significance and their relationship to neurologic syndromes. The practical significance of the various findings are outlined in the chapters on examination of infants and children, examination of the various cranial nerves, neuro-ophthalmology, central integrations of motor functions, specific study of muscles, reflexes, sensory examination, language and motor speech, autonomic function, neuroradiologic procedures, electroencephalography, electromyelography, and in the chapter on biochemical and pharmacologic aids.

The book should not be considered a textbook of neurology but rather an excellent reference book to be used in conjunction with a good textbook of neurology.

—Lewis M. Helfer, M.D.,
San Antonio.

Current Therapy 1963. Howard F. Conn, M.D., ed. 775 pp. Philadelphia, W. B. Saunders Co., 1963. $12.50.

THIS is a new edition rather than a revision of the old. Many topics have been rotated among authors from several countries, and many of the authors of this edition are contributing to "Current Therapy" for the first time. A few articles from the previous edition dealing with disease entities in which there has been little or no change in therapy have been revised or approved by their authors for this edition. In keeping with variations in the importance of certain diseases, and with the world-wide distribution of the book, certain deletions and additions of topics have been made.

Elements of Medical Statistics. J. V. Smart. 136 pp. Springfield, Ill. Charles C Thomas, 1963. $7.50.

THE AUTHOR expresses the hope that in writing the present book he will convince the medical student and young clinician that a knowledge of statistics will help to make him a better doctor, since it is becoming more and more difficult to open a medical journal without being confronted by statistics of some sort.

Viruses, Nucleic Acids and Cancer. Staff of M. D. Anderson Hospital and Dr. R. L. Clark. 659 pp. Houston, University of Texas M. D. Anderson Hospital and Tumor Institute, 1963. $16.

Viruses, Nucleic Acids, and Cancer" is the 17th symposium on fundamental cancer research and is compiled by M. D. Anderson Hospital and edited by R. Lee Clark.

The over 40 papers are well annotated with over 1,500 references. Each is essentially a review and written by a well qualified man.

Each paper is sufficiently detailed to describe preparation of specimens, microscope settings, and experimental patterns. It thus has appeal to all people from the curious to the researcher.

Several varieties of virus structure, their shape, and metabolism are covered in the first nine papers.

The second section deals with the manner of virus multiplication and their relation to bacteria.

Section three covers the same material on nucleic acid.

The possibility of virus etiology of cancer is given using several species of birds and mammals for experimental purposes. Numerous of these studies are reviewed with results.

The nature of interferon and its relation to viruses and tumor cells is discussed.

Evidence is presented for the infection and transmission theory of cancer in susceptible populations when accompanied by a carcinogen.

If animal experimentation is applicable to humans, there is an abundance of evidence here to prove the virus etiology of cancer. Furthermore, evidence indicates that one virus is capable of producing many types of cancer depending on susceptibility, type, and site of carcinogen.

As I closed the cover of this book I had the overwhelming, chilling sensation that a chapter was missing—the last. The work has been done, the experiments, most of them several times, have been performed and well observed. The answer to the riddle of prevention and treatment of cancer is ensconced in this book, but thus far the genius to put in place all the components of this jigsaw puzzle is lacking. It is my hope that this symposium will inspire that last chapter: "The Treatment and Prevention of Cancer."

I found the book inspiring, enlightening, and imaginative. It awakened dormant cells in my cortex, and opened great new vistas of thought and cogitation.

—V. L. THOMAS, M.D., Grandview.

Current Pediatric Therapy. S. Gellis, M.D., and B. Kagan, M.D. 694 pp. Philadelphia, W. B. Saunders Co., 1964. $16.

OVER 240 knowledgeable authors contributed to the writing of this book and this, together with sharp editing by Drs. Gellis and Kagan, brings forth an excellent addition to the library of anyone treating children. It is unfortunate that the size would make it difficult to carry in one's house call bag. No words are given to diagnosis—only to treatment. A happy discovery when one starts reading is this: the treatment of each condition is presented in detail, as the author sees it—not as a review of the literature with several conflicting opinions. It is readable, understandable and covers everything from amyloidosis to heat rash; minimum space is given to the very exotic disorders. Dosages are detailed, exact, and (happily) given in gr./lb. as well as mg./kg.

Pediatric favorites are especially well done; cystic fibrosis, rheu-

matic fever, epilepsy, malabsorption syndrome, diabetes, and urinary tract infections. An especially appealing section written by Dr. Kagan is "Judgment in the Use of Antimicrobial Agents." Dr. Hattie Alexander's section on H. Influenza Infection is a classic.

In summary, this book is written for the practitioner. It is practical, informative, and to the point.

—Creighton Reid, M.D., Sherman.

Christopher's Textbook of Surgery. Loyal David, M.D., 7th ed. 1,551 pp. Philadelphia, W. B. Saunders Co., 1960.

THE seventh edition of "Christopher's Textbook of Surgery" has continued the excellent fulfillment of the previous editions. In the 1,550 pages it is divided into 35 chapters. The text covers the field of general surgery as well as the specialties and subspecialties of general surgery, some in considerable detail, others only superficially. Each of the 82 contributors discusses the field lucidly, bringing to the reader the most modern concepts of etiology and therapy.

The outstanding chapters include the "History of Surgery" by Dr. Allen O. Whipple, and "Principles of Pre and Postoperative Surgical Judgment" by Dr. Harold Loffman which has been added. Dr. Mason's fine chapter on the hand remains a classic in the field, and the newer aspects of cardiac surgery are ably presented by Dr. Charles A. Hufnagel.

The text is attractively bound and is printed in an easily readable style. Visual chapters present without exception well ordered and well illustrated material with excellent reference work at the end of each chapter. Diagrams, pictures, and plates are sufficient and well chosen.

The text is designed primarily as a basic reference of surgery for the novice as well as the older student of surgery. The basic concepts of pathogenesis and treatment of surgical conditions are excellently portrayed. Relatively little attention is given to the technical details of surgical procedures.

—S. J. Kennedy, M.D., Cleveland.

Pediatric Therapy. H. C. Shirkey, M.D., ed. 1,104 pp. St. Louis, C. V. Mosby Company, 1964. $16.50.

THIS is a comprehensive review of the treatment of children. Although there is discussion of older methods of therapy, all chapters include the most recent information as well. The editor has included in his many contributors experts for each topic discussed.

The first half of this book is concerned with drug therapy, symptomatic therapy, and medical therapy of specific, systemic diseases. The chapters discussing routes of administration, home treatment, treatment of the chronically ill, and recreation therapy include a wealth of practical suggestions. Dosage and methods of calculating specific dosages are dealt with in an easily understood, concise manner.

The second half of "Pediatric Therapy" is a discussion of medical treatment according to body systems, detailed treatment of poisonings, and surgical treatment.

This book includes tables of dosages in the index, and other useful tables. It is an excellent, unusually readable and practical reference for a physician who treats children.

—Gretchen Runge, M.D., Austin.

Activities of Surgical Consultants, Vol. II, Surgery in World War II. Col. John Boyd Coates, Jr., ed. 1,062 pp. Washington, D.C., Office of the Surgeon General, Department of the Army. 1964.

THIS BOOK stands out to the reviewer because of the many men with injuries that under ordinary circumstances would have been considered disabling but with proper rehabilitation were able either to go back to active participation in the war effort or to resume useful and productive lives in a civilian capacity. The lessons learned during World War II have been put into practice across the entire land to the benefit of untold numbers. It is a testimony of ingenuity and resourcefulness with the will to survive by a people under grim and sometimes primitive circumstances with the available resources and materials at hand.

The development of centers for treatment of specialized areas began with the needs to treat orthopedic conditions, chest wounds and lesions, urinary tract disorders and injuries, vascular and cardiac wounds and diseases, abdominal injuries and unusual lesions, neurosurgical and peripheral nerve injuries and their associated diseases, diseases and injuries of the eye, ear and vestibular areas. Plastic surgery with its many necessary skills developed with great rapidity.

The use of surgical consultants brought out policies and directives for the handling of patients with the development of direct channels for such patients to specialized hospitals where trained teams worked long hours and did a tremendous job of repair and rehabilitation to the benefit of many soldiers, sailors, and airmen. The volume of such cases was so great that new methods of anesthesia, operative procedures, and after care could be developed to the benefit of patients and the medical profession the world over. It has spilled over into medical institutions of even our state as is witnessed by the cardiovascular work of Drs. DeBakey and Cooley in Houston, and one is sure it had its influence in stimulating Dr. Blocker in Galveston to continue his massive effort in the care of burns along with that of Brooke General Hospital at Fort Sam Houston.

The development in special surgical areas during World War II has resulted in the consultant system being set up for all the services of the Army, Navy, Air Force, and Public Health Service. This is

being continued during peacetime to improve patient care and as an instrument of instruction for medical officers throughout the world. It is a source of great satisfaction to the medical officer stationed in a remote area to have contact at regular intervals both from an educational and inspection point of view. It is here that deficiencies can be discussed and newer methods learned and instituted. It is even more important that those so stationed be kept up to date on the more recent developments in the practice of medicine and patient care. The point that one is trying to make is that it is a boosting morale factor for medical personnel everywhere to know that a special effort is being made to keep the best possible medical care for armed forces personnel wherever they may be stationed.

—Joe C. Rude, M.D., Austin.

The Lung and Its Disorders in the Newborn Infant. Mary Ellen Avery, M.D. 224 pp. Philadelphia, W. B. Saunders Co., 1964.

THIS BOOK is the first of a new series of monographs entitled "Major Problems in Clinical Pediatrics." It is a collection of information regarding the lung of the newborn infant, well amplified by facts obtained through the author's personal observations and experience. The development and physiologic function of the lung pre and postnatally in the normal as well as in the recognized disorders of the respiratory system of the newborn are detailed. The pathogenesis, abnormal physiology, pathology, as well as diagnoses and treatment of these disorders are outlined. Illustrations and chest x-rays are well chosen to clarify the text. An extensive bibliography is furnished for further study.

The chapters on congenital anomalies, hyaline membrane disease, resuscitation, and use of respirators are especially good and would be of interest to the practicing physicians who care for the newborn infant.

—Henry E. Martinez, M.D., Amarillo.

Drugs of Choice. Walter Modell, M.D., 1,018 pp. St. Louis, The C. V. Mosby Co., 1964. $15.

THIS is a comprehensive survey of pharmacology, pharmacy, dispensing, and medicine. It is an excellent reference and study book for both physician and pharmacist, but its detail precludes its use as a desk reference. It is limited to current remedies and does not have the facility to embrace any newly developed drugs. Supplemental information should be obtained through monthly or quarterly reviews.

"Drugs of Choice" has its greatest potential as a text for medical and pharmacy students. It makes pharmacology extremely digestible. The senior pharmacy student should be exposed to a text of this calibre. The medical terminology found therein is invaluable as a means of broadening the pharmacy student's intellectual and professional horizons. It will serve as a means to strengthen the physician --pharmacist relationship by providing a common ground for each to obtain a better insight into the other's profession.

One aspect of the book which is of particular interest to professional men is the fact that it gives unbiased testimony on the efficiency of drugs and is not a representation of the manufacturer's medical interest.

Any physician or pharmacist with 15 minutes a day to spare would have a sound investment in "Drugs of Choice" for $15.

—Barry Morgan, B.A., B.S. in Pharmacy.

"I'm here to see the incarcerated hernia."

The Care of Minor Hand Injuries. Adrian E. Flatt, M.D. 274 pp. St. Louis, The C. V. Mosby Co., 1963. $10.50.

IN THIS concise book, the author has endeavored to describe the fundamentals of the surgical management of hand injuries.

The book is divided into two parts. The first is concerned with general principles of care. The second section is concerned with specific injuries covering such entities as wounds of the skin, injuries to the nail, amputations, crush injuries, tendon and joint injuries and it also includes chapters on burns, fractures, infection, and miscellaneous injuries and wounds.

The treatment of each kind of injury is given in detail. Drawings and photographs throughout the book are well done and help to clarify the text. However, in the chapter on tendon injuries, no mention of pull out wires is given.

In wounds of the skin, in which one has a de-gloved finger, the author states that amputation is the treatment of choice. For a degloved thumb, it is the wrong treatment. The specific reasons are lucid and concise.

This book should be serviceable to those who are being trained in plastic or orthopedic surgery and also to those surgeons who, by obligation but not by choice, have occasion to treat hand injuries.

—Raymond Vespa, M.D., Odessa.

The Medical Assistant. Miriam Bredow. 477 pp. New York, McGraw-Hill Book Co., 1964. $7.95.

SINCE working in the office of a physician is "a world within it-

self," every medical assistant, especially the beginner, will be interested in reading the new second edition of Bredow's "The Medical Assistant." The author has touched on most of the regular duties required in the doctor's office, which are usually carried out by, or expected of, the medical assistant—such as helping with examinations, techniques of giving injections, application of physical therapy, preparation for x-ray, and laboratory procedures. In addition, there are brief discussions on secretarial duties, patient records, insurance in medical practice, and housekeeping hints. There is also an introductory chapter on personal adjustment and patient relations which should be required reading for every medical assistant.

It would be well for every doctor to include this new book in his medical library to acquaint new employees with the multiple problems that are unique only to such offices, and for old employees who might be developing a "stale attitude" toward their duties.

For the medical assistant contemplating the AAMA Certification Examination, this book briefly covers all sections, but is not sufficiently adequate for the inexperienced to pass the examination without more detailed study than can possibly be included in one book.

—Mildred R. Crawford, Immediate Past President, Texas Medical Assistants Association.

The First Twenty Years of the University of Texas M. D. Anderson Hospital and Tumor Institute. Staff of Department of Publications at M. D. Anderson Hospital. 564 pp.

THIS BOOK is unique in that its cover is an exact duplication of the pattern of the pink marble building itself. It is a publication that has much personal significance since I have been privileged to observe the developments from the beginning.

The early history of the development of cancer hospitals is reviewed with particular attention to the first one established at Rheims, France, along with a discussion of the reasons for its closing in 1778 due to social pressures. The Royal Cancer Hospital in London was established by William Marsden in 1891 followed by Memorial Hospital in New York City in 1884. The latter two are still actively contributing in education, training, and research in the cancer field.

Early figures in the development of a cancer program for Texas gives proper credit to such men as J. M. Martin of Dallas, J. E. Gilcreest, Dudley Jackson of San Antonio, and George W. Cox, Health Commissioner, among many others. A discussion of the legislative processes that entered into the founding of the M. D. Anderson Hospital and Tumor Institute is presented. Astute leadership has resulted in this Tumor Institute of the first class.

This book incubates a feeling of the efforts by everyone from the night watchman (see his trials and tribulations with soldiers, sailors, and their girl friends on the spacious grounds of the Baker Estate) to the Librarians (see her difficulties with cockroaches eating covers off the books in the early days) and the writers whose editorial copy was composed in a bathroom converted to secretarial space. The reviewer remembers a machine shop where lead protection safes were fabricated, and specialized instruments of various types were actually made. It took great fortitude to work under the original conditions and the vision of things to come as a stimulus to stay with it. The renovation of barracks from Camp Wallace provided an interim place for the beginning of pilot operations in the organization, and organization of patient care systems. Beginning research facilities were started here. The present facility was completed in 1954-1955 and in its construction much ingenuity was shown. For example, railway tank cars serve as storage tanks for radioactive wastes. The meal pack system by food wagons made it one of the first to use this method in the United States. A copy of a letter by Kay King is included that resulted in memorializing a room to Carroll Alan King by the institution's staff. This in itself indicates the strong feeling of dedication present. The attention paid by the staff to maintenance workers, technicians, secretaries, and workers of all types shows a refreshing atmosphere of consideration for all who carry on its work.

The institution's efforts in setting up a working cancer registry for the state indicates the scope of its activities. The M. D. Anderson group published the first book on chemotherapy of malignancies, and 14 institutions in the Southwest cooperate with its Chemotherapy Study Group. Tribute must be paid to the M. D. Anderson group for the First Yearbook on Cancer to be published annually. The numerous illustrations of the staff and workers at the M. D. Anderson Hospital unfolds the story of its activities in all its clinical and research activities. Balance, perspective, and long range planning are shown.

An inkling of the determination of the library staff in the collection and care of publications is well demonstrated by footnotes on the activities of Miss Bussey and Mrs. Mandeville on pages 248 and 249, and emphasizes the human interest side in medical history.

The Annual Symposium on Cancer Research culminating in the presentation of the Bertner Foundation Award brings to this area the most recent advances made throughout the world. It is always a privilege to attend. The Annual Clinical Conference likewise confers the same privileges for those who are interested more in patient treatment and care.

Publications of the cancer and heart bulletins received by Texas physicians emanate from this in-

stitutional beehive of activity. Color television for medical educational purposes made its debut at M. D. Anderson Hospital, also one of the first hospitals to install an efficient central transportation service.

"Statistical Accounting of the Total Patient Care Experience" is an important section, but the reviewer feels a lack of knowledge in the area to deal properly with it. It does represent an important phase of medical development.

The reviewer is pleased that Dr. T. W. Bonner cooperated with Dr. Grimmett in a predoctoral program for fellows in physics and that Dr. Shalek who now heads the department is a product of this program. Dr. Bonner and the reviewer in 1940 tried unsuccessfully to get a supervoltage set up at Hermann Hospital at the actual cost of construction but were refused. This is mentioned to emphasize the reviewer's awareness of the immense problems overcome and solved by Dr. Clark and his associates. The tribute to Dr. Randolph Lee Clark by Harry H. Ransom, Ph.D., as a medical educator and administrator with a concern for patient care, research and teaching far exceeds anything the reviewer might write. It is time, however, that the medical profession in this state does likewise.

—Joe C. Rude, M.D., Austin.

Recent Deaths

DR. M. P. DISHON

Dr. Margaret Peoples Dishon of Blanco was killed in an automobile accident July 22 en route to her clinic to answer an emergency call.

Dr. Dishon, 40, with her husband, Dr. Neil H. Dishon, had operated the clinic in Blanco since 1962.

She was born June 22, 1924, in Cleveland, Ohio, the daughter of Mr. and Mrs. A. G. Peoples. She held the B. A. and M. A. degrees from Western Reserve University in Cleveland, three years' work toward the Ph.D. at George Washington School of Medicine in Washington, D. C., and the M. D. from Ohio State University, Columbus, Ohio. She had interned at St. Rita's Hospital in Lima, Ohio, and served a six months' residency in general practice at St. Joseph's Hospital in Flint, Mich.

Before moving with her husband to Blanco, she had practiced at the Dependent Clinic at Kelly Air Force Base, San Antonio, and in San Antonio.

She was a Roman Catholic.

She was a member of Bexar County Medical Society, Texas Medical Association, and the American Medical Association. She was a member of the National Speakers Bureau of the AMA.

Married on Jan. 4, 1950, in Newport, Ky., the Drs. Dishon became the parents of three daughters, Carla Dishon, Dene Dishon, and Susan Dishon, and a son, Bruce R. Dishon,

DR. M. P. DISHON

all of whom survive their mother. With her husband and children, other survivors are her parents of Cleveland and her grandmother, Mrs. Blanche Peoples of Cleveland.

Dr. J. M. DONALDSON, SR.

Dr. James M. Donaldson, Sr., former Bonham physician, died in San Antonio on May 30. His health had been poor for several years, and he had retired from practice and moved to San Antonio to live with his son.

DR. J. M. DONALDSON, SR.

He was elected to inactive membership in Texas Medical Association in 1960.

Dr. Donaldson was 81. He was a graduate of the College of Physicians and Surgeons in St. Louis in 1906 and had done postgraduate work at Tulane University of Louisiana School of Medicine in New Orleans.

He began the practice of medicine in Dodd City, Tex., in 1906. In 1935 he moved to Bonham.

He was a member of the Bonham Chamber of Commerce, past president of the Bonham Rotary Club, and a deacon in the First Baptist Church of Bonham. He considered as a hobby the raising of fine Hereford cattle.

He was secretary of the Fannin County Medical Society for ten years.

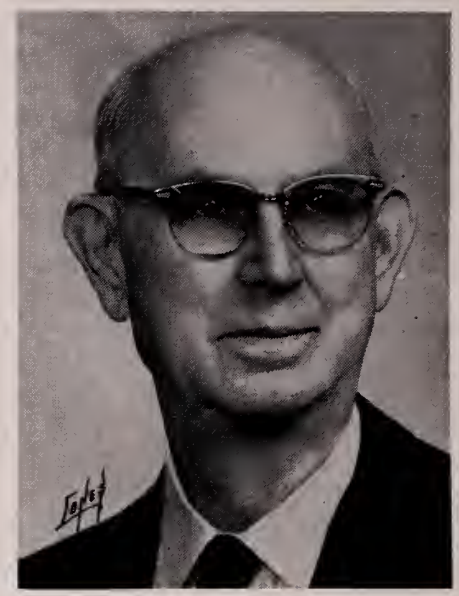
His wife was the former Rosa McLaurin of Meridian, Miss. They were married there on June 19, 1912. She died in May, 1948.

Dr. Donaldson is survived by a son, Dr. James M. Donaldson, Jr., of San Antonio, and a daughter, Mrs. Clyde Crittenden of Media, Pa.

DR. W. F. SHEPHERD

Dr. Walter Franklin Shepherd of San Antonio died June 6 of congestive heart failure resulting from arteriosclerotic heart disease. He was 82.

A graduate of Baltimore Medical College and Tulane University, Dr. Shepherd practiced medicine for 59 years in Bexar County.



DR. W. F. SHEPHERD

He was a member of the Bexar County Medical Society, Texas Medical Association, and American Medical Association.

He was a life deacon of the First Baptist Church of San Antonio, a life member of the Texas Lodge No. 8, AF & AM, and of the Alzafar Shrine.

For outstanding services rendered the nation during and after World War II, Dr. Shepherd received a special commendation and medal from President Dwight D. Eisenhower.

He is survived by his wife, Mrs. Myrtle B. Shepherd; two daughters, Mrs. Oudia Smith, Mrs. Dorthea Waitz; stepdaughter, Mrs. Doyle W. Dodd, all of San Antonio; four grandchildren; two stepsons, John E. MacDonald, Albuquerque, N. M., and L. Lloyd MacDonald, Midland; a brother, John W. Shepherd, San Antonio, and a sister, Mrs. J. H. Scoggin, Jacksonville, Tex.

DR. J. S. PEEK

Dr. John Sealy Peek, general practitioner, died May 24 in Houston from tumor of the brain. The 57-year-old physician resigned his position on the staff of Rusk State Hospital, Rusk, more than a year ago because of his health.

Named for his father's friend, banker-oilman-philanthropist John Sealy, Dr. Peek was a native of Galveston. He was the captain of the University of Texas' first golf

team in 1926 and won a Southwest Conference Golf Championship. He received degrees from the University of Texas and University of Texas Medical Branch. He interned in Galveston at John Sealy Hospital, and later did postgraduate work at the University of Pennsylvania.

His wife is the former Virginia Harriss of Galveston. The couple were married there on April 26, 1934.

Dr. Peek practiced in Brownsville for six years, and served in the U. S. Navy during World War II with the rank of commander. He set up practice in the Houston area in 1947 and joined the staff at Rusk State Hospital in 1958.

He was a member of Kappa Sigma social fraternity and Phi Rho Sigma medical fraternity.

He belonged to Cherokee County Medical Society and Texas Medical Association.

His hobby was golf, and he was a Roman Catholic.

He is survived by his wife; a daughter, Leslie; a son, Kimball, all of Houston; and two brothers, Richard of Galveston and Ben of Hitchcock.

DR. G. J. KOCHEVAR

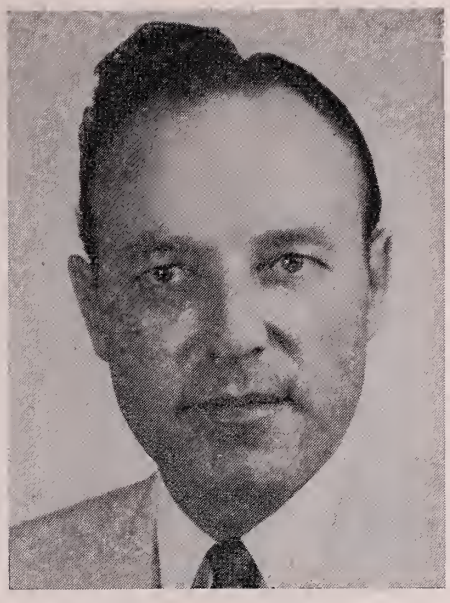
Dr. Gerald J. Kochevar, 46, of Midlothian died July 12 in a Dallas hospital where he had undergone surgery for removal of a lobe of his lung.

Born in Buhl, Minn., Feb. 13, 1918, Dr. Kochevar came from a family of physicians. He received the B.A. in 1943 and M.D. degree in 1945, both from the University of Kansas. He interned at Milwaukee County General Hospital in Milwaukee, Wis. He spent two years in the Army and moved to Midlothian in 1948 to begin his practice.

He was a past president of the Ellis County Medical Society and a member of the Texas Medical Association, American Medical Association, the American Academy of General Practice, and a staff member of the Waxahachie Sanatarium.

He was a Roman Catholic.

Survivors include his widow, Catherine, whom he married April 24, 1952, in Dallas; two sons, Ronald Kochevar of Waukesha, Wis., and Gerald John Kochevar, II, of Midlothian; his mother, Mrs. Frances Kochevar of Buhl, Minn.; and three brothers, William R. Kochevar of Red Wing, Minn., Russell



DR. G. J. KOCHEVAR

Kochevar of Hammond, Ind., and James Kochevar of Clawson, Mich.

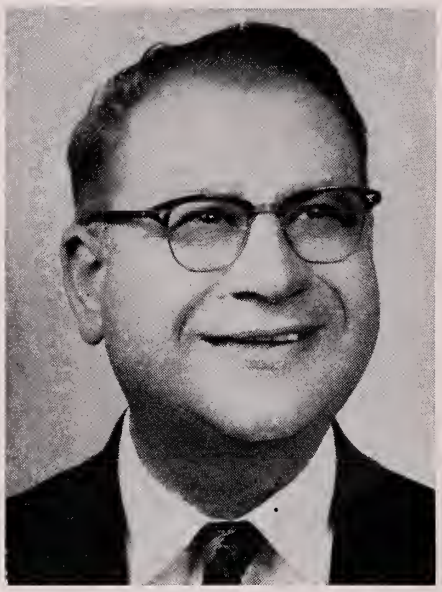
DR. F. A. MUSACCHIO

Dr. Frederick Alfred Musacchio died in Laredo May 15 of myocardial infarction. For the past seven years he had been director of the Laredo-Webb County Health Department.

Dr. Musacchio was born Nov. 8, 1906, in Rahway, N. J., the son of Pasquale and Antonietta Caporicci Musacchio. He graduated from high school in Elizabeth, N. J., and completed his premedical work at Rutgers University, New Brunswick, N. J., in 1929 when he received a bachelor of science degree. He earned his M.D. degree at the Rush Medical College, University of Chicago, in 1934.

He left medical school to enter a general rotating internship at Providence Hospital in Detroit, and after that at North Hudson Hospital, Weehawken, N. J. He served his residency in the tuberculosis division of the Herman Kiefer Hospital in Detroit. Throughout his career he continued his education through postgraduate work undertaken at several institutions including Northwestern University, University of Texas Medical Branch, City Hospital in Cleveland, Ohio, and the University of Michigan. In 1939 the University of Michigan conferred on him the degree of master of science in public health.

Early in his career he spent a year in gen-



DR. F. A. MUSACCHIO

eral practice in Detroit. After the additional training which prepared him to specialize in public health administration, he became director in 1940 of the Acadia Parish, La., Health Unit. He moved on to direct health units in St. Mary Parish, La., St. Joseph County, Mich., Hammond, Ind., and Laredo-Webb County, Tex.

Dr. Musacchio was the author of eight medical articles on the subjects of tuberculosis, diphtheria, public health history, endemic typhus, venereal disease education, and administration.

On July 8, 1942, he married Eugenie Jaschke of Crowley, La., in Crowley.

He was a member of the Webb-Zapata-Jim Hogg Counties Medical Society, Texas Medical Association, American Medical Association, Texas Public Health Association, American Public Health Association, American Trudeau Society, American Social Hygiene Association, and the United States-Mexico Border Public Health Association.

He served as first lieutenant in the Army reserves.

Dr. Musacchio's hobbies were varied and included photography, music, art, travel, and the study of German, French, Italian, and Spanish.

He belonged to the Laredo Lions Club, Laredo Civic Music Association, Laredo French Club, Elks Lodge, Laredo Chamber of Commerce, Laredo-Webb County Tuberculosis Association, and Cristo Rey Church.

He is survived by his wife, now of Crowley, La.; his daughter, Eugenie A. Musacchio of Crowley, La.; three brothers, Frank Musacchio and Dominic Musacchio of Elizabeth, N. J., and Louis Musacchio of Paterson, N. J.; and two sisters, Mrs. Frank Fillat and Mary Musacchio of Elizabeth, N. J.

DR. R. C. ROWELL

Dr. Robert Columbus Rowell, 59, superintendent of Terrell State Hospital, Terrell, died in a Dallas hospital after a short illness on July 11.

Former president and active as an alternate delegate of the Kaufman County Medical Society at the time of his death, Dr. Rowell was also a member of the Texas Medical Association, the American Medical Association, Texas Neuropsychiatric Association, and past president of the Dallas Neuropsychiatric Society. He was also a fellow of the American Psychiatric Association and a fellow of the American College of Hospital Administrators.

Born in Anson, Sept. 10, 1904, Dr. Rowell graduated from Anson High School. He attended Hardin-Simmons University for two years, then went to Baylor University College of Medicine, where he received his M.D. in 1930.

His state hospital career began at San Antonio State Hospital in 1930. From 1937 to 1941 he served as assistant superintendent of Rusk State Hospital. From 1941 to 1949, he was a psychiatrist at Wayne County General Hospital, Eloise, Mich. In 1949 he returned to Texas as superintendent of Abilene State Hospital. In June, 1950, he was named superintendent of Austin State Hospital.

Dr. Rowell came to Terrell as superintendent of Terrell State Hospital in February, 1953, and served in that capacity until his death.

Up to the time of his illness, Dr. Rowell was active in the Lions Club, having been a past president, director of Greater Terrell United Fund, and as a director of Terrell Chamber of Commerce. He was a Lutheran. A project for a chapel of all faiths at the state hospital was started during Dr. Rowell's tenure as superintendent.

Survivors include his wife, Willie Mae Hansen Rowell of Terrell, two daughters, Janice and Elizabeth, three granddaughters, and his mother of Meridian.

COMING MEETINGS

American Medical Association, Clinical Meeting, Miami Beach, Fla., Nov. 29-Dec. 2, 1964. Annual Meeting, New York City, June 20-24, 1965. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 19, 1964; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

SEPTEMBER

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 10-12, 1964. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Medical Association Congress on Occupational Health, Houston, Sept. 26-27, 1964. Dr. Henry F. Howe, 535 N. Dearborn St., Chicago, Sec.

Southwest Regional Cancer Conference, Fort Worth, Sept. 25-26, 1964. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Texas Heart Association, Dallas, Sept. 25-27, 1964. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, Houston, Sept. 26-27, 1964. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Pediatric Society, Dallas, Sept. 24-26, 1964. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Blackford Memorial Cancer Lectures, Denison, Sept. 13, 1964. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

OCTOBER

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, New York, Oct. 24-29, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, New York, Oct. 25, 1964. Dr. Robert A. Peterman, 3 Craig Court, Totowa Boro, N. J., Sec.

American College of Physicians, Los Angeles, Oct. 8-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Sept. 29-Oct. 2, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 5-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Medical Women's Association, New York, Oct. 16-20, 1964. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 10-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

Association of American Physicians and Surgeons, Inc., New Orleans, La., Oct. 8-10, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

International College of Surgeons, Mexico City, Oct. 6-11, 1966. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Zigmund Kosicki, 1220 North Stanton St., El Paso, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Association for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Surgical Society, San Antonio, Oct. 4-6, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, San Francisco, April 12-15, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, New York City, June 17-21, 1965. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American Congress of Physical Medicine and Rehabilitation. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Boca Raton, Fla., Feb. 25-March 2, 1965. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society, New York, May, 1965. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Hospital Association. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Neurological Association, Atlantic City, N. J., June 14-16, 1965. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Chicago, Nov. 6, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., See.

Medical Society of United States & Mexico, Phoenix, Ariz., Dec. 9-12, 1964. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Radiological Society of North America, Chicago, Nov. 11-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum, New Orleans, La., March 14-16, 1965. Dr. W. P. Coleman, 1828 Calhoun St., New Orleans, La., Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, Ameriean College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, Waco, Nov. 8, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. Leonard R. Robbins, 3618 Yoakum, Houston, Sec.

Texas Division, American Cancer Society, San Antonio, November, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedie Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. Charles Peterson, 1333 Moursund, Houston, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, San Antonio, May 1-2, 1965. Dr. Fred P. Thomas, 2342 Maroneal, Houston, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society on Aging, Clinical Medicine Section, San Antonio, April 29-May 2, 1965. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc., Houston. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 7-9, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, See.

United States-Mexico Border Public Health Association, Los Angeles, April 26-29, 1965. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 9-10, 1965. Dr. J. E. Albright, 2481 Medical Center, Corpus Christi, See.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

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FOR Amendment 3 On November 3

THE KERR-MILLS program has operated with great success in Texas for nearly three years to provide hospital, medical, surgical, radiation, and nursing home benefits for those on Old Age Assistance. Some 30 percent of those 65 and older in our state are OAA recipients. Recently, Blue Cross, from which the State Department of Public Welfare purchased group insurance for persons on OAA, reported that during the past year 89,391 Texans were admitted to hospitals under the Old Age Assistance program. These elderly men and women stayed an average of 9.3 days. The total hospital and professional benefits paid out by Blue Cross over the past 12 months for these people amounted to $21,773,209. All of us may be justly proud of our present medical care program for the aged.

However, there is at hand an opportunity to enlarge this program by establishing the second part of the Kerr-Mills plan, called Medical Aid for the Aged. MAA is designed to provide services for about 3 percent of all elderly Texans who still need and want outside help in meeting health care costs.

On Nov. 3, Texas voters will decide whether to include more senior citizens with those who may receive medical care paid for from public funds. Appearing third on the ballot in a list of proposed constitutional amendments will be one which would permit the legislature to authorize vendor payments for medical care for needy individuals 65 and older. These payments would be made for needed medical services to those elderly persons unable to pay for such care and who are not eligible for Old Age Assistance.

The Texas Medical Association is officially supporting passage of this amendment. Once approved by the voters, it will enable the State Legislature to implement the second phase (MAA or Medical Aid for the Aged) of the Kerr-Mills program.

The November election will provide us with an

opportunity to complete an outstanding program for the needy aged of our state. I hope that you will encourage your patients and friends to "Vote FOR Amendment 3 on November 3."

—JOHN M. SMITH, JR., M.D., San Antonio,
Chairman,
Council on Medical Jurisprudence.

Coronary Artery Disease In the Diabetic Patient

ABOUT 70 to 80 percent of diabetic patients die of vascular disease of the heart, brain, and kidneys whereas some 50 percent of total deaths are caused by atherosclerotic coronary artery disease alone. Prevention or delay of atherosclerosis in diabetics is probably best accomplished by avoiding obesity and by careful continuous control of diabetes.

Studies on the metabolism of the myocardium in even mild diabetes show that utilization of glucose, lactate, and pyruvate is reduced with even moderate hyperglycemia, but their utilization is restored to, or towards, normal by returning the blood sugar to normal with small doses of insulin.

Extreme hyperglycemia, sometimes associated with acidosis or coma, is a frequent complication of acute myocardial infarction even in previously well-controlled diabetics. Hypoglycemia deserves respect and in cardiac patients may cause complications such as angina, tachycardia, arrhythmias, congestive failure, or myocardial infarction. Even though hypoglycemia should be avoided, fear of hypoglycemia does not justify withholding insulin necessary for reasonable control of diabetes. Acidosis is, of course, an absolute indication for the use of insulin, often in large doses. Diabetic acidosis can itself precipitate myocardial infarction.

Insulin is also indicated in patients who have coronary artery disease or acute myocardial infarction with uncontrolled diabetes manifested by hyperglycemia without acidosis. Oral agents might be successful especially if no acute stress or infarction is present.

To avoid hypoglycemia, it is usually safe to aim for blood sugar levels of about one and one-half times normal, or perhaps 150 mg. per 100 ml. Once reasonable control of the diabetes has been attained, if better control seems possible without risk of hypoglycemia, it should be attempted. Often better or even ideal control can be obtained without significant risk of hypoglycemia.

The mortality rate in diabetics with myocardial infarctions is higher than in nondiabetics, but when patients with uncontrolled diabetes are excluded, the mortality rates are comparable.

In conclusion then, hypoglycemia should be avoided in all patients with diabetes, and especially in those with coronary artery disease, but one should not allow fears of hypoglycemia, even in patients with symptomatic coronary artery disease, to be exaggerated so that reasonable control of diabetes is sacrificed.

—JOHN W. CHRISS, M.D., Corpus Christi,
President, Texas Diabetes Association.

Renaissance of The Electrocardiogram

A DECADE AGO electrocardiography had a decline in many scientific circles. By that time, the method was relatively old, and it lacked the dramatic advances being made in such fields as hemodynamics and phonocardiography. Few papers on electrocardiography were given at scientific meetings. The electrocardiographer was criticized for relying too much on empiricism and on his past experiences. He was unable to satisfactorily translate his impressions into acceptable mathematical data, even though he could measure intervals, amplitudes, and electrical axes. The method and the science tended to be discredited by such clichés as, "After all, the heart is primarily a pump, not an electrical generator."

The heart's main function is *obviously* that of a pump. But it is an *electric* pump, and such a pump must have a proper supply and distribution of electricity to function. The importance of these electrical aspects of the heart has recently been emphasized by developments in cardiac monitoring, in cardiac pacemaking, and in electrical defibrillation and electrical conversion.

Fortunately, the science is now advancing at an increased rate. Electrocardiographic diagnoses have been placed on a more rational basis by vectorcardiography, intracardiac electrodes, and experimental work in animals. Correlation of electrocardiograms with hemodynamic data, phonocardiography, and surgical and pathologic findings has resulted in more valid interpretations. Steps have been taken toward computer study, although it is doubtful that the human element in tracing analysis can ever be entirely replaced by a machine. In scientific meetings and publications, more and more time and space are devoted to the new aspects of electrocardiography.

Actually, the renaissance has little meaning to the physician who treats patients, for as far as he is concerned the electrocardiogram had no decline. He realized all along that electrocardiography had limitations, and that it would never substitute for sound clinical judgment. It stood by him through the years as a valuable laboratory procedure, which he could correlate with his clinical findings. It repeatedly demonstrated its value to him in the diagnosis and management of the various types of heart disease. It enabled him to precisely recognize and treat cardiac arrhythmias, in which field it has no peer. It revealed evidences of chamber hypertrophy, conduction disturbances, myocardial infarction, and ischemia. It helped him to detect and manage electrolyte disturbances.

The test of time is the ultimate measure of the value of any procedure or therapy. The electrocardiogram has stood this test well. Recording instruments are now indispensable equipment in all physicians' offices. More tracings are taken each year in all hospitals. Courses in electrocardiography are increasingly popular, and house officers and medical students insist on electrocardiography as part of their basic training.

There can be no antagonism between the various disciplines of cardiac investigation. Progress in one ultimately leads to progress in the others. It requires clinical judgment to determine if and when each is needed, and in the end, all need to be correlated. But the electrocardiogram is and will continue to be one of the first laboratory steps in cardiac evaluation, after the history and physical examination. Few other procedures can match its simplicity and its value.

—MILTON R. HEJTMANCIK, M.D., Galveston.

St. Louis Encephalitis And Health Officers

ELSEWHERE in this issue is a report of the Houston director of health on the epidemic of St. Louis encephalitis (SLE) that plagued Houston and Harris County this past summer. It is a simple, factual account of the detection of an epidemic of arbovirus encephalitis by a qualified professional health officer and of how his conclusions were verified by a professional state health laboratory officer. It is a simple account but a fascinating story of the actions of two veterans, both highly trained, widely experienced health officers who in a never-ending battle constantly detect, confirm the presence of, and stamp out disease. Every biological scientist should take pride in their accomplishments.

Dr. Charles A. Pigford, veteran Texas health officer and president of the Texas Public Health Association, carefully reviewed mortality versus morbidity statistics for Houston. With the first report of six cases and three deaths he assumed there must be arbovirus encephalitis in Houston. Immediate demands for information concerning unreported cases, plus a review of deaths reported as required by law, supplied evidence that viral encephalitis was present. He placed paired sera in the hands of J. V. Irons, Sc.D., Director of Laboratories, Texas State Department of Health, who confirmed his deductions by careful laboratory analyses. Thus did two professionals in public health establish unquestionably the presence of an SLE epidemic before a significant number of case reports were sent to the local health department.

Texas medicine is fortunate to have these dedicated and competent health men. National recognition has and will come to them. Let us add our thanks, admiration, and good will.

Although authorized by the Legislature in 1947, a school of public health and environmental medicine does not exist within our state boundaries. These—and other dedicated health officers—must have training outside our boundaries and be imported to work in Texas. Perhaps it is time to assess our assets—or lack of them—and plan for the future.

—JOHN RANDOLPH HALL, M.D., M.P.H., Houston.

Sam Thompson Loan Fund

TEXAS MEDICAL Association is unique in many ways. One is the large size of its student loan program. This was made possible by the generosity of the late Dr. and Mrs. Sam Thompson of Kerrville.

"Dr. Sam," as he was lovingly called by those who knew him, was born in Claiborne Parrish, La. When he was ten, he moved with his parents to Arkansas where he attended public schools. He later enrolled in the Kentucky School of Medicine, but had to teach school on alternate years to finance his education, delaying his graduation until 1904.

He practiced medicine in El Dorado, Ark., until 1911 when he developed tuberculosis. At this time he moved to San Angelo to regain his health. In 1913, he became medical director of the Texas State Tuberculosis Sanatorium at Carlsbad, Tex. (later McKnight State Hospital).

In 1917 he moved to Kerrville, bought a ranch on the outskirts, and converted the ranch house to an eight-bed hospital for tuberculosis. This grew finally into a 100-bed institution that attracted patients from all over the United States. Dr. Sam had studied abroad and was considered an authority in his field. He was president of TMA in 1953-1954, and received many high honors at both the state and national levels. He worked hard and invested wisely.

As he neared his last years, he considered several benevolent endeavors that he might endow, but remembering his difficulties in financing his own medical education, he finally decided to put his life savings into a student loan program. This was set up for needy medical students at the University of Texas medical schools under the administration of the Board of Trustees of the Texas Medical Association.

The first loans were made in 1959, and to date there have been made a total of 341 loans amounting to $145,000.

The student is eligible to receive a total loan of $3,000, that is, $1,000 per year beginning with his second year. Under the terms of the will, a straight 4 percent interest is charged until payment which begins on the fourth year after graduation, 10 percent the first year, 20 percent the second year, 30 percent the third year, and 40 percent the fourth year.

Group coverage life insurance has been purchased from Great American Life Insurance Company of Dallas at $0.41 per $1,000. Premiums are paid by the Fund at no extra expense to the borrower who is issued a certificate of insurance in the amount involved.

The total assets of the Fund now exceed $1,000,000. The annual income from investments and interest on loans is in excess of loans made each year.

Repayments will begin this year amounting to $375. In 1965, anticipated payments will exceed $1,500; in 1966, about $4,000; and over $10,000 is expected in 1967. At this rate, it becomes apparent that this Fund has great potential.

Many are aware of the gift of the Sam Thompson Room in the Headquarters Building, and also that Texas Medical Association recently has received the corpus of Mrs. Sam Thompson's estate which, after certain obligations, has been pledged to the support of the TMA library.

It probably never occurred to Dr. and Mrs. Thompson that their generosity was outstanding. They possibly saw it simply their duty to give back to medicine what it so generously had given to them.

—BYRON P. YORK, M.D., Houston.

Why Belong to the AMA?

AT LAST COUNT 93 percent of the total membership of Texas Medical Association belonged to the AMA. This is a good record but what about the other 7 percent? Nationally, approximately 73 percent of the nation's 276,000 physicians are AMA members. This leaves about 75,000 physicians who should be members of the AMA. This additional membership could bring in over 3 million dollars in dues.

Nonmembers give many excuses for not joining the AMA. Some say they disagree with the philosophy of the AMA and believe that it is becoming too political in its activities. Others say it does nothing for the individual doctor, and others object to paying dues at the national level. The most common reason probably is simply lack of interest in medical organization. This includes those who never attend their local society or state association meetings, and refuse to participate in any medical activities.

It might be valuable to remind all physicians of a few of the basic accomplishments of the AMA and of its services to individual physicians so that each can judge for himself whether his membership is worthwhile.

Each member receives the *Journal of the American Medical Association,* one specialty journal of his choice, a reception room copy of *Today's Health* plus *The AMA News,* a fortnightly newspaper. The subscription rates of these publications average about $37 per year.

According to the AMA constitution the objectives of the Association are "to promote the Science and Art of Medicine and the betterment of Public Health." Accomplishments along these lines have been too numerous to mention here but a few will be discussed briefly.

The availability of the publications discussed provides a member with a voluminous amount of scientific material plus services such as scientific information on new drugs, physician placement, management information, postgraduate courses, and medical films on loan.

The AMA promotes the nation's health in numerous ways along with its state and county societies, by encouraging more people to use seat belts in autos, calling for protection of high school athletes from sports injuries, warning parents about dangers to children from plastic bags; teaching physicians and the public to recognize and treat alcoholism as a medical problem, promoting legislation designed to secure proper labeling of potentially dangerous household chemicals and educating the public on proper use to avoid poisonings, recruiting more students into medical careers, and educating the public about the foolishness of food faddism and the dangers of medical quackery.

The AMA has councils which deal with many problems, and as a result of the enormous amount of work done by these councils the AMA exerts a tremendous influence toward the betterment of medical education and the availability of continuous postgraduate training. The AMA also serves as a clearinghouse for information on state legislation of medical interest, and analyzes medical legislation introduced in Congress, keeps up-to-date records on all physicians, and offers scores of other services designed to keep the physician abreast of the times and to help him practice the best medicine.

It is difficult to understand how a physician who earns his livelihood by the American system of the practice of medicine can refuse to invest a portion of his time and money toward the preservation of that system. If an individual disagrees with some of the activities and philosophy of the AMA he may join and express his views where they will be heard by his fellow doctors. He may even contribute a good idea.

—E. P. Hall, Jr., M.D., Fort Worth,
Alternate Delegate to
American Medical Association.

letters to TEXAS MEDICINE

Amendment 1 Advocated

In addition to Amendment 3 which is being submitted to Texas voters for approval on November 3, there is another important proposal which affects doctors, their families and patients as citizens of this state.

The Texas Medical Association long has supported wise and prudent investment of monies which it has been fortunate enough to receive. Likewise it believes in these principles in the expenditure of public funds.

I would like to urge doctors and their friends to support the State Board of Education and vote for Amendment 1 which will assist the Board, charged with the investment of income for Texas schools through the Permanent School Fund, to maintain a perpetual fund for our schools.

Although the Constitution adopted in 1876 declares the fund to be a perpetual one, an amendment adopted in 1891 permitted the Legislature to transfer in any one year not more than 1 percent of the principal to the Available School Fund for application upon current expenses. Since 1891 there have been few transfers, but within the last decade approximately $20 million of the principal has been transferred and spent.

I strongly concur with the State Board of Education that the fundamental concept of prudent investment of trust funds requires that principal be maintained intact and only the income from that principal should be applied to current expenses.

Please vote for Amendment 1 on November 3. This amendment will protect this trust fund from future dissipation by prohibiting further transfers of principal and it will not deprive the Available Fund of transfer of income as it is earned. Thus we can safeguard funds for the education of our children.

Sincerely,
JOHN M. SMITH, JR., M.D.,
San Antonio.

Poverty Among Abundance

Our attention is called to the problem of the migrant workers in the current issue of the *Texas State Journal of Medicine*, September, 1964, page 764, "Help for Migrant Harvesters" and pages 774-775, "Negro Woman Doctor Serves Migrants."

The articles point out the abject poverty in the midst of great abundance.

In recent years, quite a few people have become aware of the migrant worker. For instance Michael Harrington in the book "The Other America" (Poverty in the United States), Chapter 3, reviews the subject. In the appendix of his book, Mr. Harrington interprets poverty as follows:

> Poverty should be defined in terms of those who are denied the minimal levels of health, housing, food, and education that our present stage of scientific knowledge specifies as necessary for life as it is now lived in the United States. Poverty should be defined psychologically in terms of those whose place in the society is such that they are internal exiles who, almost inevitably, develop attitudes of defeat and pessimism and who are therefore excluded from taking advantage of new opportunities.
>
> Poverty should be defined absolutely, in terms of what man and society could be. As long as America is less than its potential, the nation as a whole is impoverished by that fact. As long as there is the other America, we are, all of us, poorer because of it.

Sincerely yours,
KURT LEKISCH, M.D.,
Midland.

Educational Program On Health Care Of the Aged

Once again we members of the Texas Medical Association have an excellent opportunity to present a strong united front.

An intensive national educational program for health care for the aged is being sponsored and financed by the American Medical Association this month. State and county organizations have been urged to plan appropriate programs to coincide with the national observance of Community Health Week, Oct. 18-24.

Purpose of this educational program, called "Health Opportunity Program for the Elderly", is to inform the public of the broad range of health care now available to the elderly who are unable to pay for it. This strong, positive approach is invaluable. It places emphasis upon the already existing health care programs under Kerr-Mills, which are far better than the Medicare tax bill. The AMA has a suggested program which includes educational advertisements for newspapers, information pamphlets for distribution to the public, posters and speeches.

Each county society should join wholeheartedly in this comprehensive educational program. The county society is the logical group to carry out this program because local members have the knowledge and ability to select the proper daily or weekly newspapers. The county society should assume as much of the cost of this program as possible. Allied professions and business men should be encouraged to sponsor additional advertisements in local papers.

In this second phase of "Operation Hometown", we must tell our story in strong and able words, giving the public the true picture of the health care already available to the aged.

Let us unite in an educational program to carry our message on medical care for the aged.

Maf E Johnson

President
Texas Medical Association

Total Valve Replacement For Acquired Valvular Heart Disease

Results in 171 Patients

RESEARCH & INVESTIGATION

TOTAL REPLACEMENT of the destroyed heart valve with a ball-and-seat prosthesis when medical management or previous surgery has failed is currently the most acceptable method of management for acquired disease of the heart. This study reviews an experience during 21 months with 171 patients having the mitral or aortic valve replaced either with the Starr-Edwards[9,11] prosthesis or a sutureless type of ball valve developed by Magovern.[7]

Total replacement was reserved for mitral disease causing dense calcification or loss of valve substances and when transventricular commissurotomy[4] or annuloplasty was not considered practical. All patients undergoing operation for acquired aortic valve disease had total replacement because reconstruction of this valve generally has proved unsatisfactory. Patients selected for valve replacement were in Class III or IV range of cardiac function and were evidencing progressive cardiac deterioration. Many had intractable cardiac decompensation manifested by unrelenting pulmonary congestion, hepatomegaly, or even ascites, despite intensive medical management.

Previous reports of a more preliminary nature from this center[3,8] emphasized surgical considerations and techniques in more detail. This study was concerned chiefly with factors influencing early and late mortality and results obtained 6 to 21 months after valve replacement.

From the Cora and Webb Mading Department of Surgery, Baylor University College of Medicine, and the Surgical Service of St. Luke's Episcopal Hospital, Houston, Texas.

Material and Methods

Between July 1, 1962, and April 1, 1964, 171 patients underwent mitral or aortic valve replacement with Starr-Edwards ball-and-seat valves or Magovern sutureless ball-and-seat valves. The age range in the 113 males and 58 females was from 13 to 72 with the majority between 40 and 60 years of age (Table 1). Sixty-three patients had mitral valve replacement, and 106 had aortic valve replacement, while 2 patients had both valves replaced at the same operation. Six patients undergoing valve replacement had an additional procedure, such as valvotomy performed on a secondarily involved valve.

TABLE 1.—*Age Range in Mitral and Aortic Valve Replacements.*

Age Range (Decades)	Type of Valve Involvement			
	Mitral	Aortic	Combined	Total
10-19	1	3	1	5
20-29	7	12	--	19
30-39	16	16	1	33
40-49	12	29	--	41
50-59	19	26	--	45
60-69	8	18	--	26
70-79	--	2	--	2
Total	63	106	2	171

TABLE 2.—*Etiology of Valve Disease in Mitral and Aortic Valve Replacement.*

Etiology	Valve Replacement				
	Mitral	Aortic	Combined	Total	%
Rheumatic fever	42	50	2	94	55.0
Calcific (No RF)	11	20	--	31	18.1
Congenital	--	8	--	8	4.7
Luetic	--	4	--	4	2.4
Traumatic	--	2	--	2	1.1
Undetermined	10	22	--	32	18.7
Total	63	106	2	171	100.0

The authors review the experience of 21 months during which time 171 patients had either the mitral or aortic valve replaced by the Starr-Edwards prosthesis or a sutureless type of ball valve developed by Magovern. Results of the procedures indicate worthwhile advances in valve replacement.

DENTON A. COOLEY, M.D.
THOMAS G. NELSON, LT. COL. MC, USA

Most patients had previous rheumatic fever attacks and virtually all patients under 30 years requiring valve replacement had repeated attacks or episodes of bacterial endocarditis (Tables 1 and 2). All patients gave a history of prolonged pulmonary or hepatic congestion, disabling cardiac arrhythmias, intractable angina or syncopal attacks (Table 3). These findings in various combinations placed 109 patients (64 percent) in Class III and 62 patients (36 percent) in Class IV (Table 4). Class II patients were not considered sufficiently disabled to warrant valve replacement. Electrocardiographic, hemodynamic and roentgenographic contrast studies further substantiated the severe cardiac functional impairment present in all patients. Physiologic data obtained by heart catheterization (Tables 5 and 6) established that disability was related predominantly to valve destruction rather than to primary vascular, coronary, myocardial, or pulmonary disease. Catheterization measurements were also of special value in correlating the surgical risk of mitral patients with elevated pulmonary artery pressures (Table 5) and for delineating the major disease site in those patients (31

TABLE 3.—*Clinical Data in Mitral and Aortic Valve Replacement.*

	Mitral	Aortic	Combined	Total	%
Total Patients	63	106	2	171	
Number having:					
Left heart failure	63	94	2	159	93.0
Right heart failure	23	16	1	40	23.3
Angina, syncope, arrhythmias	31	40	1	72	42.1
Multiple valve involvement	21	30	2	53	31.0
Coronary artery disease	10	35	0	45	26.3
Previous surgery	23	15		38	22.2

TABLE 4.—*Cardiac Function in Mitral and Aortic Valve Replacement: Relationship to Mortality.*

	Mitral	Aortic	Combined	Total	%	Died	% Deaths
Class III Group	40	69	_	109	64.0	14	12.84
Class IV Group	23	37	2	62	36.0	18	29.03
Total	63	106	2	171		32	18.71

TABLE 5.—*Catheterization Data in 50 Mitral Valve Replacement Patients.*

Pulmonary Artery Pressure* (mm. Hg)	No. of Patients	No. of Deaths
Systolic		
100 +	4	3
75-100	16	8
50-75	20	3
25-50	10	1

*Resting stage, breathing room air.

TABLE 6.—*Catheterization Data in 60 Aortic Stenosis Patients Undergoing Valve Replacement.*

Systolic Pressure Gradient (mm. Hg)	No. of Patients
120 +	8
100-120	22
80-100	26
60-80	4

percent) with involvement of more than one valve.

Patients selected for surgery generally were not responding satisfactorily to medical treatment and more than 20 percent had recurrent incapacity after previous valve surgery. No patient was refused treatment because of poor myocardial reserve or disease of the coronary or pulmonary vessels when physical and catheterization data indicated a destroyed, malfunctioning valve.

A simplified technique of cardiopulmonary bypass[1, 2] utilizing a disposable oxygenator and five percent dextrose prime under normothermic conditions was employed for all valve replacements. Previous reports[3, 8] described in more detail the application of this bypass method for valve replacement. A median sternotomy was employed for aortic valve exposure and either a right or left sternotomy was employed for approach to the mitral valve. At the initiation and termination of cardiopulmonary bypass, careful venting and refilling techniques prevented the occurrence of air embolism. Carbon dioxide spray was used to flood the field as further precaution against air embolism. During either type of valve replacement, efforts were made to keep the heart contracting quietly at a near normal rhythm by perfusion of the myocardium with oxygenated blood. In some aortic replacements myocardial perfusion was accomplished by cannulating the coronary ostia whereas in mitral replacements, the aortic clamp was opened frequently to provide myocardial perfusion. After excision of the diseased valve, the Starr-Edwards prosthesis was sutured to the recipient tissue with interrupted 2-0 Dacron mattress or figure-of-eight sutures.

In 35 recent aortic valve replacements, the Magovern sutureless ball valve, which employs multiple metallic pins for fixation, was utilized, with resultant simplification of insertion and lessening of bypass time. In placing this prosthesis, it is essential that virtually all calcium be excised from the aortic wall to produce an even, pliable surface for engagement of the pins. Coronary perfusion usually was not required during insertion of the Magovern valve since the total bypass time was usually less than 15 minutes. Six patients underwent replacement of either the mitral or aortic valve and repair by valvotomy or commissurotomy of the other valve. Two patients had both valves replaced with Starr-Edwards prostheses. The average cardiopulmonary bypass time for double valve replacement was 70 minutes; for replacement of one valve and repair of another, 50 minutes, and for single valve replacement, 30 minutes.

After surgery, all patients received prophylactic antibiotic therapy. Digitalis was continued if the patient was digitalized prior to operation, but in many instances digitalis could be discontinued after valve replacement. Diuretics were employed only on specific indication. Blood replacement was controlled carefully to avoid overtransfusion and the average replacement for all patients throughout hospitalization was 1600 ml. Patients with severe pulmonary hypertension or congestion were managed with tracheostomy and assisted respiration for 24 to 72 hours after operation. Initially, anticoagulants were not employed, but after the development of embolic complications in a few patients (Table 8), anticoagulation was used prophylactically in selected patients beginning on the fifth day after surgery and continuing for six months. Patients with a previous sinus rhythm who developed atrial fibrillation or flutter after operation were treated by Lown's method[6] of synchronized monopulse defibrillation to correct the arrhythmia.

Results

The overall mortality rate of 18.7 percent included early and late deaths (Tables 7 and 8). Previous reports from this center[3, 8] indicated mortality risks of 13.5 and 18 percent, but in these surveys, late follow-up was of shorter duration and incomplete.

Eight of the 14 early deaths resulted from the first 67 operations (12 percent) while 6 occurred after the last 104 operations (6 percent), indicating an improvement in early surgical management. Deaths due to hemorrhage, air embolism, and heart block have been eliminated in recent experience.

Fifteen of the 18 late deaths occurred within six months of operation, indicating this to be the period of greatest risk for the valve replacement patient. Three late deaths occurred at 9, 13, and 18 months after operation. One was due to unexpected coronary artery occlusion, one resulted from occlusion

TABLE 7.—*Cumulative Mortality in Mitral and Aortic Valve Replacement.*

Valve Involvement	No. of Patients	No. of Deaths			
		Hospital	Late	Total	%
Aortic stenosis	55	3	4	7	12.7
Aortic insufficiency	51	3	5	8	15.7
Mitral stenosis	18	3	1	4	22.2
Mitral insufficiency	45	5	6	11	24.4
Combined	2	0	2	2	100.0
Total	171	14	18	32	18.7

TABLE 8.—*Causes of Death in Mitral and Aortic Valve Replacement.*

	Mitral	Aortic	Combined	Total
Early Deaths				
Myocardial failure	4	2		6
Cardiac arrhythmias	0	2		2
Hemorrhage	1	1		2
Air embolism	1	1		2
Renal failure	1	0		1
Coronary insufficiency	1	0		1
Total	8	6		14
Late Deaths				
Myocardial failure	2	2	1	5
Thrombo-embolism	2	1	1	4
Coronary insufficiency	0	2	0	2
Detached prosthesis	0	2	0	2
Cardiac arrhythmias	1	0	0	1
Infection	1	1	0	2
Dissecting aneurysm	0	1	0	1
Unexplained	1	0	0	1
Total	7	9	2	18

of the coronary ostia from a propagating thrombus at the prosthetic site, while one was due to mesenteric artery thrombosis or embolism.

Myocardial failure caused 34 percent of deaths (Table 8) and occurred only in Class IV patients with unusual degrees of cardiomegaly or pulmonary hypertension. Most of these were in the mitral group and a majority of these patients had multivalvular involvement. Included were patients unable to maintain satisfactory cardiac output after operation and those dying of intractable pulmonary congestion.

Late complications involving the prosthesis, including thrombo-embolic accidents, infection, and detachment of the prosthesis, caused an additional 25 percent of all deaths. Thrombo-embolism occurred in four patients with death due to secondary involvement of the coronary, cerebral, or mesenteric vessels from thrombus formation at the prosthetic site. Two deaths from systemic infection occurred in patients with a preexisting history of bacterial endocarditis. Prosthetic detachment in two patients was due to breakage of sutures or avulsion of intact sutures from the recipient tissue. These last patients died of progressive heart failure once there was gross incompetence at the valve replacement site. Our present policy is to consider as candidates for reoperation those patients developing heart failure and a new murmur indicating prosthetic incompetency. Three patients recently underwent reinsertion of a new prosthesis with two successes.

Only three deaths resulted from coronary artery insufficiency or blockage despite the existence of significant coronary artery disease in one-fourth of all patients. Two of these three deaths were unexpected and occurred in patients not suspected beforehand of having this condition.

Of 139 survivors, follow-up in 135 was obtained either by physical examination (80 percent) or patient questionnaire (20 percent). A report of exact physical status was unavailable for 4 patients although all are known to be alive. Within two to three months after operation most patients returned to normal activity, had greatly increased exercise tolerance and were relatively free of symptoms.

Eighty patients (57 aortic; 23 mitral) were operated upon more than six months ago. Sixty are working full time and are virtually free of symptoms while 16 are working part time or are doing normal housework and have minimal symptoms such as mild angina or dyspnea only on heavy exertion. Four patients are partially disabled.

Fifty-nine patients were operated on less than six months ago. One patient is partially disabled. All others have fully recovered from operation and demonstrate greatly improved cardiac function. However, these patients are still within the period of maximum risk following valve replacement. Further time should pass before making a final evaluation of the results in this group.

Five of the 139 survivors are partially disabled, although 3 of these consider themselves improved as compared to their preoperative state. Two patients developed cere-

bral embolic accidents from which they are now recovering, one has persistent angina but of a lesser degree than before operation, one has a partially disabling cardiac arrhythmia, and one patient had a myocardial infarction with subsequent recovery. Most of the 139 survivors no longer require special diets or diuretics and many have discontinued the use of digitalis.

Discussion

In selection of patients for valve replacement, few contraindications were accepted in patients having progressive disability from a destroyed valve. A higher risk of operation was expected in the Class IV group and those having advanced cardiomegaly, coronary artery disease, multiple valve involvement and severe pulmonary hypertension. Satisfactory results were obtained even when these hazards were present, justifying this liberal policy in selection of candidates for valve replacement. Indeed, replacement now appears justified when severe valve deformity exists before evidence of advanced heart failure appears. For example, in Class III patients having only moderate cardiomegaly, compensated heart failure, or mild pulmonary hypertension, the surgical risk was approximately 10 percent, whereas the late Class IV patients, with numerous associated hazards, had a risk four to five times greater. Yet, in this latter group, the only hope for survival was corrective surgery since all such patients were in the last stages of cardiac decompensation and many were operated upon virtually in extremis.

None of the complications or deaths were attributable to the technique of cardiopulmonary bypass. The advantage of the simplified method of bypass, now used successfully by us in over 700 open heart operations, was fully borne out of this patient group. The physiologic benefits included freedom from bleeding problems, electrolyte disturbances, and cerebral, renal, or pulmonary complications. Elimination of the need for fresh, heparinized blood in the extracorporeal circuit and avoidance of hypothermia provided desirable flexibility and shortened both anesthetic and total operating times. However, this simplified technique of cardiopulmonary bypass is not restricted to short procedures nor does it demand hasty intracardiac repair.

During bypass, efforts were made to keep the heart contracting quietly by using controlled myocardial perfusion. This degree of cardiac activity did not interfere with the repair; cardiac resuscitation was simplified, indicating good myocardial support; and there was less risk of an unrecognized conduction injury. In 35 recent Magovern aortic valve replacements, coronary artery perfusion was eliminated in all instances since the total time of bypass commonly was less than 15 minutes.

The technique combining normothermia and myocardial perfusion provided prompt resumption of normal heart activity upon termination of bypass, and ventricular fibrillation occurred in only 10 percent of instances. When ventricular defibrillation was required, the direct current (DC) defibrillator proved superior to alternating current defibrillators in providing conversion to sinus rhythm. The DC defibrillator was also used successfully several days after operation for correcting arrhythmias occurring in patients with previous sinus or abnormal rhythm. The simplicity and ease of cardioversion[6] was sufficiently convincing so that at present no patient with atrial flutter or fibrillation is dismissed from the hospital without an attempt at cardioversion. Successful conversion to sinus rhythm was usually possible before dismissal or at a second attempt six weeks later.

Prophylactic anticoagulation was not employed until the occurrence of two late deaths (one mitral; one combined replacement) from embolic accidents. Since controversy exists as to the efficacy of anticoagulation,[5,10] it was decided to anticoagulate only selected patients and allow the remaining patients to serve as controls. At present, mitral patients having a giant left atrium, thrombus in the atrium, or history of embolism are given anticoagulants. Patients undergoing multiple valve procedures were also anticoagulated, and if an embolic episode developed after surgery, therapy was

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started. In addition, a portion of the aortic replacement group followed at this center are receiving prophylactic anticoagulants. Thrombo-embolic accidents occurring after surgery may ultimately be the major disadvantage of valve replacement with synthetic materials. A sutureless type of valve prosthesis not having a cloth sewing ring may provide better protection against the late complications of thrombosis and infection, and during the past several months no such complications have occurred with the Magovern valve. Our early experience with the holding properties of this valve is favorable, and this or a similar type of fixation may ultimately prove better than sutures in preventing late prosthetic detachment.

Myocardial failure and complications involving the prosthesis together accounted for more than one-half of all deaths. Results would be improved by not operating upon the patient of Class IV status with advanced cardiomegaly and pulmonary hypertension. As yet, we have not wished to refuse treatment for such patients, because satisfactory rehabilitation has been obtained in over 50 percent, and some of the more dramatic improvements have occurred in this otherwise hopeless group. Late complications affecting the prosthesis were especially vexing since virtually all of these occurred in patients who were benefited by operation prior to the complications. A study is presently under way to determine if anticoagulation is of value in controlling thrombosis at the prosthetic site.

Particularly impressive has been the number of patients, previously totally disabled, who were improved so greatly that they have discontinued the use of special diets and cardiac medications and are now working full time. All patients surviving the early period following valve replacement have shown considerable and progressive cardiac improvement. Late or persistent cardiac failure has not occurred in any patient beyond three months after operation in the absence of complications involving the valve itself. Overall results have been most gratifying in altering the generally grave prognosis of patients with acquired valvular heart disease.

Summary

Total replacement of the mitral or aortic valve with a ball-and-seat prosthesis was employed in 171 patients with a combined early and late mortality rate of 18.7 percent. All patients were in the Class III or IV group of cardiac function and were evidencing progressive cardiac deterioration despite intensive medical management or previous surgery. The valves were all inserted during cardiopulmonary bypass utilizing a disposable oxygenator primed with 5 percent dextrose under normothermic conditions.

Early, or hospital, mortality rates declined steadily as improvements in surgical technique and management developed. Nearly all late deaths occurred within six months after operation, and most were due either to myocardial failure or prosthetic complications. Deaths due to heart failure occurred in Class IV patients with giant cardiomegaly, extreme pulmonary hypertension, or other associated cardiovascular abnormalities. Complications developing at the prosthetic site consisted of thrombosis, infection, and detachment of the prosthesis.

Surviving patients have been greatly improved, and only five patients in 139 survivors are moderately disabled. Within two to three months after operation nearly all patients demonstrate a reduction in cardiac size, relief of disabling symptoms, and most patients under 60 have returned to a full-time occupation.

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Total Prosthetic Replacement Of the Aortic Valve

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RESEARCH & INVESTIGATION

SURGICAL THERAPY of aortic valve disease often is limited by the condition of the diseased valve. Stenotic valves frequently contain extensive calcific deposits and far advanced fibrous tissue thickening which preclude satisfactory commissurotomy and mobilization of the leaflets. In most cases of valve regurgitation, remaining leaflet tissue is inadequate for plastic repair.

Successful surgical management of such cases requires excision and prosthetic replacement of the valve. Recent development of caged-ball aortic valve prostheses now allows effective valve substitution in most instances. This report summarizes experience with our first 150 consecutive cases of total replacement of the aortic valve employing Starr-Edwards[8] or Magovern[7] prostheses.

Clinical Material

From December, 1962, until April, 1964, 150 patients underwent total replacement of the aortic valve with caged-ball prostheses. Ninety were males and 60 were females. Although most were in the fifth and sixth decades of life, two patients were less than 20 and two were more than 70 years of age (Table 1). Rheumatic fever was the most frequent established etiology, whereas in 34 instances the specific cause of the valve lesion remain undetermined (Table 2).

In 78 patients, findings suggested primarily aortic valve stenosis, and in 72 valve regurgitation predominated. Frequently valves were both stenotic and incompetent; however, an attempt was made to classify each patient according to the major physiologic defect in the valve. Ninety-five patients were considered Class III cardiacs and 55 were in Class IV, according to the New York Heart Association classification. Relative frequency of signs and symptoms are listed in Table 3.

TABLE 1.—*Age Range of 150 Patients.*

Age (Yr.)	No. of Patients
10-19	2
20-29	14
30-39	24
40-49	45
50-59	36
60-69	27
70 and over	2
Total	150

TABLE 2.—*Etiology of Aortic Valve Disease.*

Etiology	No. of Patients
Rheumatic fever	70
Calcification without rheumatic history	29
Congenital	10
Luetic	4
Traumatic	3
Undetermined	34
Total	150

TABLE 3.—*Frequency of Signs and Symptoms.*

Clinical Findings	No. of Patients
Left heart failure	135*
Angina, syncope, or significant arrhythmia	52*
Coronary artery disease	46
Multiple valve involvement	44
Previous valve surgery	21
Right heart failure	18

*Among 15 patients not manifesting left heart failure, indication for operation was either angina or syncope.

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Described is experience with 150 consecutive patients requiring total aortic valve replacement. Development of the caged-ball aortic valve prostheses allows such a procedure to be undertaken with acceptable risk and an excellent chance of cardiac rehabilitation for the patient.

Included in this series were 44 patients with concomitant involvement of one or more other valves. In most patients, operation was limited to aortic valve replacement, but in several, supplementary procedures such as mitral commissurotomy were employed. Not included were patients requiring replacement of more than one valve and these patients will be reported subsequently.[5]

No patient was refused operation due to end-stage cardiac deterioration. In a number of instances it was necessary to induce anesthesia with the patient almost upright due to pulmonary edema unresponsive to intensive medical therapy. A few patients virtually were moribund at the start of surgery. However, no patients considered as Class II cardiacs were accepted for operation. Until long-term results of caged-ball valve prostheses are available, only those patients in whom a relatively poor prognosis can be expected from conventional medical measures should be accepted for valve replacement.

Technique

The method of cardiopulmonary bypass employed for operation in these patients has been described in detail previously.[3, 4] Disposable plastic oxygenators were primed with 5 percent dextrose in distilled water, and normothermia was maintained. Perfusion of one or both coronary arteries with normothermic blood was utilized in most instances for insertion of Starr-Edwards prostheses. Due to the short period of aortic occlusion required for mechanical fixation of Magovern prostheses, coronary perfusion was not utilized for insertion of these valves. Duration of cardiopulmonary bypass averaged 34 minutes for Starr-Edwards prostheses and 14 minutes for Magovern prostheses. Cardiac hypothermia was not used in any of these patients.

Excellent exposure was effected by a median sternotomy incision. While a longitudinal aortotomy was used in the first few patients, a transverse aortotomy was found to offer superior access to the aortic valve and was employed in most instances (Fig. 1a). The major portion of the diseased leaflets was removed, exercising care not to weaken continuity of the aortic wall or produce injury to the atrio-ventricular conduction mechanism. Special care was taken to provide a smooth, relatively calcium-free site for mechanical fixation of Magovern prostheses.

Horizontal mattress or figure-of-eight sutures of two-zero Mersilene were used for fixation of Starr-Edwards prostheses. Double armed sutures first were passed through the remaining rim of leaflet tissue, incorporating a major portion of the aortic wall in each bite (Fig. 1b). After passing the sutures through the sewing ring of the prosthesis, the prosthesis was guided into place beneath the coronary orifices and sutures were tied and cut (Fig. 1c). Magovern prostheses were fixed as described by Magovern and associates[7] (Fig. 2).

Prior to repairing the aortotomy particular care was taken to refill cardiac chambers and the ascending aorta completely with evacuation of all air. A sump routinely inserted into the left ventricular apex aided in prevention of air embolism. Usually, the patient was put into the Trendelenburg position during aortotomy closure to insure further against cerebral air embolization.

When necessary during operation, defibrillation was accomplished by direct current countershock applied directly to the heart. This technique also was used oc-

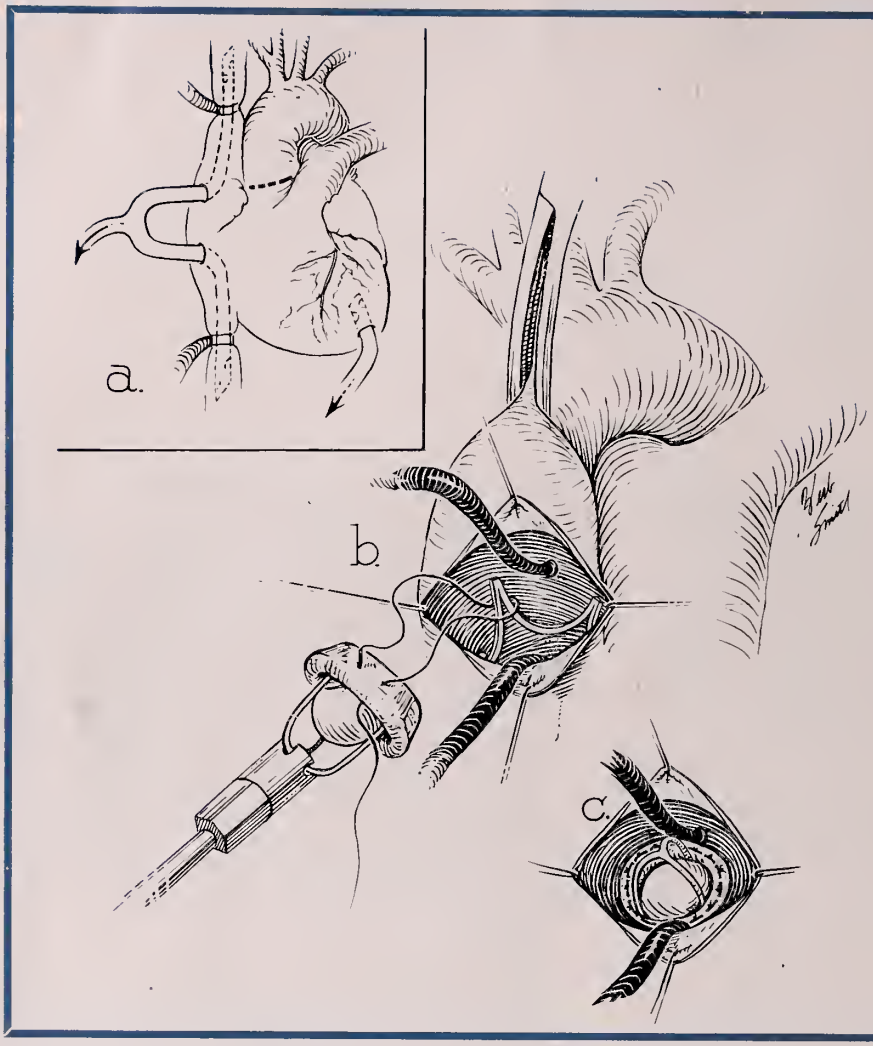

Fig. 1. Drawings illustrating total replacement of the aortic valve with Starr-Edwards prosthesis. a. Cardiopulmonary bypass begun and left ventricle emptied by sump in apex. Valve exposure through transverse aortotomy. b. Following excision of major portion of diseased leaflets, horizontal mattress sutures passed through remaining rim of leaflet tissue, incorporating major portion of aortic wall in each bite. Double armed sutures then passed through sewing ring of prosthesis. c. Prosthesis guided into place beneath coronary orifices, sutures tied and cut.

casionally for conversion of other abnormal rhythms. External direct current countershock was found quite helpful in correcting arrhythmias occurring after operation.[6] Currently, no patient with atrial fibrillation or flutter is discharged from the hospital without an attempt at cardioversion, and patients with such arrhythmias receive countershock when they return for recatheterization.

Following operation, prophylactic antibiotics were given for seven to ten days and occasionally longer. Cardiac glycosides usually were continued, although often it was possible to eliminate use of these soon after valve replacement. Anticoagulants were not given after operation in most patients; however, in recent months an occasional selected patient was anticoagulated. Experience to date has not determined precisely the need for anticoagulants following aortic valve replacement, while several clear-cut indications have been seen in certain patients undergoing mitral or double valve replacement.

Results

Among the 150 consecutive patients undergoing total prosthetic replacement of the aortic valve, there were 12 early or hospital deaths, an operative mortality rate of 8.0 percent. Causes of these deaths are listed in Table 4. Cardiac arrhythmias were responsible for four of these deaths; three resulted from myocardial failure; and two each were attributable to coronary insufficiency and hemorrhage. Significantly, seven of the early deaths occurred in the first 56 patients as operative techniques were being developed and standardized, a mortality rate of 12.5 percent. In the last 94 patients there have been only 5 operative deaths, a reduction in mortality rate to 5.3 percent.

As of April 1, 1964, there had been 14 late deaths (Table 4), giving a total of 26 deaths or a cumulative mortality rate of 17.3 percent (Table 5). Prosthetic complications such as detachment, infection, or thrombo-

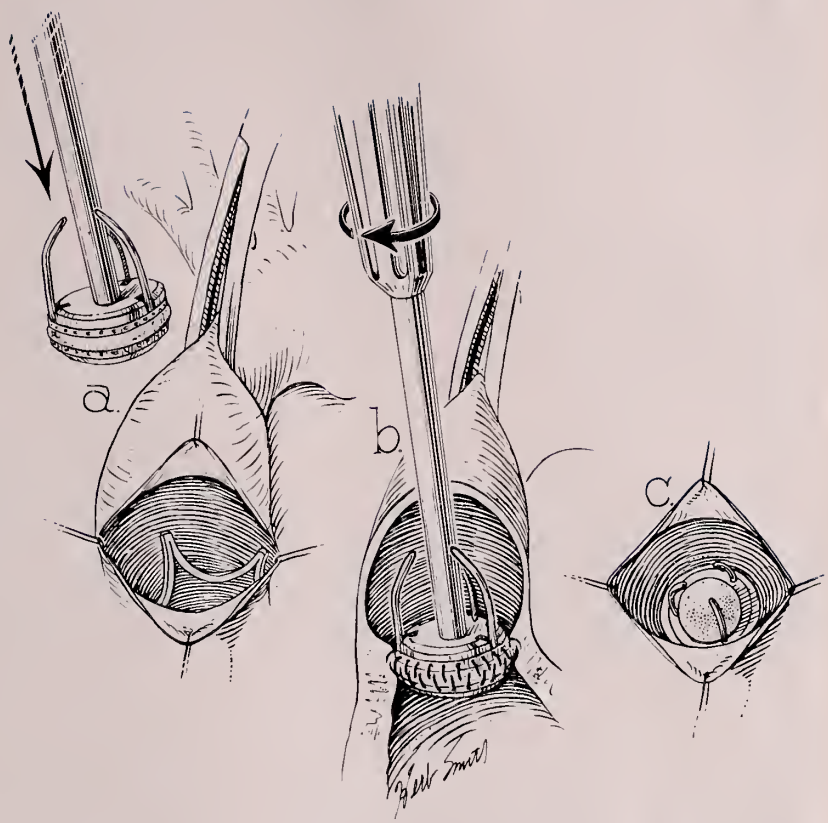

Fig. 2. Drawings illustrating total replacement of the aortic valve with Magovern prosthesis. a. Following valve excision prosthesis on handle introduced through transverse aortotomy. b. Handle rotated to extend pins for mechanical fixation. c. Handle removed and ball inserted into cage.

embolism caused five late deaths and usually occurred within the first six months following operation. Coronary artery disease was responsible for five late deaths and myocardial failure caused two. One patient was killed in a traffic accident and in one instance a dissecting aneurysm resulted in death of the patient. Particularly in the older age group, cumulative mortality rate obviously will increase as time passes. Although a number of these late deaths were unrelated to aortic valve replacement per se, all deaths from whatever cause were included.

One hundred and twenty-four patients were alive and detailed information was available on 118. In six instances insignificant data was obtained for functional evaluation, but each of these patients was known to be alive. Among the 118 patients on whom follow-up data were available, 67 were operated upon more than six months previously while 51 had undergone aortic valve replacement during the preceding six months.

Most patients followed more than six months have returned to full-time work and are virtually free of symptoms. Most no longer require special diets or diuretics, and many have discontinued use of cardiac glycosides. A few are working part time or are doing normal housework with minimal symptoms, such as angina or dyspnea only on strenuous exertion. Three are disabled partially, one from myocardial infarction with recovery, one from a cerebrovascular accident with recovery, and one by angina on lesser degrees of exertion.

All 51 surviving patients operated upon during the previous six months have recovered fully from aortic valve replacement. These patients are considered still within the period of maximum risk following operation and time must pass before a valid evaluation of functional results can be made. How-

TABLE 4.—*Cause of Death in 26 Patients.*

Cause	No. of Patients
Early (Hospital)	
Arrhythmia	4
Myocardial failure	3
Coronary artery disease	2
Hemorrhage	2
Air embolism	1
Total	12
Late	
Coronary artery disease	5
Detached prosthesis	3
Myocardial failure	2
Infection	1
Thrombo-embolism	1
Dissecting aneurysm	1
Traffic accident	1
Total	14
Cumulative Total (April 1, 1964)	26

ever, the majority have demonstrated greatly improved cardiac function and only two are disabled partially, one by angina and one by an intermittent arrhythmia.

Discussion

Technique of cardiopulmonary bypass employed for total prosthetic replacement of the aortic valve in these patients offered impressive advantages over other methods.[3, 4] Many adverse pathophysiologic changes associated with open heart surgery recently have been attributed to the large pool of homologous blood required to prime various types of pump oxygenators, and use of 5 percent dextrose in distilled water to prime disposable plastic oxygenators allowed elimination of many such problems in this group of patients.[1] Significantly, not a single death from renal insufficiency was seen in this series, confirming more satisfactory maintenance of renal function by such a technique of cardiopulmonary bypass.[2] Improved capillary circulation associated with this method of perfusion allowed elimination of the need for hypothermia,[1] shortening perfusion time required and reducing frequency of arrhythmias.

Previous experience with aortic commissurotomy and other plastic procedures on this valve have been disappointing. Incidence of complications using leaflet types of aortic valve prostheses has been high and restenosis has occurred in a number of patients in whom such a prosthesis was employed. Twenty-one of the 150 patients in this series had undergone previous surgery on the aortic valve, either in this center or elsewhere, and re-operation was required by either recurrence of stenosis or development of regurgitation. Currently, replacement with a ball-valve prosthesis is utilized in all patients undergoing operation for acquired aortic valve disease and in a few older patients with congenital deformity of the aortic valve.

Selection of patients for total prosthetic replacement of the aortic valve is aided by information obtained from catheterization and often opacification of the left chamber of the heart and aorta and occasionally the coronary arteries. However, presence or absence of coronary artery disease does not contraindicate operation when aortic valve disease is functionally significant. Many patients with associated coronary artery disease have been rehabilitated by relief of the pressure gradient across the aortic valve with reduction in work load imposed on the myocardium.

Significant time has not passed to allow prolonged prosthetic evaluation, therefore employment of aortic valve prostheses in Class II cardiac patients does not appear indicated. However, once a patient with aortic valve disease demonstrates evidence of cardiac decompensation or develops angina or syncope, prognosis with conventional medical therapy no longer is favorable. It is at this period in the course of acquired aortic valve disease that prosthetic replacement of the valve should be considered. The patient

TABLE 5.—*Cumulative Results in 150 Patients (April 1, 1964).*

Predominant Valve Lesions	No. Patients	No. Alive	Deaths		Cumulative Mortality Rate (%)
			Early (Hospital)	Late	
Aortic stenosis	78	67	5	6	14.1
Aortic regurgitation	72	57	7	8	20.8
Total	150	124	12	14	17.3

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should not be allowed to progress into a Class IV cardiac before undergoing surgical correction, for experience in this series demonstrates an increase in cumulative mortality rate of 11.6 percent to 27.3 percent should this occur. Now that many of the technical problems associated with aortic valve replacement have been overcome, no patient should be denied benefits of such a procedure until operation is forced by imminent death and is associated with a significantly increased mortality rate.

Summary

Successful surgical management of acquired aortic valve disease usually requires excision of the diseased valve with total prosthetic replacement. Development of caged-ball aortic valve prostheses now allows such a procedure to be undertaken with an acceptable risk and an excellent chance of cardiac rehabilitation. Experience with 150 consecutive patients requiring total aortic valve replacement has demonstrated a reduction in operative mortality rate to 5.3 percent. Most patients have returned to normal or near normal activity and many have been able to discontinue special diets and cardiac medications. Once a patient with acquired aortic valve disease demonstrates evidence of cardiac decompensation or develops angina or syncope, valve replacement should be considered at this time.

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Drug Okayed in Indian Rites

Indians using the drug peyote in "sincere" religious rites are not violating the narcotic laws of the State of California. So the California State Supreme Court has ruled.

In reversing the decision of the lower court in the case of three Navajo Indians charged with possession of narcotics, the Supreme Court opinion stated that the issue involved freedom of religious expression which is guaranteed by the U. S. Constitution.

Surgical Treatment of Traumatic Wounds Of the Heart and Blood Vessels

CLINICAL MEDICINE

RECENT ADVANCES in cardiovascular surgery when applied to patients with trauma to the heart or major blood vessels have made restoration of normal circulatory dynamics the goal in the care of these individuals. Hand in hand with improved surgical technique goes responsibility on the part of those caring for acutely injured patients to utilize these new principles. This report is concerned with the management of 565 patients with acute cardiovascular trauma in the city-county charity hospitals of Houston and Harris County, with description of technique utilized for each type of wound.

Wounds of the Heart

Cardiac wounds may be caused by blunt trauma or as a result of penetrating or perforating injury. Blunt trauma, sustained in an ever increasing number of cases as a result of automobile accidents, may produce contusion of the myocardium by direct forces exerted through the thoracic wall or spine. Rapid deceleration may result in tearing injuries related to attachment of the heart and great vessels. Distant compression, transmitted via adjacent organs or the column of blood in the aorta, may result in subintimal coronary hemorrhage and valvular, myocardial, or septal perforation. Myocardial damage also may be related to shock, hemorrhage, and stress. The most frequently encountered cardiac wounds are the result of penetrating injuries made by knives, icepicks, and other sharp instruments. Perforating wounds characteristically are produced by bullets, and usually only those wounds inflicted by low velocity firearms are compatible with life.

Cardiac injury should be suspected in any patient with precordial contusion or penetrating injury. Myocardial contusion may be detected by electrocardiographic changes varying from minor T-wave depression to a picture of severe myocardial infarction, and the patient should be examined for signs of pericardial tamponade (hypotension, restlessness, diminished pulse pressure, paradoxical pulse, elevated venous pressure, and diminished heart sounds). If myocardial continuity is disrupted and the pericardial opening is of sufficient size, tamponade does not occur, and clinical signs may be only those of hemothorax and hemorrhagic shock.

The critical level of intrapericardial pressure is 20 cm. of saline.[9] With even slight increases above this level, intrapericardial pressure exceeds venous pressure, arterial pressure falls and cardiac arrest rapidly ensues. Restoration toward normal circulatory dynamics may begin with aspiration of as little as 5-10 ml. of intrapericardial fluid. Cardiac output also may be increased by elevation of venous pressure as a result of rapid intravenous infusion of saline, plasma expanders, or blood.

Myocardial contusions are treated similarly to myocardial infarctions, governing the period of bed rest by the time necessary for electrocardiographic changes to return to normal. Those patients with penetrating injuries are observed closely and pericardicentesis is performed if signs of cardiac tamponade appear.[2] The paraxyphoid route of pericardial aspiration is preferred to intercostal space approaches, because coronary artery injury is less likely by this method (Fig. 1). Following initial aspiration the patient should be transported to an intensive care unit for close observation. If needed, a second pericardicentesis is performed in the operating room. Should the second aspiration fail or a third aspiration be deemed necessary, immediate thoracotomy and cardiorrhaphy are performed concomitantly with

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New developments in cardiovascular surgery have made possible better care for patients with heart or major blood vessel trauma.

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pericardicentesis.[7] In more desperate cases immediate thoracotomy may be required, either in the emergency room or in the operating room (Fig. 2).

When thoracotomy is necessary, a left anterior fourth or fifth intercostal space incision may be used and continued across the sternum if exposure of the right atrium or right ventricle is required. Primary control of hemorrhage is by finger pressure. Several deep, heavy mattress sutures then are placed beneath the finger, followed by a continuous 4-0 or 5-0 vascular suture close to the wound margin for complete hemostasis (Fig. 3a). Wounds adjacent to coronary vessels may be repaired by deep mattress sutures placed beneath the vessels (Fig. 3b). Occasionally, delayed surgery is required for retained foreign bodies within the heart or pericardium and for delayed effusion with tamponade. Intracardiac shunts and valvular injuries eventually may require cardiopulmonary bypass for repair after definitive anatomical diagnosis.[3, 5]

Among 177 patients with penetrating wounds of the heart alive on admission to the hospital, 45 died, an overall mortality rate of 25.4 percent. However, this includes 10 patients who died before any treatment could be instituted and 12 patients in whom only immediate thoracotomy was performed for cardiac resuscitation. With exclusion of these 22 deaths the corrected mortality rate was only 14.9 percent. Of 110 patients treated primarily by pericardicentesis the overall mortality rate was 14.5 percent and only 4.6 percent in those requiring aspiration alone. In those patients requiring thoracotomy in addition to pericardicentesis the mortality rate was 21.7 percent if cardiorrhaphy was performed without delay, rising to 57.1 percent if cardiac arrest was allowed to occur. This fact adds further support to the concept that an aggressive approach is necessary should the response to pericardicentesis not be immediate and lasting.

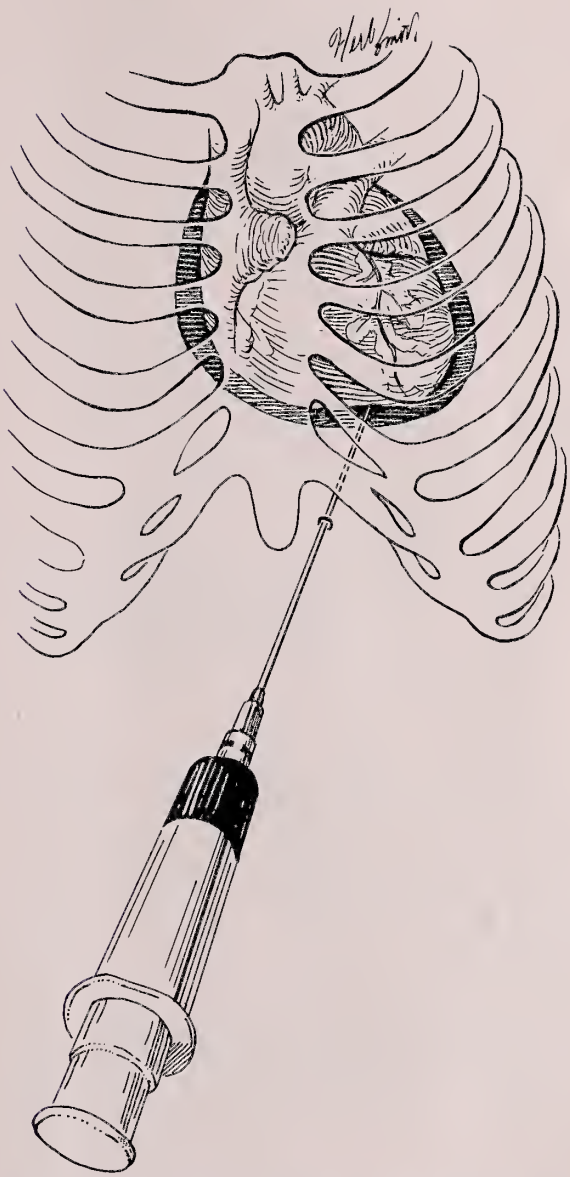

Fig. 1. Drawing illustrating correct needle placement for paraxyphoid pericardicentesis.

Aortic Wounds

Wounds of the aorta may be produced by blunt or penetrating injury. Rapid deceleration associated with automobile accidents may allow the mobile aorta to undergo a shearing injury with intimal tearing and

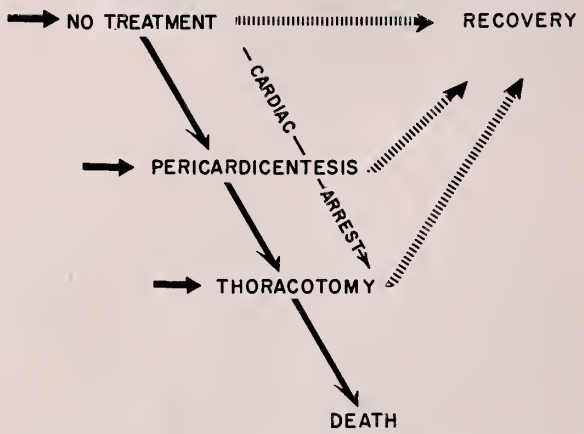

Fig. 2. Drawing illustrating plan of treating penetrating cardiac injuries.

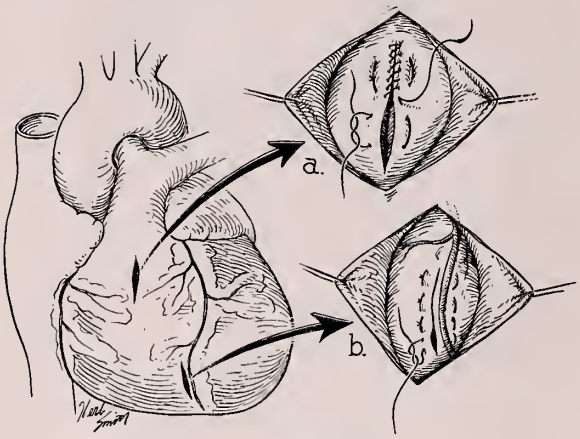

Fig. 3. Drawings illustrating method of repair of cardiac injuries. (a) Away from and (b) adjacent to coronary arteries.

medial disruption at the points of relative fixation: immediately distal to the aortic annulus, distal to the subclavian artery at the ligamentum arteriosum, and at the area where the aorta passes through the diaphragm. Disruption of the entire aortic wall usually is fatal immediately. However, more commonly the intimal and medial disruption result in subsequent aneurysm formation, requiring surgical intervention months or even years after the initial traumatic episode.

Penetrating injuries of the aorta, while often immediately fatal, can be managed successfully if they are suspected and resuscitation is instituted immediately.[1] Among 38 patients with penetrating wounds of the aorta alive on admission to the hospital 12 were saved, a salvage rate of 31.6 percent.[7] Four patients with wounds of the intrapericardial aorta were managed successfully, aided by the fact that pericardial tamponade occurred with temporary containment of hemorrhage. One of two patients with stab wounds of the transverse arch of the aorta survived.[4] One of eight patients with penetrating wounds of the descending thoracic aorta and 6 of 24 patients with penetrating wounds of the abdominal aorta were salvaged.

Diagnosis of this injury is largely by suspicion and immediate operation. In no other situation is immediate surgery such an essential part of resuscitation. Control of hemorrhage by finger pressure or pack should be performed while blood volume is restored.[1] Temporary clamping of the lower thoracic aorta occasionally is helpful with abdominal aortic injury. After blood volume and pressure are restored, repair is carried out by passing sutures beneath the occluding finger or by suture following application of a partial occluding clamp or proximal and distal clamps. Lateral aortorrhaphy is possible in the majority of instances due to the large luminal size of the aorta.

Arterial Wounds

The greatest number of arterial wounds in both civilian and military experience are the result of penetrating injuries by knives, ice picks, gunshot wounds, and missiles, resulting in either total or partial disruption of the vessel wall. Blunt injuries produce contusion to the vessel wall and may be sustained by passage of a high velocity missile in close proximity to the vessel or by fracture or dislocation adjacent to the vessel.[11] Arterial continuity is not interrupted and occlusion is due to subintimal or mediál hemorrhage with compression of the arterial lumen.[12] At exploration this type of injury may be detected by firmness to palpation of the injured area associated with distal arterial spasm and absence of distal pulses.

The diagnosis of acute arterial injury is obvious should there be external hemorrhage, discernible hemorrhage into a body cavity, or loss of distal pulses. However, diagnosis is more difficult with partial lacerations tamponaded by surrounding tissue in which distal pulses are intact. If doubt exists as to arterial injury, arteriograms often are helpful. In certain instances exploration is indicated when a penetrating wound or missile tract lies in close proximity to a major artery.

Consideration first is given to control of hemorrhage either by finger pressure or by application of proximal and distal noncrushing vascular clamps. The injured vessel then is dissected carefully, and proximal and distal control is obtained. Regional heparinization usually is performed by distal injection of 10 mg. of aqueous heparin in 10-30 ml. of saline. Concomitant venous injuries may be repaired while circulating blood volume is replaced. Attention then is turned toward restoration of arterial continuity, both to preserve adequate perfusion of the distal part and to prevent subsequent complications, such as formation of a false aneurysm or arteriovenous fistula. Acute arteriovenous fistulas are managed by concomitant repair of artery and vein. When an arteriovenous fistula becomes evident in the immediate postinjury period there is no need for the time honored delay in surgical intervention. With current techniques of reestablishing arterial and venous continuity there is no advantage to awaiting development of collateral circulation as was required when ligation and excision was the accepted method of treating these injuries.[8]

Lacerations produced by sharp instruments such as knives and glass may be repaired by lateral arteriorrhaphy, taking care to flush the vessel proximally and distally before completion of the suture line (Fig. 4). Application of a patch graft and intimal suture occasionally may be necessary in order to prevent constriction of a small vessel or distal dissection[10] (Fig. 5). With greater vessel wall destruction, excision and end-to-end anastomosis (Fig. 6) or graft replacement (Fig. 7) may be performed depending upon the length of the damaged segment. If a graft is needed, crimped, knitted grafts of Dacron have proved most satisfactory for this purpose. Bypass grafts may be employed when long arterial segments are thrombosed due to intimal or medial dissection (Fig. 8). Concomitant sympathectomy may be indicated following repair of distal extremity vessels. The more distal the injury the more demanding is its repair, for although ligation of proximal extremity vessels may be accompanied only by symptoms of relative ischemia, ligation of distal extremity vessels may lead to frank gangrene (Fig. 9).

With injury to the carotid arteries a polyethylene internal shunt is inserted rapidly to prevent cerebral ischemia during repair[6]

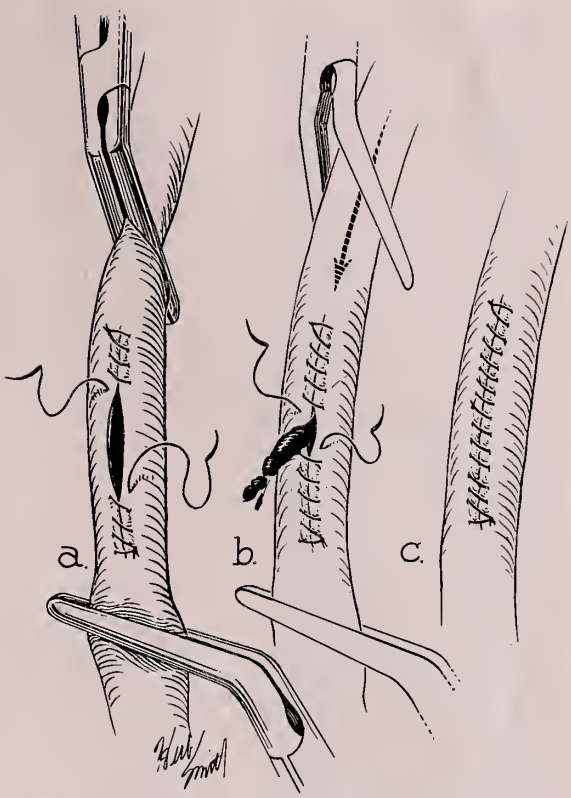

Fig. 4. Drawings illustrating method of lateral arterial repair. (a) Over and over cardiovascular sutures started at each end of defect after application of proximal and distal vascular clamps. (b) Distal, then proximal, flushing performed to remove clots. (c) Completed repair.

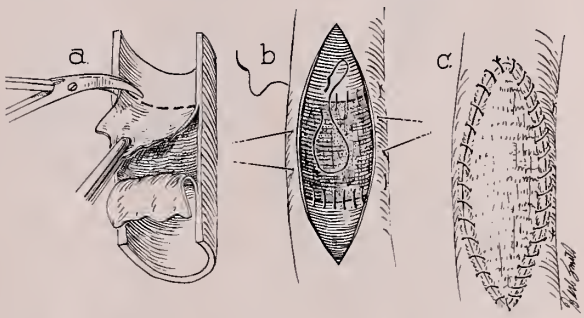

Fig. 5. Drawings illustrating method of small vessel repair following blunt trauma with subintimal hemorrhage. (a) Excision of damaged intima. (b) Intima sutured with multiple interrupted sutures to prevent dissection. (c) Completed repair. Patch graft utilized to prevent constriction of vessel lumen.

(Fig. 10 and 11). Visceral arterial injuries are complicated by problems in exposure, difficulty in control of hemorrhage, and ischemia to that organ or portion of the intestinal tract receiving its arterial supply from the vessel. Penetrating wounds of the cervico-thoracic region with injury to the subclavian vessel are managed best by first obtaining proximal control either by a ster-

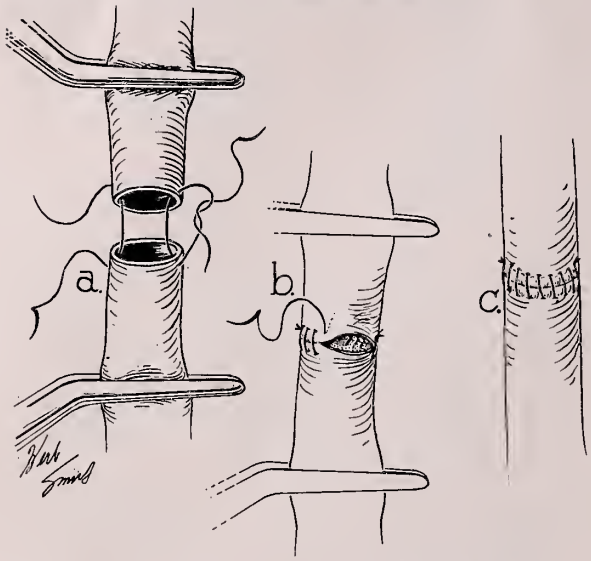

Fig. 6. Drawings illustrating method of end-to-end anastomosis. (a) Sutures placed at each side of vessel after application of vascular clamps. (b) Posterior, then anterior suture line performed. (c) Completed repair.

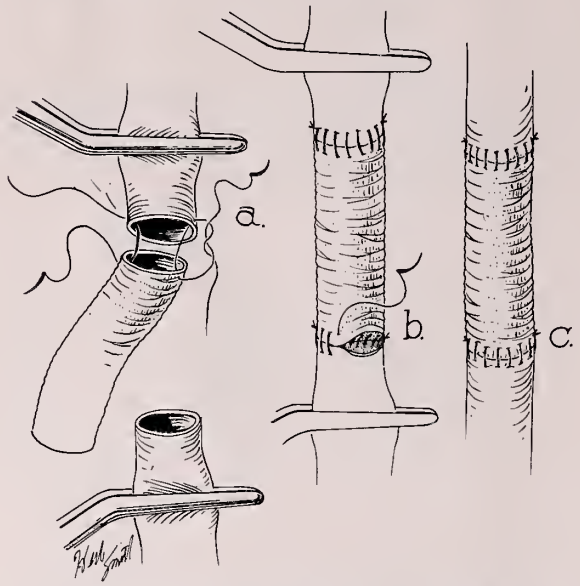

Fig. 7. Drawings illustrating method of graft replacement. (a) Proximal anastomosis started after excision of injured segment and application of vascular clamps. (b) Proximal, then distal anastomosis performed. (c) Completed graft.

nal splitting or anterior third intercostal space incision.[14]

Among 289 acute arterial injuries, repair was performed in 217 instances with a mortality rate of only 4.6 percent. Amputation was required 13 times, but the majority of these were necessitated by associated injuries, massive soft tissue or bone destruction rather than the arterial injury itself.[7]

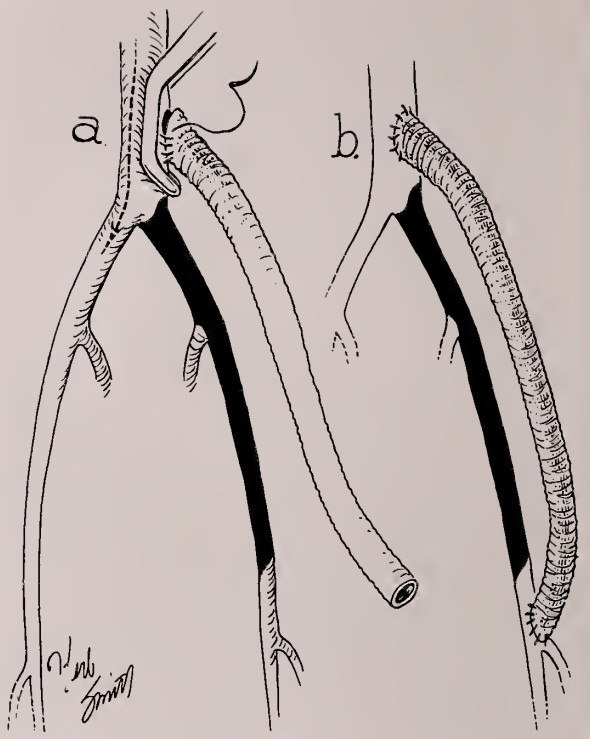

Fig. 8. Drawings illustrating application of bypass principle to establish blood flow distal to area of thrombosis. (a) Proximal anastomosis performed after application of partial occluding vascular clamp. (b) Completed bypass.

Distal pulses were restored in 186 instances with viability of the distal part being maintained in an additional 59 cases in which a palpable peripheral pulse was not obtained.

Twenty-two deaths occurred in the entire series of 289 arterial injuries, an overall mortality rate of 7.6 percent. Nine of these deaths were associated with wounds of the iliac arteries, five with carotid artery wounds, and four with wounds of the superior mesenteric artery. A more aggressive approach in the management of major injuries combined with improved technical approaches for repair of carotid and visceral arterial injuries should result in an even lower mortality rate.

Vena Caval Wounds

Wounds of the vena cava may be caused by both blunt and penetrating injuries. Any surgeon exploring a retroperitoneal hematoma should be prepared to deal with an injury of the inferior vena cava. Once thought better left undisturbed, these hematomas should be explored to rule out injury to the duodenum, pancreas, and other retroperitoneal structures.

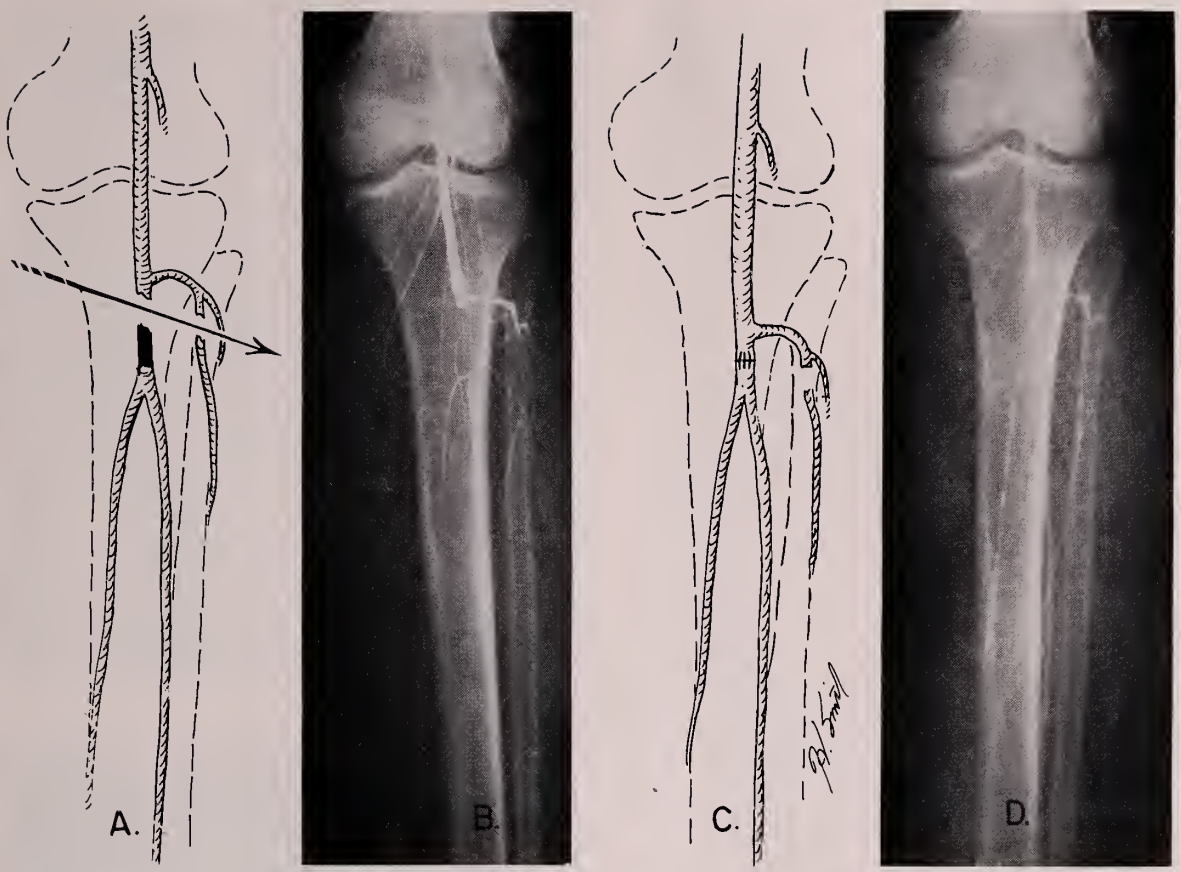

Fig. 9. Gunshot wound transecting popliteal artery distal to origin of anterior tibial artery. (a) Drawing illustrating injury. (b) Arteriogram demonstrating injury prior to surgical repair. (c) Drawing illustrating area of repair in relation to distal femur. (d) Arteriogram after operation demonstrating area of repair and patency of distal vessels.

Control of hemorrhage, as with aortic and major arterial wounds, is by direct pressure. Following restoration of blood volume the vena cava may be isolated above and below the injury and occluded with noncrushing vascular clamps, or suture may be performed beneath the occluding finger. In dealing with through-and-through wounds the posterior defect may be repaired from the inside, and if a posteriorly placed wound is inaccessible an anterior venotomy may be performed with suture of the defect from the intraluminal side. Large distal defects in a critically ill patient may be managed by ligation above and below the area of injury; however, all wounds above the renal veins must be repaired (Fig. 12). Under no circumstances should permanent control of vena caval injury be attempted by packing.[13]

Among 61 patients with penetrating wounds of the vena cava alive on admission to the hospital, 31 died, an overall mortality rate of 51 percent. All except one of the 61 patients had major associated injuries. The predominant cause of death was hemor-

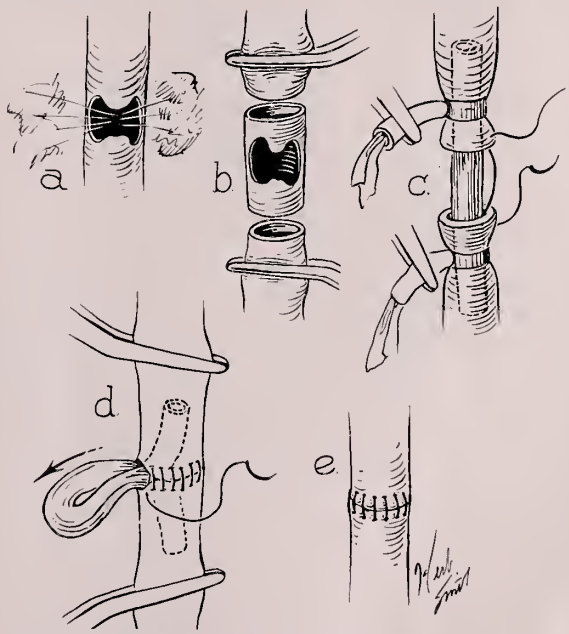

Fig. 10. Drawings illustrating method of repair of irregular lacerations of the carotid artery using an internal shunt. (a) Irregular laceration. (b) Injured area excised. (c) Internal shunt inserted. (d) Internal shunt removed immediately prior to completion of the end-to-end anastomosis. (e) Completed repair.

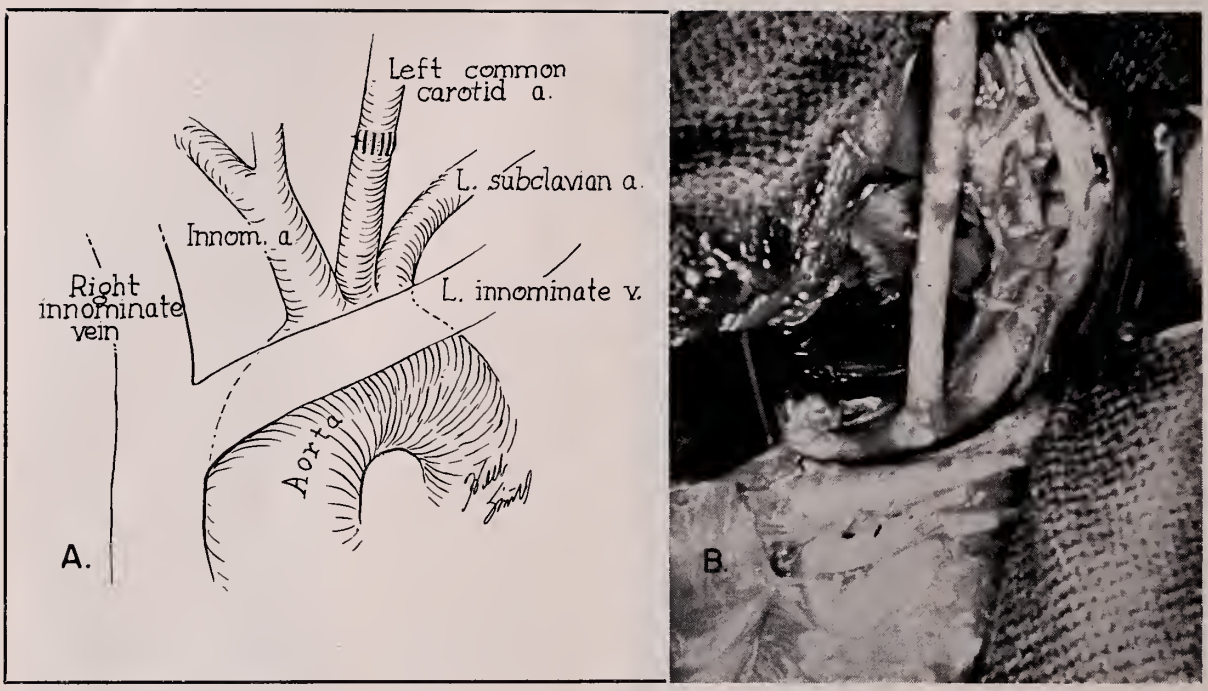
Left common carotid a.
L. subclavian a.
Innom. a
Right innominate vein
L. innominate v.
Aorta
A.
B.
C.

Fig. 11

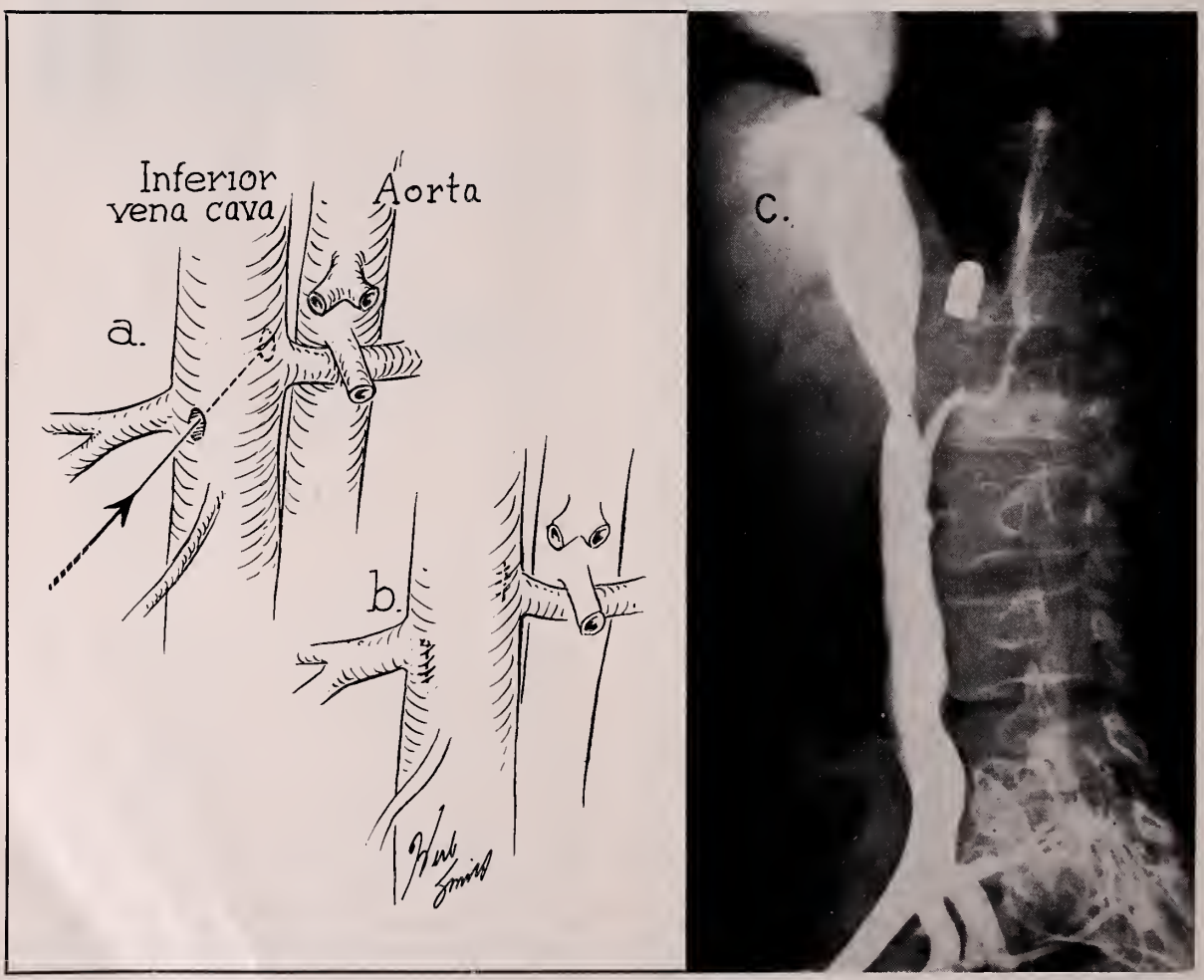
Inferior vena cava
Aorta
a.
b.
C.

Fig. 12

Fig. 11. Facing page, above. Complete transection of the left common carotid artery just distal to aortic arch. (a) Drawing and (b) Operative photograph demonstrating completed end-to-end anastomosis (arrow) following internal shunt removal. Patient fully recovered without neurologic deficit.

Fig. 12. Facing page, below. Through-and-through gunshot wound of inferior vena cava at level of renal veins. (a) Drawing illustrating injury. (b) Completed repair. (c) Inferior vena cavagram demonstrating patency of inferior vena cava six months after repair of injury illustrated in (a).

rhage, frequently not from the vena cava itself, but from an associated aortic or major arterial injury.[7]

Injuries to other veins are managed similarly. Except under extenuating circumstances major veins should be repaired, reserving ligation for smaller tributaries and branches. Such a policy is of particular importance in the management of extremity injuries to prevent postoperative edema and improve total extremity blood flow.

Comment

The primary consideration in management of patients with traumatic wounds of the heart and blood vessels is control of hemorrhage, for hemorrhage is the threat to life, and indeed the cause of death in most instances. Primary control is effected best by application of pressure followed by the judicious application of noncrushing vascular clamps. Definitive repair then is performed after blood volume is restored. In the case of carotid artery wounds an internal shunt should be inserted as soon as technically possible.

Continued hypotension in spite of adequate blood replacement demands explanation in the operating room, where the bleeding source may be identified and controlled simultaneously. With trauma to the cardiovascular system, surgical intervention does not follow, but is an integral part of resuscitation. Techniques of definitive repair vary with the specific injury and always should be aimed toward restoration of normal circulatory dynamics.

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Summary

Recent advances in cardiovascular surgery applied to the patient with trauma to the heart or major blood vessels have made restoration or normal circulatory dynamics the goal in the care of these individuals. From experience gained in the management of 565 patients with acute cardiovascular trauma, including 177 with penetrating wounds of the heart, 38 with aortic injuries, 289 with acute arterial injuries, and 61 with vena caval injuries, improved surgical techniques for the management of each type of wound have been ascertained. These methods of management should be an integral part of the armamentarium of all physicians caring for acute trauma.

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Pediatric Cardiovascular Surgical Emergencies

CLINICAL MEDICINE

THE NEWBORN period is one of critical adjustment. During the first few months of extrauterine existence the infant by virtue of its small size is relatively more susceptible to alterations in his external and internal environment than is his adult counterpart. It is in this phase of life that congenital heart disease exacts its greatest toll. In fact, of all infants born alive with defects of the heart and great vessels, 60 percent die during the first year, and half of these die during the first month of life.[7] In 4,000,000 live births in the United States during 1958 there were 113,000 deaths during the first year of life and an estimated 8,000 of these were due to anomalies of the cardiovascular system.[10]

Infants with cardiovascular defects experience difficulty primarily from hypoxemia and/or heart failure depending on the nature of the anomaly. Medical therapy with oxygen, digitalis, and diuretics may afford enough improvement that surgical therapy can be delayed until a later date when the infant is larger and better able to tolerate anesthesia and operation. More often than not, however, drug therapy brings only temporary or no relief of hypoxemia and cardiac failure, and death is imminent. Under these circumstances operation is mandatory and must frequently be done on an emergency basis.

For purposes of discussion cardiovascular anomalies may be divided into two groups, depending on the basic pathologic and physiologic mechanism at work:

(1) Cyanotic anomalies (includes Fallot's tetralogy, tricuspid atresia, pulmonary atresia, total anomalous pulmonary venous drainage, and transposition of the great vessels).

(2) Acyanotic anomalies (includes patent ductus arteriosus, ventricular septal defect, coarctation of the aorta, aortic stenosis, pulmonary stenosis and vascular ring).

The purpose of this article is to describe each anomaly briefly with reference to diagnosis and treatment and to present data on a group of 550 infants less than 12 months of age with anomalies of the heart and great vessels who were operated upon, frequently on an emergency basis.

Cyanotic Anomalies

In infants with cardiovascular anomalies cyanosis usually results from an obstruction to pulmonary blood flow coupled with a right-to-left shunt allowing unoxygenated blood to enter the systemic circulation. The classic example of this is Fallot's tetralogy. Patients with tetralogy have decreased pulmonary blood flow because of stenosis of the pulmonary valve or infundibulum, and a right-to-left shunt is present through a ventricular septal defect. Also part of the pathologic picture is right ventricular hypertrophy and dextroposition of the aorta. Infants with this complex are cyanotic but do not have cardiac failure. Roentgenograms of the chest (Fig. 1) show a small heart with decreased pulmonary vascular markings. Right axis deviation is present in the electrocardiogram. Hypoxemia may be severe enough to result in "spells" of unconsciousness and when these appear, prognosis for survival is poor.[6] Oxygen therapy usually

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Value of surgery in reducing deaths from congenital heart disease is shown.

GRADY L. HALLMAN, M.D.
DENTON A. COOLEY, M.D.

produces only temporary relief and operation is urgently indicated. Total correction of Fallot's tetralogy during the first year of life is associated with such excess risk that a palliative operation is done in these small, cyanotic infants. An increase in pulmonary blood flow is obtained by anastomosis of a systemic vessel, subclavian artery or aorta, to a pulmonary artery. Cyanosis is relieved and the infant is followed until six or eight years of age when a complete repair can be done utilizing cardiopulmonary bypass.[5]

Fig. 1. Posteroanterior roentgenogram of chest in a five-month-old infant with Fallot's tetralogy. Note the small heart and clear, hypovascular lung fields. A systemic pulmonary shunt was done and relieved the hypoxemia.

Infants with tricuspid atresia have decreased pulmonary blood flow because of complete obstruction at the tricuspid valve. A right-to-left interatrial shunt through the foramen ovale results in severe cyanosis. Roentgenograms of the chest show a small heart with decreased pulmonary vascular markings. Tricuspid atresia is the only cyanotic congenital heart anomaly with left axis deviation on the electrocardiogram. As in Fallot's tetralogy, the primary problem is hypoxemia, and relief can be obtained by construction of a systemic-pulmonary artery shunt to increase the pulmonary blood flow. There is no satisfactory corrective operation for tricuspid atresia at the present time.

Patients with pulmonary valvular atresia and intact ventricular septum have marked hypoxemia and rarely live beyond the first few months of life without surgical therapy.[4] Some pulmonary blood flow may be maintained by a patent ductus whose spontaneous closure precipitates an anoxic crisis that requires an emergency operation. If the right ventricle is sufficiently well developed, a corrective operation may be performed in which the pulmonary valve is opened using a brief period of caval inflow occlusion. Frequently, however, the right ventricle is hypoplastic and palliation must be afforded by construction of a systemic-pulmonary arterial shunt as in the first two anomalies discussed.

In total anomalous pulmonary venous drainage all of the pulmonary venous blood drains into the right atrium where it mixes with the systemic venous blood. A portion of this mixture passes through the tricuspid valve and on to the lungs while some passes through an atrial septal defect into the left atrium for systemic distribution with resultant cyanosis. Circulatory dynamics may be disturbed to such an extent that heart failure

and cyanosis threaten survival. Precise diagnosis usually requires cardiac catheterization and angiocardiography. Operative correction entails use of the pump oxygenator so that pulmonary venous blood may be directed to the left atrium and the atrial septal defect closed.[3]

The aorta arises from the right ventricle in patients with transposition of the great vessels and carries unoxygenated blood back out into the systemic circulation. The pulmonary artery arises from the left ventricle and carries oxygenated blood back to the lungs. There are, thus, two closed circuits and survival depends on the degree of mixing between them. Communications between the two isolated systems may exist in the form of a patent ductus arteriosus, ventricular septal defect, or atrial septal defect. Infants with transposition are usually deeply cyanotic because the aorta arises from the right ventricle. Blood flow through the lungs is normal, however, and pulmonary vascular markings on chest roentgenograms aid in differentiating this anomaly from those cyanotic conditions characterized by decreased pulmonary blood flow. Cyanosis and hypoxemia may be severe and can require an emergency surgical procedure even during the first few days of life. Using a closed technique an atrial septal defect is created to allow mixing between the two circulations and is usually very effective in relieving cyanosis.[1] Survival to an older age is then possible at which time a complete correction can be done using cardiopulmonary bypass.

Acyanotic Anomalies

Infants with acyanotic cardiovascular anomalies experience difficulty primarily from the presence of large left-to-right shunts and from valvular or other obstructive lesions. A patent ductus arteriosus allows blood to shunt from the aorta into the pulmonary artery. After passing through the lungs this extra blood overloads the left side of the heart and may cause heart failure. A typical continuous "machinery" murmur may be present or the murmur may be systolic only in timing. Roentgenograms of the chest show an enlarged heart with increased pulmonary vascular markings due to the augmented pulmonary blood flow. In some cases cardiac catheterization may be necessary to differentiate this lesion from ventricular septal defect. If the shunt through the ductus is large, heart failure may be severe and poorly responsive to medical therapy. Under such circumstances operation is urgently indicated. Suture ligation or division and suture constitute definitive treatment for patent ductus arteriosus and yield gratifying results.

The left-to-right shunt through a ventricular septal defect produces the same hemodynamic problems as patent ductus in regard to left heart overload but may have more severe consequences because of its larger volume. Heart failure, growth retardation, and recurrent pneumonia may constitute serious problems. Chest roentgenograms show cardiac enlargement and increased pulmonary vascularity (Fig. 2). Both patent ductus and ventricular septal defect are accompanied by left atrial enlargement evident on the barium esophagram in the lateral and

Fig. 2. Posteroanterior roentgenogram of chest in a three-month-old infant with ventricular septal defect. Note cardiac enlargement and increased pulmonary vascular markings due to increased flow. Pulmonary artery banding produced relief of heart failure.

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right anterior oblique views. This results from the large volume flow handled by the left atrium which must accommodate the blood it usually receives from the systemic circulation and right heart plus the blood that has short-circuited through the shunt. Open heart repair of a ventricular septal defect during the first few months of life carries an excessively high mortality,[2] so a palliative operation is done to decrease the pulmonary blood flow and hence the volume of the left-to-right shunt. This is accomplished by constricting the pulmonary artery with circumferential cotton or Teflon tape.[8] Pulmonary artery banding relieves heart failure and allows the infant to survive to the age of four to six years when the ventricular septal defect can be closed with the aid of cardiopulmonary bypass.

Coarctation of the thoracic aorta produces a characteristic picture of hypertension in the upper extremities and absent pulses in the lower. The obstruction to blood flow, usually just distal to the left subclavian artery, may cause severe heart failure which is difficult to control with medical measures.[9] Resection of the constricted area with primary end-to-end aortic anastomosis is the operation of choice and results in a high survival rate when there are no associated intracardiac anomalies.

Stenosis of the aortic or pulmonary valve may result in left or right heart failure respectively. In pulmonary stenosis with intact ventricular septum there may be no right-to-left shunt and cyanosis is usually not present unless heart failure is extreme. The offending valve can be opened utilizing caval inflow occlusion and the ventricular burden relieved.

Congenital anomalies of the aortic arch may form a "vascular ring" around the trachea and esophagus. The resultant respiratory and alimentary obstruction may lead to recurrent pneumonia and death. Complete relief of obstructive symptoms can be obtained by division of the offending vascular structure.

Comment

Congenital heart disease should be suspected in every infant with hypoxemia and/or cardiac failure manifested by cyanosis, rapid respiration, rales, tachycardia, and hepatomegaly. Edema may be difficult to detect or absent. A heart murmur may not be present. The heart on roentgenographic study may be enlarged or normal in size and the pulmonary vascular markings decreased, normal, or increased depending on the nature of the anomaly. Electrocardiograms help establish a diagnosis and cardiac catheterization with angiocardiography usually supply the final answer. If an infant is extremely ill, it may be necessary to operate on the basis of a clinical diagnosis alone, since catheterization and angiocardiography would carry an added risk.

Between 1954 and 1964, 550 infants with cardiovascular anomalies were operated upon during the first year of life at the Texas Children's Hospital (Table 1) and nearly 80 percent were operated upon during the first six months (Table 2). These were all patients who failed to respond to medical therapy or who were so gravely ill that survival did not seem likely. Most operations were performed on an urgent or emergency basis occasionally in the evening or at night. Table

TABLE 1.—*Results of Cardiovascular Surgery During the First Year of Life.*

Anomaly	No. Patients	Living	%
Patent ductus arteriosus	100	92	92
Transposition of the great vessels	89	62	70
Fallot's tetralogy	76	65	85
Ventricular septal defect	62*	43	69
Coarctation of aorta	58†	43	74
Tricuspid atresia	35	29	83
Total anomalous pulmonary venous drainage	28	14	50
Pulmonary valvular atresia	21	9	43
Aortic stenosis	15	13	87
Pulmonary stenosis	11	10	91
Vascular ring	8	8	100
Miscellaneous	47	14	30
Total	550	402	73

*Includes 32 infants in whom open-heart repair was done—mortality 44 percent.

†Includes 11 patients with preductal coarctation and associated intracardiac anomalies, all of whom died.

TABLE 2.—*Age Distribution in Patients Undergoing Surgery During First Year of Life.*

Age	No. Patients	Percent
0-3 Months	320	58
3-6 Months	113	20
6-9 Months	63	12
9-12 Months	54	10
Total	550	100

1 reveals that survival is related to the severity of the anomaly and the magnitude of the operation required for its palliation or correction. The miscellaneous category includes patients with various complex and incurable anomalies including many in whom only an exploratory thoracotomy was done as a final effort to rule out the presence of a curable lesion.

Summary

Congenital heart disease is a major cause of death in the newborn period and exacts its greatest toll during the first few months of life. A small infant seriously ill with hypoxemia and/or cardiac failure who does not respond immediately to medical therapy constitutes a surgical emergency. Between 1954 and 1964, 550 infants with cardiovascular anomalies underwent operation during the first year of life, frequently on an emergency basis. Various palliative and corrective procedures were done utilizing both closed and open heart techniques. A survival of 73 percent of patients in this consecutive, unselected series illustrates the value of surgical therapy for congenital cardiovascular anomalies when medical therapy is ineffective.

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Cholesterol Level, Artery Disease Unrelated

A study of 1,700 patients with hardening of the arteries revealed no definite correlation between the level of cholesterol in the blood and the nature and extent of their disease, a Baylor University research team reported in the Aug. 31 issue of the *Journal of the American Medical Association.*

Eight out of 10 patients, or 78 percent, had cholesterol levels within the normal range for Americans, according to the report.

The values for patients with widespread atherosclerotic disease were not remarkably different from those among patients whose disease was more limited, Dr. H. Edward Garrett, Evan C. Horning, Ph.D., Billy G. Creech, Ph.D., and Dr. Michael DeBakey, Houston, wrote in the *Journal.*

Furthermore, there was no real difference in cholesterol values between patients with occlusive disease in which arteries become blocked and those with aneurysmal disease in which arteries develop bulges, they said.

Diabetic Nephropathy

MAX ELLENBERG, M.D.

REVIEW ARTICLES

THE INCREASING frequency and ominous import of renal involvement in diabetes underscore its clinical significance. Of prime consideration for a more fundamental understanding of this condition is realizing that there is no single entity underlying the renal pathology. On the contrary, it is a conglomerate of glomerular, arterial, and infectious elements. This has resulted in the concept of, and the use of the term, diabetic nephropathy.

Pathologic Components

Glomerulus.—(a) The essential feature of *nodular glomerulosclerosis,* first described by Kimmelstiel and Wilson,[10] is a nodular mass of laminated hyaline material. The nodules may be single or multiple in any individual glomerulus, and there may be few or many glomeruli affected. The nodule is typically in the center of the glomerular lobule, surrounded by a spared, patent outer wall of the capillary loop, with nuclei congregated around its periphery (Fig. 1). A most important aspect of this lesion is its specificity; it is the only pathognomonic finding that documents the diagnosis of diabetes. The few presumed exceptions to this statement will be referred to later. (b) *Diffuse glomerulosclerosis* is frequently present by itself or may coexist with the nodular lesion. It consists of widespread circumferential hyaline thickening of capillary walls (Fig. 2). Although it is not recognized as diagnostic for diabetes, there are several distinguishing characteristics which may differentiate diffuse glomerulosclerosis from similar glomerular pathology in the nondiabetic individual. The lesion begins in the periphery of the glomerulus and with progress there is no diminution in glomerular size.[9] The lesion originally created considerable confusion because early literature did not always differentiate the nodular and the diffuse changes. Many discrepancies and subsequent denial of lesion specificity ensued until their individuality was recognized. (c) *The exudative lesion* is a homogeneous, eosinophilic, fibrinoid deposit located either in the capillary lumen or in the capsular space, and is picturesquely described as the "capsular drop" (Fig. 3). The location differs from nodular or diffuse glomerulosclerosis, the latter being either intramural or intercapillary. The exudative lesion lacks histologic features sufficiently characteristic to permit precise differentiation to establish specificity.[1] However, when present in diabetes, the lesion is associated with the nodular or diffuse glomerulosclerotic lesion. Experimentally produced lesions that presumably simulate the specific diabetic lesions, and indeed have been mistaken for them, are these exudative lesions.

Vascular Involvement.—Arteriosclerosis and arteriolosclerosis, both extra- and intrarenal, are almost always present in varying degrees in diabetic nephropathy.

Pyelonephritis.—This may be acute or chronic, diffuse or focal. Pyelonephritis is far more common in the diabetic than in the nondiabetic and plays a definite role in the clinical and pathologic manifestations of diabetic renal disease.

It is the combination of all these factors, namely glomerular, vascular, and infectious, occurring in varying degrees of severity and frequency that constitutes the entity of diabetic nephropathy.

Clinical Picture

Since all the described factors are unpredictable as to their presence or degree of severity when present, there is little wonder that there is no uniform, predictable clinical

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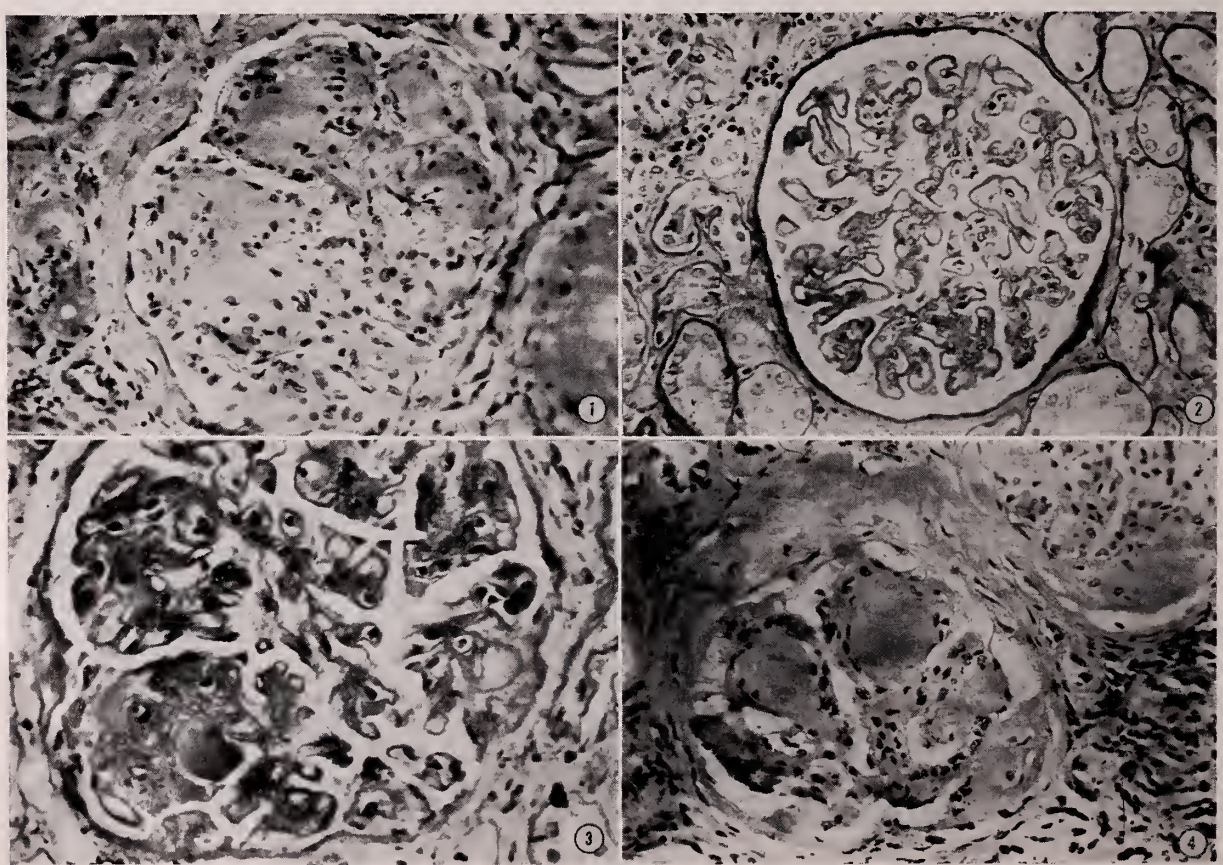
Diabetic nephropathy is defined in this review of renal involvement in diabetes as a conglomerate of glomerular, vascular, and infectious components with no reliably predictable clinical counterpart. There is no single entity underlying the renal pathology, Dr. Ellenberg declares.

picture of renal involvement in the diabetic. With the initial pathologic description of the nodular lesion of glomerulosclerosis, Kimmelstiel and Wilson also included a clinical picture consisting of diabetes, hypertension, albuminuria, and edema. This has been referred to as the K-W syndrome and is actually a nephrotic syndrome. The eponym of K-W for both a pathologic finding and clinical syndrome has been thoroughly confused and confusing. The term has been used indiscriminately on the assumption that the clinicopathologic conditions are mutually dependent, and that if one is present, the other necessarily exists. However, the facts indicate that the two entities are not dependent and do not follow one on the other.[11] The eponym must be eliminated before a clear understanding may be obtained. One should speak of diabetic nephropathy, and the nephrotic syndrome.

Only 10 to 15 percent of nodular glomerulosclerosis is associated with a nephrotic picture; contrariwise, a nephrotic picture may occur without the simultaneous presence of the K-W lesion. Actually there is a closer relationship of the clinical nephrotic syndrome to the pathologic picture of diffuse glomerulosclerosis. In assessing the significance of edema, an essential component of the nephrotic syndrome, it must be remembered that edema may result from congestive heart failure. Heart failure, whether a result of arteriosclerosis, hypertension, or pyelonephritis, is a most common accompaniment of diabetic nephropathy and is ordinarily responsible for a considerable portion of the edema. Other unusual conditions producing a nephrotic syndrome such as renal amyloidosis, nephrotic glomerulonephritis, or the rare instances of renal vein thrombosis or diffuse collagen disease may be the underlying factors. Furthermore, with modern day therapy, edema may be minimized, deferred, prevented, or reversed.

If the complete picture, which has been embellished since its original clinical description, consisting of diabetes, hyperten-

Fig. 1. Glomerulus with nodular glomerulosclerosis.
Fig. 2. Glomerulus with diffuse glomerulosclerosis (periodic acid–Schiff stain).
Fig. 3. "Capsular drop" of exudative glomerular lesion.
Fig. 4. Renal biopsy in Case 1 showing classical nodular glomerulosclerosis.



sion, azotemia, hypoproteinemia, hyperlipemia, albuminuria, edema, retinopathy, and neuropathy is present, then one may be highly suspicious—but never certain—of the presence of nodular glomerulosclerosis. Albuminuria is the one finding that is entirely characteristic, omnipresent, and ominous in all forms of diabetic nephropathy. Indeed, in any instance of albuminuria of undetermined etiology, diabetes must be considered in the differential diagnosis.

Neuropathy and Nodular Glomerulosclerosis.—The inclusion of neuropathy as part of the clinical picture merits further discussion. I have never seen the lesion of nodular glomerulosclerosis at autopsy or on renal biopsy without the clinical counterpart of neuropathy being present, especially as manifested by absent ankle jerks. The significance of absent ankle jerks as an abnormal finding regardless of age, and its further significance as an indication of the presence of diabetes and diabetic neuropathy has been documented.[6] If, as experience suggests, the nodular lesion is always associated with absent ankle jerks, then a nephrotic syndrome in diabetes with a normal neurologic status points to a different underlying pathology. Actually in two such instances (that is, nephrotic syndrome and normal neurologic examination), renal biopsy established the diagnosis of exudative nephropathy and both patients responded dramatically to corticosteroids. This implies differing pathogenetic mechanisms for the exudative lesion versus the glomerulosclerotic lesion, and suggests the interrelationship of the capillary derangements in the diabetic glomerulus and neuron.

There is almost complete agreement that nodular glomerulosclerosis is diagnostic for diabetes mellitus. However, there are instances in which the lesion has been found at autopsy without corroborative clinical evidence of the prior presence of diabetes during life. The following case reports indicate that overt clinical diabetes may be completely absent and yet there may be classical nodular lesions of glomerulosclerosis.

Case Reports

Case 1.—This was the first admission to Mount Sinai Hospital of a 64-year-old Negro woman. Her symptoms began one year ago with dyspnea on exertion followed by edema which progressively increased to involve the lower extremities and the abdomen. The past history was noncontributory, and included no previous knowledge of diabetes. The family history revealed that her mother had diabetes and hypertension.

On examination the patient exhibited generalized anasarca. Pulse rate was 48 and irregular; blood pressure 100/60 mm. of mercury. Bilateral cataracts were present, obscuring visualization of the fundi. The neck veins were engorged and hepatojugular reflux was present. The heart was enlarged to the left and a harsh apical systolic murmur was present. There was mild respiratory distress and a few crackling rales were present at the lung bases. Neurologic examination revealed bilateral absence of the ankle jerks.

Laboratory examinations revealed two plus albuminuria and no glycosuria; blood urea nitrogen, 118 mg. per 100 ml.; cholesterol, 113 mg. per 100 ml.; and fasting blood sugar, 83 mg. per 100 ml.* Total protein was 7.1 gm. per 100 ml. with albumin 2.8 and globulin 4.3. Esbach determination showed 1 gm. of albumin per liter of urine. Liver biopsy revealed only passive congestion. A plain film of the abdomen showed calcification of the splenic artery. Results of lumbar puncture were normal except for an increase of the spinal fluid protein to 104 mg. per 100 ml.

A diagnostic problem existed in that the entire picture could not be explained by congestive heart failure, but there seemed to be an associated element of renal disease. In view of the extensive anasarca, the family history of diabetes, the absent ankle jerks, peripheral vascular disease, calcification of the splenic artery, and the elevated spinal fluid protein, diabetes was suspected despite the normal fasting blood sugar and absence of glycosuria. A two-hour postprandial blood sugar showed a diagnostic abnormal elevation to 190 mg. per 100 ml. A glucose tolerance test confirmed this finding.

Because of the difficulties in treating the edema, therapy with prednisone was instituted. This resulted in greatly increased diuresis and the development of obvious clinical diabetes. A renal biopsy done at this time revealed the classic Kimmelstiel-Wilson lesions in the renal glomeruli (Fig. 4).

Comment.—The presence of disproportionate anasarca, absent ankle jerks, peripheral vascular disease, and calcification of the splenic artery in a female, the family history of diabetes, and the elevation of spinal fluid protein suggested the diagnosis of diabetes. This was confirmed by a two-hour postprandial blood sugar determination, a glucose tolerance test, and later, steroid diabetes. The renal biopsy, which revealed nodular glomerulosclerosis, is now readily understandable on a diabetic basis. At the same time, the biopsy helped considerably in evaluating and understanding a complex clinical picture.

Case 2.—This 58-year-old Negro woman was ad-

*Blood sugar determinations were made using the Somogyi method. By this method 100 mg. per 100 ml. is the upper limit of fasting blood sugar, and 120 mg. per 100 ml. is the accepted upper limit of the two-hour postprandial value.

mitted to the Mount Sinai Hospital because of vomiting, weakness, and dizziness. For the previous year she had manifested the classical evidences of a nephrotic syndrome, accompanied by azotemia, anemia, lethargy, nausea, and failing vision. Several weeks before, she had noted an ulcer on the right big toe. Family history revealed that her mother had diabetes mellitus. There was no history of diabetes in the patient.

On examination generalized anasarca, including facial edema, was present. The patient was lethargic and there was a uremic odor to the breath; blood pressure was 160/100 mm. of mercury; fundi showed advanced retinopathy with exudates and hemorrhages; there was no evidence of congestive heart failure; all peripheral pulses were palpable; there was a necrotic ulcer at the tip of the right toe with surrounding cellulitis; neurologic examination revealed absence of the knee jerks, the ankle jerks, and vibratory sensation in the toes.

Laboratory examinations revealed two- to four-plus albuminuria with many white cells and rare casts; hemoglobin, 5 gm. per 100 ml.; blood urea nitrogen, 50 mg. per 100 ml.; total protein 5.5 gm. per 100 ml. with albumin 2.6 gm. and globulin 2.9 gm.; cholesterol 310 mg. per 100 ml., and creatinine 7.6 mg. per 100 ml. Esbach determination showed 5 gm. of protein per liter of urine. Fasting blood sugar was 64 mg. per 100 ml. The electrocardiogram was unremarkable.

The patient at first was thought to have a nephrotic syndrome secondary to chronic diffuse glomerulonephritis. However, the absent knee and ankle jerks, the necrotic ulcer of the toe in the presence of good pulses, and the family history of diabetes mellitus strongly raised the question of diabetes mellitus even though the fasting blood sugar was normal. An oral glucose tolerance test showed a two-hour value of 133 mg. per 100 ml.; the intravenous glucose tolerance test showed a rise at one-half hour to 220 mg. per 100 ml., and at one hour to 191 mg. per 100 ml. These abnormal values validated the diagnosis of diabetes.

The patient was readmitted six months later with acute pyelonephritis. There was now a significant increase in azotemia with a rise of the blood urea nitrogen to 146 and the creatinine to 17.4 mg. per 100 ml. The urine was loaded with gram-positive cocci and white cells in addition to the persistent four-plus albuminuria. A blood culture yielded paracolon bacillus. Although energetically treated, the patient died in renal coma shortly after admission.

At autopsy, classical Kimmelstiel-Wilson lesions, namely nodular hyalinization of the glomeruli, as well as arterio- and arteriolosclerosis were present; in addition, acute pyelonephritis was evident.

Comment.—The presence of a nephrotic syndrome, a family history of diabetes, a necrotic ulcer of the toe in the presence of good pulses, and the absence of deep reflexes pointed to the diagnosis of diabetes. The elevated glucose tolerance test curves were corroborative. This could easily have been designated another "unexplained case" of Kimmelstiel-Wilson lesion unrelated to diabetes.

Other cases further illustrating this point have been encountered.[3, 4, 8]

It is evident that nodular glomerulosclerosis may occur in the absence of manifest aberration of carbohydrate metabolism. This may well account for those few cases in the literature where this lesion is said to occur in nondiabetics. Hence, it would augment the evidence for the specificity of this lesion in the pathologic documentation of diabetes mellitus. Diabetes must be suspected in the differential diagnosis of any renal lesion of uncertain or unproved etiology. Such suspicion may be enhanced by the presence of a family history of diabetes, retinopathy, neuropathy—especially as manifested by absent deep reflexes—peripheral gangrene, or severe arteriosclerosis. The presence of any of these alone or in combination should lead to further investigation of the carbohydrate metabolism. The finding of a normal fasting blood sugar is without diagnostic significance in these and comparable situations. It is essential to do at least a blood sugar determination two hours after administering 100 gm. of glucose, followed as indicated by a glucose tolerance test. Only in this way can the unknown and unsuspected diabetic be detected.

It is increasingly evident that diabetes mellitus is a generalized fundamental disease process, only one aspect of which is a disorder of carbohydrate metabolism. Other aspects include retinopathy, neuropathy, arteriosclerosis, and the complications of pregnancy. This concept carries with it the implication that any or all of these lesions may occur independently of hyperglycemia and glycosuria and, by the same token, may be the presenting clinical manifestation of diabetes mellitus.[5] It further implies that those facets of diabetes mellitus other than abnormal carbohydrate metabolism are not complications of diabetes but are integral parts and basic manifestations of the syndrome of diabetes mellitus, probably genetically conditioned and related.

It is difficult to impugn poor control as the only basis for these lesions when alteration of the glucose metabolism may be so inconspicuous. It follows that insulin insufficiency need not be a factor in their development. Whether there is some unknown basic feature common to all these facets of diabetes, or whether there is another hormone involved in addition to insulin is speculative.

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Pyelonephritis.—This occurs more frequently in diabetics and is a potentially serious complication. Since infection in the well-controlled diabetic (as determined by the broadest criteria) is hardly, if at all, more common than in the nondiabetic, other factors must be at play. Studies implicate neuropathy with neurogenic bladder disturbance leading to urinary stasis and secondary infection as the pathogenetic basis of pyelonephritis in diabetes. When there is a complete bladder paralysis, this is readily apparent. However, there are incipient and less advanced conditions which are more common and where the residual urine may be only 200 to 300 cc. Our data indicate the relatively frequent occurrence of these situations and the high degree of correlation with pyelonephritis.[1] There is reason to believe that the infection is conditioned by the stasis in the flow of urine.

In obstructive urogenital conditions, the paucity of cures and frequency of recurrences in chronic urinary tract infections is well known. These infections are often refractory to antimicrobial therapy, not only because of inadequacy of drug therapy, but primarily because of the element of obstruction.

Since in many cases removal of the obstructive process results in a return of normal renal function it is mandatory that the disease be diagnosed before irreversible changes occur from hydrostatic pressure and infection. Early bladder dysfunction is readily overlooked, for the disturbances in urination tend to be insidious in onset. The earliest symptom is difficulty in emptying the urinary bladder, then incomplete emptying, then a prolongation of the interval between voidings and thus the establishment of residual urine. Significant urinary obstruction may exist despite adequate urinary output.

Therapy is determined by the extent of involvement but always requires urinary diversion, be it catheter drainage, suprapubic cystotomy, or transurethral resection of the vesical neck, all accompanied of course by antibiotic and chemotherapeutic agents. At times these measures have a dramatic impact on the clinical picture, and may be literally lifesaving.

The importance of considering the possibility of obstruction in all cases of urinary infection and azotemia in the diabetic must be emphasized. Present knowledge indicates that this is the only aspect of diabetic nephropathy which may lead to a direct and rewarding therapeutic approach.

Relationship of Nephropathy to Severity of Diabetes.—Although there have been some claims that nephropathy in diabetes occurs primarily in the mild diabetic and rarely in the ketotic-prone diabetic with previous history of coma, they have not been substantiated. However, the observation that the diabetic state may be remarkably improved with sharp reduction in insulin requirements or even elimination of insulin, has been repeatedly confirmed. Because decreasing insulin requirement is not necessarily confined to progressive diabetic nephropathy, but is reportedly observed in diabetics with any form of renal lesion leading to azotemia, this may be a phenomenon without specificity. It has been attributed to the anorexia of azotemia and the unpalatability of the prescribed low-salt diet. These are admittedly important factors, but recent investigative reports have yielded some intriguing attempts to further elucidate this apparent improvement in carbohydrate tolerance. The impetus to these investigations was supplied by the observation of the frequent occurrence of abnormal glucose tolerance tests in azotemia—entirely independent of diabetes.[2] A summary of the experimental results indicates that there is no apparent deficit of insulin synthesis or release in these circumstances; further the experimental efforts indicate hypoglycemic, epinephrine, and glucagon unresponsiveness; the experiments further indicate normal peripheral glucose utilization. These factors suggest a deficiency in hepatic glycogen storage and release.[12] This could explain at least part of the amelioration of diabetes with azotemia, as measured by insulin requirements.

Treatment

Diabetic Nephropathy.—No specific therapy is available at present, and symptomatic therapy is the mainstay of management. The diabetes is relatively easy to manage in terms of insulin and diet.

The blood pressure may be controlled by the use of antihypertensive medication, but care must be exercised to prevent sudden falls in blood pressure with acceleration of

uremia. In the presence of poor renal function, the dietary protein should be reduced to 30 to 50 gm. per day, but adequate caloric intake by carbohydrate and fat supplementation must be assured to prevent malnutrition, especially in the presence of severe proteinuria. The fluid intake should be scaled to the point of producing a urine volume of 1200 to 1500 cc. daily, lest nonprotein nitrogen retention develop more rapidly.

The management of edema may be difficult because of the multiplicity of causative factors, such as renal insufficiency, cardiac decompensation, and hypoalbuminemia. The rate of formation of edema depends as usual on the intake of salt and water regardless of the basic cause of the edema. Salt restriction will therefore delay quantitatively the accumulation of edema and may even lead to its mobilization. However, the degree of cardiac failure, and especially the degree of renal insufficiency will be deciding factors in this process. Drastic reduction in salt intake should be carried out only under careful supervision of blood and urinary electrolytes.

Patients with cardiac failure are maintained on digitalis and may be given mercurial diuretics as long as they respond and renal insufficiency is not too severe. Ammonium chloride and other acid-forming salts may be helpful in promoting mercurial diuresis when renal function is adequate, but the patient should be observed carefully for the development of acidosis. The benzothiadiazines may be of considerable benefit. Aldosterone antagonists, particularly in combination with the benzothiadiazine agents, may help in keeping the patient edema-free.

The evaluation of a possible underlying neuropathic vesicle involvement is mandatory. If present, therapy offers the most satisfactory approach at this stage.

Complications of chronic renal failure, as well as associated clinical conditions which may further aggravate the underlying situation, must be constantly kept in mind since these may be remediable, and their intelligent management may do much to maintain comfort and prolong life. These include disorders of fluid and electrolyte balance, severe anemia, renal and extrarenal infection, possible associated obstructive uropathy, and gastrointestinal disturbances with resultant nutritional vitamin depletion.

There is no known method of reversing the pathologic process of diabetic glomerulosclerosis. The effectiveness of the rice diet and other low salt diets is not proved. Reports of alleviation of diabetic vascular disease following pituitary ablation or bilateral adrenalectomy have been published; on the whole the results of these drastic procedures have not been encouraging insofar as nephropathy is concerned. Improvement, at best, has been limited to hemorrhagic retinopathy. Much more study is required. Until then the procedures must be viewed only as experimental.

Summary

1. Diabetic nephropathy is a conglomerate of glomerular, vascular, and infectious components with no reliably predictable clinical counterpart.

2. The lesion of nodular glomerulosclerosis is pathognomonic for the diagnosis of diabetes and may exist without overt carbohydrate abnormality.

3. The importance of possible neurogenic vesicle dysfunction as a component of diabetic nephropathy is stressed in view of its therapeutic implications.

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Diabetic Diarrhea

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REVIEW ARTICLES

DIARRHEA in the diabetic patient may be the result of any of the recognized factors that produce diarrhea in the nondiabetic. In addition, there are at least three categories of diarrhea which are either specific for diabetes or so intimately allied that they merit detailed consideration.

One form of diarrhea occurring in the course of diabetes has sufficiently distinguishing characteristics to be a recognizable clinical entity, and is referred to as diabetic diarrhea. The term "diabetic enteropathy" effectively denotes that the clinical symptomatology is attributable to the dysfunction of the small intestine.

Malabsorption syndrome has been observed in diabetics with diarrhea. The unresolved relationship between these two syndromes is a matter of intense interest.

The diarrheas that are pancreatic in origin often occur in conjunction with a diabetic state.

Diabetic Enteropathy

The syndrome of diabetic diarrhea was first recognized in 1936.[2] It was differentiated from pancreatic diarrhea by the demonstration of normal exocrine pancreatic secretion. Administration of active pancreatic juice to these patients was completely lacking in therapeutic efficacy in contrast to its success in cases of diarrhea resulting from chronic pancreatitis. In 1945 the frequent association of this condition with evidences of diabetic neuropathy and specifically with evidences of autonomic nervous system involvement was noted.[12] The latter included such manifestations as gastric atony, orthostatic hypotension, and neurogenic vesical dysfunction. Sheridan and Bailey[13] stressed the intermittency of the diarrhea, its nocturnal exacerbation, and the frequent occurrence of nocturnal fecal incontinence. They also stressed the frequent presence of neuropathy and, in cases with neuropathy, the almost constant presence of elevated spinal fluid protein.

Further study added the existence of a disordered motor function (deficiency pattern) of the small bowel visualized by x-ray examination.[9] There was segmentation, considerable variation of the caliber of the intestinal lumen, localized coarsening, scattering, puddling, irregularity, and partial obliteration of folds with preservation of the mucosal pattern (Fig. 1) ; occasionally, varying degrees of gastric retention were noted. This observation indicated, for the first time, that diabetic diarrhea was related to small bowel dysfunction. However, there was little correlation between clinical symptoms and the type or degree of abnormality seen on roentgenograms.

Steatorrhea was not documented in diabetic diarrhea until 1956.[4] The symptoms in these cases, namely intractable diarrhea with postprandial and nocturnal exacerbation and fecal incontinence, were in no way different from those cases previously reported in which steatorrhea had not been demonstrated. Notwithstanding the presence of steatorrhea, these patients did not have clinical evidences of a malabsorption syndrome. Finally in 1960 the association of malabsorption syndrome with diabetes was first recognized.[7]

Although diabetic patients are subject to diarrhea caused by any recognized factors, they are particularly prone to diabetic diarrhea; malabsorption syndrome and diarrheas that are pancreatic in origin must be differentiated.

Fig. 1. Normal small bowel mucosa in diabetic diarrhea.

Ordinarily these patients present with a history of diarrhea, usually of several months' or, less frequently, of several years' duration. The intermittency of the diarrhea with spontaneous exacerbations and remissions of unpredictable and varying length forms a highly characteristic and constant feature of the clinical picture. During the acute phase, loose stools are passed frequently, sometimes as many as 20 to 30 a day. As a rule, most of this diarrhea takes place in the late evening, night, and early morning. Nocturnal fecal incontinence is not uncommon and is often a source of great embarrassment to the patient; some of our patients have deliberately withheld information of diarrhea so as to avoid discussing the incontinence.

The diarrhea is usually unassociated with significant abdominal distress or rectal tenesmus. The stools, which do not contain pus or blood, are watery, brown, and relatively homogenous unless steatorrhea is present.[6]

On examination the patients as a group are surprisingly well-nourished and usually appear undistressed in spite of the discomfort and annoyance of the clinical manifestations. Weight loss ordinarily is not remarkable. Abdominal and rectal examinations are noncontributory. The only significant physical findings are the almost uniform presence of evidences of diabetic neuropathy, including other manifestations of autonomic nerve involvement.

Several authors have reported that the incidence of achlorhydria and achylia gastrica is increased in diabetics as compared to the nondiabetic population. However, a recent investigation indicated that acid secretion does not tend to be low in diabetics, that diabetes per se is not a cause of progressive diminution of acid secretion, and acid secretion is not influenced by the severity or duration of the diabetes.[11] In the author's cases, the significance of impaired gastric secretory activity has been inconspicuous and in one of the most severe cases, the gastric secretion was abnormally elevated.

In cases of suspected diabetic enteropathy, the enzymatic components of the exocrine pancreatic secretions are uniformly normal. The various parameters ordinarily relied on in the study and the detection of malabsorption syndrome have been applied in the study of many cases. Vitamin A fasting levels, vitamin A absorption tolerance curves, blood carotene levels, prothrombin times, Schilling test, serum calcium, phosphorus, alkaline phosphatase, measure of fat excretion in the stool over a 72-hour period, and the xylose excretion test must be determined. Although in no one patient have all these tests been consistently abnormal, it is significant that in many of our patients one or more of these tests have been abnormal. Whether this is any indication of a relationship to malabsorption syndrome, or whether these findings are a manifestation of diabetic diarrhea, entirely unrelated to the sprue syndrome, will be discussed later. The relationship, if any, is still conjectural.

The x-ray findings in the small bowel characteristically reveal a pattern of disordered motility. Of the radiologic signs mentioned, increased secretion and areas of dilatation are among the more consistent manifestations. Although these abnormalities are present in most cases, nevertheless there are some cases of diabetic diarrhea where abnormal x-ray findings are minimal or even entirely absent. It is of interest that the severity of abnormality as visualized by the x-ray is not necessarily related to the severity of the clinical manifestation. In addition, during the periods of remission, the x-ray abnormality persists.

Carefully controlled postmortem study of the intestinal tract of diabetic patients with diarrhea, diabetic patients without diarrhea, and nondiabetic patients failed to reveal any distinguishing characteristics in the mucosal, muscular, neurologic, or vascular components.[3] There is only one case report of a postmortem examination in a patient with diabetic diarrhea and steatorrhea which mentions definite abnormalities in the my-

enteric plexus of the small bowel.[8] One of the difficulties is that little is known of the normal pattern of the nerve plexuses of the small bowel and considerably less of any pathology in this area. On clinical grounds there is good reason to anticipate that more intensive study in this direction, with a greater appreciation of abnormal deviations, will be fruitful.

In our patients with diabetic diarrhea without malabsorption syndrome, the small bowel mucosal biopsy has revealed consistently normal findings[6] (Fig. 2); this has been the experience of other investigators. The pathology as encountered in malabsorption syndrome will be discussed later.

Pathogenesis.—Despite the inability to detect histologically the involvement of the autonomic nervous system in the small bowel, there are nevertheless several features which tend to implicate this as the most likely factor. The most impressive is the frequent occurrence of associated involvement of one or more components of the autonomic nervous system. Further, autonomic nervous system involvement in other situations such as post-vagotomy, or following resection of the sympathetic ganglia, has also been observed to result in diarrhea, with coexisting comparable x-ray changes. There are few medical diseases that present clinical evidences of autonomic system involvement and among these, diabetes is probably the most prominent.

Fig. 2. Motility disorder pattern of small bowel as seen in patients with diabetic diarrhea.

Although gastric atony and gastric retention frequently coexist with diabetic diarrhea, there are many cases of diabetic diarrhea without associated gastric atony. Of practical interest is the fact that the presence of an atonic stomach with a relatively nonfunctioning pylorus appears important in the failure to successfully perform small bowel biopsies. In our hospital experience, attempts at small bowel biopsy have been attended by a remarkably high percentage of success in all conditions with the exception of diabetic diarrhea. In this latter category unsuccessful attempts have been disturbingly frequent. We attribute most of these failures to the gastric atony and the patulous pylorus. A similar experience has been recorded.[14]

In essence then, the clinical picture of diabetic enteropathy is characterized by intermittent, recurrent attacks of nocturnal and postprandial diarrhea, a tendency to nocturnal fecal incontinence, an x-ray picture of disordered motility (deficiency pattern) of the small bowel, the concomitant presence of autonomic and peripheral neuropathy, and normal exocrine pancreatic function. The diarrhea is further characterized by the absence of pus and blood and is usually unassociated with significant abdominal distress or rectal tenesmus.[6]

Malabsorption Syndrome in Diabetes

The author encountered five cases of malabsorption syndrome which, superficially at least, could be and were originally accepted as cases of diabetic diarrhea. Each had diarrhea consisting of copious, greasy, light-colored, foul-smelling stools. The stool contained fat globules microscopically, and frank steatorrhea was demonstrated in 72-hour stool collections. In addition to a low fasting blood vitamin A level, there was impaired absorption of vitamin A as determined by the tolerance test. The blood carotene level was low. The x-ray examination of the small bowel revealed the characteristic picture as observed in nontropical sprue, namely marked segmentation, scattering,

puddling, widening of the bowel, irregularity, and increased secretion (Fig. 3). The pancreatic exocrine studies revealed normal findings. Biopsy of the small bowel mucosa, which was done in four of the five patients, revealed a uniform picture of clubbing and stunting of the villi, complete absence of the normal delicate villi, edema, and considerable cell infiltration including eosinophilia

Fig. 3. In a biopsy of the small bowel in a case of malabsorption syndrome, clubbing, edema, and cellular infiltration can be seen.

Fig. 4. Raentgenagram shows the small bowel changes in malabsorptian syndrame and diabetes.

(Fig. 4). This description, of course, is identical with that of nontropical sprue. Therapeutically these cases responded dramatically to steroids.[7] One patient was placed on a gluten-free diet and has shown satisfactory improvement.

Whether these cases represent a coincidental occurrence of diabetes and nontropical sprue, or whether they represent a specific manifestation of diabetes per se may be questioned. In favor of the former are the identity of the clinical, pathologic, and therapeutic findings with nontropical sprue. In diabetic diarrhea without malabsorption, the small bowel mucosal biopsy has been uniformly normal and there has been no responsiveness to the administration of corticosteroids. On the other hand several factors suggest a possible relationship between these manifestations, thus forming a separate entity: (1) Such association has been only recently observed. (2) In the past a flat glucose tolerance curve has been found so uniformly that it is considered a diagnostic characteristic of sprue. (3) Steatorrhea has been observed not infrequently in cases of diabetic diarrhea even though the other components of malabsorption syndrome have not been present. (4) In the author's cases with a malabsorption syndrome and diabetes of long duration (three over 30 years) there are remarkably few evidences of vascular degenerative changes that one ordinarily anticipates. (5) Many of our cases of diabetic diarrhea and of diabetes with malabsorption had laboratory findings that were either borderline or crossed the dividing line in both directions; in this connection there is a hybrid case report[5] in which there was observed severe diabetic neuropathy with diabetic diarrhea, gastric atony, and a full-blown malabsorption syndrome. (6) Sprue itself is a heterogenous syndrome which is already being broken up into gluten-free diet responsiveness and unresponsiveness, and specific unrelated etiologic entities such as total gastrectomy, partial gastrectomy, Whipple's disease, hepatic disease, duodenal and jejunal diverticulitis, Hodgkin's disease and other lymphomata, granulomatosis, jejunal ileitis, entero-enteric fistulas, gastroileostomy, regional enteritis, amyloidosis, agammaglobulinemia, infarction of the bowel, ulcerogenic tumor of the pancreas (Zollinger-Ellison syndrome), absence of beta-lipoprotein, chronic pancreatitis, carcinoma of the pancreas, primary biliary cirrhosis,

Dr. Ellenberg, associate attending physician at Mount Sinai Hospital, New York, presented this paper before the April, 1964, meeting of the Texas Diabetes Association in Houston.

carcinoid tumor, hookworm disease, *Strongyloides stercoralis*, carcinoma of cecum, and neomycin administration. Several other points require emphasis. For example, it has been shown that successful treatment of some cases of nontropical sprue results in reversion of the characteristic pathologic villiform changes of the small bowel mucosa to a relatively normal state. Following neomycin administration definite small intestinal mucosal changes have been observed in association with the production of a malabsorption syndrome. The changes in the small bowel consisting of clubbing, edema, and cellular infiltration are similar to that of sprue. On withdrawal of the drug both the malabsorption syndrome and the pathologic changes are reversed.[10] This suggests that the accepted pathologic histology of sprue is not necessarily specific. Studies of small bowel biopsies following partial gastrectomy have also revealed changes comparable to those assumed to be diagnostic of sprue.[1] Thus the sprue syndrome is not necessarily a disease entity; instead, it may comprise several entities. This syndrome may well represent a nonspecific response of the small bowel to many unrelated pathogenetic mechanisms, conditioned by a limited repertory of reactivity of the intestine. Regardless of the moot question of relationship, the importance of recognizing the association of diabetic diarrhea with malabsorption cannot be overstated, because in this syndrome the administration of corticosteroids has a consistent, favorable, and at times, even a lifesaving effect.

Pancreatic Diarrheas

Special mention must be made of the diarrheas in chronic pancreatitis, carcinoma of the pancreas, and cystic fibrosis of the pancreas which may simulate diabetic enteropathy. These pancreatic diarrheas, which usually lead to steatorrhea, are prone to exhibit roentgenologic changes in the small bowel.

For the differentiation between these pancreatic diarrheas and diabetic diarrhea, the abnormal diminution or absence of exocrine pancreatic secretions is all-important. By the successive use of I^{131}-labeled oleic acid and I^{131}-labeled triolein, the impairment of digestion in pancreatic diarrhea may be distinguished from the impairment of intestinal absorption in the malabsorption syndrome. Furthermore, the diarrhea may respond to the administration of large doses of pancreatin. In addition, in chronic pancreatitis a history of recurrent episodes of abdominal pain is often elicited and calcifications in the region of the pancreas may be present. Carcinoma of the pancreas is suggested by abdominal pain coincident with the onset of glycosuria, and x-ray demonstration of infiltration of the wall of the duodenal loop with alteration of its normal configuration by an extrinsic malignancy. Cystic fibrosis (mucoviscidosis) is characterized by recurrent, severe, prolonged respiratory infections, malnutrition, and a positive sweat test.

It follows that the recognition of diabetic enteropathy per se remains entirely a diagnosis by exclusion since there are no pathognomonic symptoms, signs, or laboratory findings. In the absence of any other definitive diagnosis, the history of intermittent episodes of diarrhea with spontaneous remissions, nocturnal fecal incontinence, normal pancreatic exocrine studies, x-ray picture of a disordered motility pattern of the small bowel, and presence of neuropathy are suggestive of this condition.

The presence of a malabsorption syndrome and of pancreatic diarrhea can usually be proved by the means mentioned in the previous discussion of these entities.

Therapy

There is no specific therapy of diabetic enteropathy without malabsorption syndrome but the disease has spontaneous remissions. The latter make it difficult to evaluate the favorable responses which have been obtained in some cases with the use of chloramphenicol and neomycin. Nevertheless, a therapeutic trial is merited; the author administers Chloromycetin, 250 mg. three times

a day for one week, with continuing doses if there is a salutary response. The possible relationship of the effect of the antibiotics on serotonin production rather than on control of pathogenic organisms has been suggested and is in the process of exploration.[15] The results of the administration of vitamin B_1, vitamin B_{12}, liver, pancreatic extracts, and vagolytic agents have not been encouraging.

In contrast, the response to corticosteroids of all reported cases of malabsorption syndrome is prompt and dramatic. This treatment rapidly results in cessation of diarrhea, weight gain, return of appetite, energy, strength, increase in well-being, and reversal of the abnormal biochemical findings. The increased glycosuria that may accompany the corticosteroids is readily controlled by an appropriate increase of hypoglycemic agents. Insufficient experience prevents any comment on the therapeutic effectiveness of a gluten-free diet; one patient treated with this regimen is doing well.

The therapy of the diarrheas resulting from disorders in the pancreas, as well as all other causes of diarrhea, is identical in diabetic or nondiabetic patients.

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Defect Found Among Amish Believed To Be Genetic

The excellent genealogic records of the Old Order Amish have helped medical scientists discover a possible genetic basis for an anatomic malformation, which, though rare, can prove fatal.

The defect occurred in two pairs of sisters born to Amish parents living in Lancaster County, Pa., and in western New York state, according to a report in the Sept. 14 *Journal of the American Medical Association*. Termed hydrometrocolpos, the defect is an abnormal wall within the vagina preventing drainage of a watery fluid which collects in most newborns.

The genealogy of the two families revealed that all four parents descended from Christopher Beiler, born about 1727, and his wife, Barbara Yoder, suggesting that a gene responsible for the defect was carried by Beiler or his wife.

The Amish population of more than 40,000 is divided into many small inbreeding groups, each with a high rate of consanguinity.

An inbred group can provide an opportunity to discover "new" recessive disorders, according to an accompanying *Journal* editorial. Some recessive genes may be so rare that under ordinary circumstances persons are affected very infrequently.

The editorial said "a great deal could probably be accomplished in the way of reducing the frequency of certain recessive disorders if the Amish of different communities would intermarry."

Management of Diabetes Mellitus In Adolescent Patients

GEORGE H. LOWREY, M.D.

CLINICAL MEDICINE

GREAT DIFFICULTIES are encountered in managing diabetes in adolescents. There are many reasons. The more common problems are reviewed, and some suggestions are given for handling them.

As the diabetic child enters and passes through adolescence, the control of his diabetes has a tendency to deteriorate.[2, 3, 13] I observed 76 children from the time they were 11 until they reached age 17. Poor control increased in incidence up to age 14, and then was maintained at an even level through age 17. I based the measure of control on the level of fasting blood sugars, glycosuria as determined three to four times daily, and the frequency of complications requiring medical supervision. In his review of diabetic school children of Stockholm, Sterky[13] states, "the frequency of cases of poor diabetic control was 18% in the prepubertal group and 42% in the postpubertal group."* These findings were based on much the same measurements of control as I used. In both series of patients, larger doses of insulin were required in the girls than in the boys. In my series, an average of 77 units daily was necessary for the girls and only 59 units for the boys. A contributing factor is the decreased physical exercise and the increasing obesity in the girls when compared to the opposite sex.

As the child with diabetes grows older there is a steady increase in insulin requirements once stabilization has been accomplished; this increase roughly parallels the age of the child or his body weight.[9, 16] During the adolescent growth spurt, insulin requirements increase out of proportion to the increase in height or weight. The larger dose of insulin tends to increase the chances for both hyper- and hypoglycemia. "Juvenile" diabetics lack the cushion of residual endogenous insulin that the "adult" diabetic has.

The physical growth and sexual maturation during adolescence are important elements of this picture. The pituitary growth hormone has some anti-insulin, or hyperglycemic, effects. On the other hand, we cannot be sure that this hormone is especially important in the problems that we are considering. First, there is no proof that the hormone is increased in amount at the time of adolescent growth. Second, there still is debate concerning the existence or nature of the hyperglycemia effect when growth hormone is at physiologic levels. Some evidence indicates that the hyperglycemic factor may be independent of the growth hormone.[15] The increase in body mass which causes greater insulin requirement also results from the gonadal hormones. Both androgenic and estrogenic hormones are increased as adolescence progresses, and this causes not only maturation but also growth in size. There is no evidence that these hormones interfere in any way with the action of insulin or that they are directly hyperglycemic.

With the accelerated rate of growth there is an increase in food consumption. This adds to the insulin requirements and con-

*Sterky uses "postpubertal" when referring to girls 13-16 years old, and boys 14-18 years old.

Physical growth, sexual maturation, emotional instability, and rebellion against authority are normal in adolescence. Singly and in combination, these characteristics affect the control of diabetes. Successful management depends on both the psychological approach and medical treatment.

tributes to the "brittleness" of the patient's condition. Girls today omit breakfast often. This is apparently a status symbol. The desire to remain slender and the misconception that one can decrease the total caloric intake by avoiding breakfast are partly to blame. Skipping breakfast usually leads to a still greater consumption of calories at other times during the day. In the diabetic this adds to the confusion by precipitating hypoglycemic episodes in the late morning hours and possible hyperglycemia later in the day.

Reduction or lack of physical exercise also has a deleterious effect on the control of diabetes. Muscular work reduces requirements of insulin in all patients.[6,10] The precise mechanism of reduction is not presently known, but the effect is so noticeable that some observers have postulated that an insulin-like substance is produced by the working muscle.[5] There is little need to encourage young children to exercise as this is a natural attribute of the age, but as they approach adolescence exercise seems to decrease to a great extent. This is particularly true of girls, and contrary to some government authorities, it is not unique to the United States as Sterky[13] emphasizes in discussing diabetic children in Stockholm. Lack of exercise also predisposes toward obesity, and this combination occurs frequently. The association of reduced physical exercise, moderate obesity, large doses of insulin, and poor control was seen in girls more often than boys in a ratio of about eight to one in my group of children.

The character of the adolescent is one of rebellion and revolution. These youngsters are caught in the emotional conflict between complete independence and reliance on their elders for economic aid as well as for knowledge that comes with experience. The extremes of dress and fashion that are so typical of this age reflect these characteristics better than any clinical description. Adolescents are terribly concerned about themselves, their appearance, general health, and functions of various portions of the body. When some part or parts do not function in a normal way they tend to become hypochondriacal. Adolescents need to conform to the group for security as well as acceptance by their peers. Anything that sets one aside or makes him different will have great emotional implications.

To quantitate the part that emotional responses play in the management of the youthful diabetic is difficult. The diagnosis of diabetes is a shocking experience both for the child and his family, and the effects of it are never completely overcome. It is paradoxical that thorough education concerning the nature of the disease, and the techniques that must be learned for adjustment to it, exposes the patient to the knowledge of possible complications, and fears naturally occur. It is important to transform these fears into a stimulus leading to cooperation and the best possible control. Continuing tension and anxiety must be avoided for they will disrupt a smooth diabetic course.

The diabetic child's most frequent verbal complaint concerns the dietary restrictions which he must face every day of his life. This is not surprising, because it is the diet which interferes most frequently with his ability to copy the behavior of his peers. The diet is the most often ignored instruction that we give.[8,14] Although some authors state that insulin injections are the procedures to which the greatest objection is raised, this certainly has not been my experience. Second only to diet is the active rejection to performing and recording urinalyses. The stigma of being diabetic almost has been outgrown by the time these patients reach adolescence. Nevertheless, a certain degree of stigma always remains as a source of irritation.

Since diabetes enforces a certain way of life, the child and his family will react according to preexisting patterns. I always emphasize that this is a disease of the family and not of a single individual in the family. Although the attitudes of both the mother and father are important, the mother's attitude usually has the greatest effect. Bruch[1] has pointed out that her reaction may take one of three general forms: (1) Perfection. There is good control of the diabetes but poor development of the child's personality. (2) Tolerance. The disease is accepted as a part of the family situation, and control is often good and normal development of the child results. (3) Hostility and masochism. This results in poor control, and the disease frequently is used as a weapon in the battle between child and parent. I have seen a number of fathers who are in this category. They react to the child's disease as though it were a smirch on the "good family name." They seem to resent

the lack of perfection, and subsequently reject the child and avoid a close relationship with him. Each of us who has cared for diabetic children will recognize the three categories, and would agree that the first and third categories are important reasons for failure to maintain satisfactory control.

A final emotional factor concerns the child's attitude toward the future. He or she cannot help learning of the complications which can occur, and this in turn leads to periods of serious depression. Many diabetic patients have periods of unreasonable behavior which can be interpreted as self destructive. Although the psychiatrists say that similar behavior may occur in adult diabetics, it is much more prominent in young patients.[14] Such behavior is most frequent when major adjustments must be made, and when the emotional and socioeconomic environments of the home are suboptimal. As a result of this type of attitude, the child will go on eating binges and purposely reduce or omit his insulin. I had one teenage boy hospitalized for mild or severe acidosis 11 times in a 12-month period. Intensive psychotherapy in this instance was beneficial.

Methods of Management

These are some of the problems encountered in treating the adolescent with diabetes mellitus. The following methods of approaching them probably are not unique, but I find them useful.

A diet is provided which is adequate to meet growth and exercise requirements. I know of nothing that will lead more quickly to rebellion against the diet than to underfeed these young people. If obesity is a problem, it must be faced squarely and discussed honestly. Most of these patients are vain enough that they wish for a good appearance, and in contrast to younger children and adults, they often will accept a diet that will cause them to lose weight. To avoid large amounts of carbohydrates at any one feeding, we rarely exceed a daily total of 250 gm. of carbohydrate regardless of the total number of calories in the diet. Because I most commonly use NPH or lente insulin, five feedings in the day are used with the three usual meals and afternoon and bedtime snacks. All feedings must contain some protein both to satisfy hunger and to give a duration of protection against possible hypoglycemia. Approximately 20 percent of the total calories is in the form of protein as it must be remembered these are growing children.

To induce the girls to increase their exercise, I encourage them to buy the Royal Canadian Air Force booklet, "XBX Plan for Physical Fitness." (A similar booklet has now been published by our own government.) I must admit that the reception of this suggestion has not been entirely successful. The present generation of maturing girls seems to regard physical exercise as an anathema. Many schools in Texas have no physical education program for girls beyond the eighth or ninth grade. Occasionally, by involving the whole family in a sports program, the goal is accomplished. Unfortunately, I have done such a good job of teaching these children that the size of insulin dosage is relatively unimportant that I can no longer use the reduction of insulin dosage as an inducement for them to increase physical activity.

Relative to the dosage of insulin it is important to emphasize that the Somogyi effect is present in some juvenile diabetics.[11,12] The exacerbation of diabetes by excessive insulin dosage results from stimulation of the adrenal glands in response to asymptomatic hypoglycemia. First there is excessive output of epinephrine, and this is followed by an increased output of the cortical steroids, both as physiologic defense mechanisms. These raise the blood sugar level and cause glycosuria, and the patient's interpretation is that he needs more insulin. This vicious cycle eventually results in ketosis, hyperglycemia, and increasing frustration for the patient and physician. It also may result in severe insulin reactions if the patient omits all or part of a meal. In relation to the insulin dosage, I never attempt to obtain the same degree of control with young diabetics as with adults. I permit and encourage a small amount of sugar in the urine in most specimens during the day. The danger of permanent damage to the brain from severe insulin reaction must always be kept in mind.[4,7]

When the dosage of insulin exceeds 60 units daily and control is irregular, I have found mixtures of insulins to be helpful. Combining regular with lente or with NPH at these relatively large total doses helps to prevent low blood sugar levels late in the day but affords a better means of keeping

the morning levels acceptable. Rarely, I have found the most satisfactory mixtures to be lente with either semi- or ultralente. I would stress that frequent changing of the quantity or type of insulin leads to trouble. Modification of the diet to obtain smoother control is more apt to be successful. Obviously, individual situations must be dealt with accordingly.

I have been disappointed in the oral preparations (tolbutamide, chlorpropamide and phenformin) as a means of aiding control in the young patient.

The need for psychological guidance for the adolescent diabetic deserves special emphasis. The physiologic as well as the emotional instability of puberty is aggravated by the peculiarity of the disease and the uncertainty of the patient's existence. The parents' anxiety must be allayed as much as possible by reassurance and careful education. Neither oversolicitude nor absolute perfectionism is desirable. The most helpful antidote for these parental reactions is a good educational introduction to the disease. No matter how hard we try, if the introduction to diabetes has been a terrifying experience for the parent or patient, and if a proper educational approach has not been made at this time, we will not be able to accomplish all of our goals thereafter.

The family and the child must be led to face the situation realistically. The fact that diabetes mellitus does exist should not be obscured. The fact that it does exist also means that a few restrictions will be imposed for the rest of the person's life, at least within our present knowledge. It should be pointed out that few people in the world live without some handicaps. As in all aspects of living, there are rules that one must follow to survive and to be compatible with society. An individual who crosses the street must look both ways in order to avoid being hit by an oncoming automobile. The diabetic must follow rules in order to avoid serious problems. Although restrictions do exist, it should be emphasized that these interfere very little with the routines of living in the modern world. Finally it should be stressed that diabetics can and do lead very successful, happy, and productive lives.

It is my firm belief that a person who is particularly interested and has knowledge of diabetes should introduce the patient and his family to the management of diabetes. Much as I respect the individual pediatrician or general practitioner, my own experience has been that they too often impart fears and wrong conceptions of the disease and its management which are extremely difficult to correct or overcome at any time in the future.

Particularly during adolescence, the diabetic may use his disease as a means to accomplish his desires, good or bad. Usually this happens when the parent has assumed too much of the responsibility for management of the diabetes. I believe that after the child is ten years old he should give his own insulin, run all of his own urine tests, and decide when and if any change in insulin dosage should be made. In our clinic, patients are educated to do all of these things, and seldom is it necessary for them to consult the physician. As a matter of fact, I think most of our patients know more about diabetes mellitus and its management than does the average physician. During the adolescent's outpatient visits I do not exclude the parent from the examining room, but my conversation is directed to the patient and not to the parent. If parents interfere I usually ask them to discontinue, and occasionally have requested that they leave the examining room. Giving the adolescent greater responsibility for his own control, and discussing situations directly with him instead of channeling dialogue through the parent will result in a sense of responsibility and satisfaction of accomplishment on the part of the patient. Such an approach will resolve many of the problems that are common to this age group.

Some of the genetic factors are discussed with the parent and the patient. I discuss these quite frankly and in terms of most recent knowledge. One must be very realistic with these children, and we do not discourage them from the anticipation of rearing a family. At the same time, it is emphasized that there are definite liabilities and that these should be taken into consideration when thinking about marriage and children. The considerable improvement in treatment over the years should be emphasized along

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with facts about further research which is already in progress. The outlook for a future generation of diabetic children is even brighter than that of today.

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Sensor To Measure Living Cell Temperatures

Engineers at the University of Texas are perfecting a sensor so tiny it can measure temperatures inside a living cell without disturbing the cell's delicate balance.

Many practical applications are foreseen for the sensor. One of the first will be a study of burns, conducted in collaboration with Dr. T. G. Blocker, Jr., and Dr. C. P. Artz at the University of Texas Medical Branch in Galveston. With the aid of the sensor, medical researchers hope to learn more about how heat is conducted from cell to cell in burn cases, and apply that knowledge in treating burn victims. The sensor will be used to record temperatures in tissue as a burn is occurring. The temperatures then will be correlated with cell damage caused by the burn. From those correlations it is expected that a better understanding of burns will be developed.

Other possibilities for the sensor include research in cancer and infectious diseases.

The sensor is a microscopic thermocouple made by joining two different metals. The junction between the two metals is a thermocouple. When heated or cooled an electrical resistance develops across the junction. The thermocouples are prepared by depositing in a high vacuum a thin layer of each metal on a quartz fiber 1/20th the diameter of a human hair. The layers overlap at the point where the thermocouple is to be located. The researchers' effort now is aimed at fixing the thermocouple at the tip end of the fiber rather than along the axis and improving techniques for inserting the fiber into a living cell. Whole networks of such fibers could then be placed in groups of cells to continuously record temperature changes through electrical readouts.

Management of Diabetes Mellitus During Anesthesia and Surgery

CLINICAL MEDICINE

THE SURGICAL complications of diabetes, and the conditions which are complicated by diabetes, encompass almost every surgical subspecialty. The anesthesiologist is ultimately responsible for the short-term management of the diabetic state during surgical correction of these complications. Further, pituitary stalk section and adrenalectomy are surgical procedures aimed at cure or amelioration of the diabetic state or its vascular complications.[7]

Therefore, the anesthesiologist needs a store of fundamental information about the natural history of the disease.[2, 12] He must recognize the influence of such entities as infection, pregnancy, and stress, and of the various anesthetic agents and techniques on the diabetic state.[10] A knowledge of the rapidity and severity of changes in insulin requirements during affliction with these complications and after their correction is of paramount importance. Errors in management, immediately before, during, and after operation, can lead to severe acidosis, electrolyte deficiency from osmotic diuresis, or to profound hypoglycemic shock. Clinical recognition of these conditions is difficult, if not impossible, under anesthesia unless procedures for the analysis of blood sugar and acetone are available. The analyses must be accurate within the limits of clinical importance, rapid, and simple enough in all phases to be performed by an anesthesiologist without diverting his attention unduly from his patient.

Methods

The authors studied several simplified laboratory procedures[4] which are safe for operating room use and require minimal available equipment, and compared the results to standard laboratory procedures for glucose and acetone. They then applied these procedures in the management of 52 diabetic patients undergoing all types of surgical and anesthetic procedures. (Included in this figure are 11 patients who were in the first series reported.[4])

Adult stable diabetics who were *well-controlled* on diet alone, or diet plus oral hypoglycemic agents, presented few problems related to their diabetes in the anesthetic and post-anesthetic periods. Therefore, these cases were omitted from consideration here.

Diabetics who required insulin for control were given nothing by mouth for at least six hours prior to surgery. No preoperative insulin was given. Samples were drawn for blood sugar analysis by the Dextrotest method before intravenous administration of 5 percent dextrose in water. Acetone determinations were not done initially unless poor control was suspected. Throughout surgery blood sugars, urine volumes, urine sugars, and plasma acetones were done at one- to two-hour intervals. Glucose was administered during surgery as 5 percent dextrose in water or 5 percent dextrose in Ringer's lactate at a rate usually not exceeding 25 gm. per hour. Regular insulin was administered intravenously in doses of 10 to 20 units if the blood sugar exceeded 400 mg. per 100 ml. or if acetone appeared in the plasma.

In this way alterations of insulin-glucose ratio were compensated by individualized dosages, eliminating errors which may result from empirical administration of insulin.

Results

Fifty-two diabetics have been managed

Withholding insulin before surgery allows the anesthesiologist to perform simple periodic tests during the procedure, and to give insulin only in the amounts needed, if any.

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following the precepts of blood sugar analysis and appropriate insulin therapy. The following cases illustrate some unusual features which made the technique especially valuable in the patient's management.

Case 1.—The patient, a 42-year-old white man, has had known diabetes for 12 years. Three years prior to this admission, he required a change from diet and oral hypoglycemics to insulin for control of his disease. It became progressively more difficult to control, and he suffered from diabetic retinopathy, peripheral neuropathy, and recurrent ulcerations on his feet and toes. At the time of admission he was receiving neutral protein Hagedorn (N.P.H.) insulin, 35 units in the morning and 5 units in the evening. His blood sugar tests ran in the 250 to 300 mg. per 100 ml. range, but efforts to improve this level resulted in excessively low blood sugar levels. Urine tests ran two to three plus for sugar; acetone was negative. His admission hemoglobin was 7.8 gm. per 100 ml. Upper gastrointestinal series revealed a large mass in the stomach and stool guaiac tests were positive for occult blood. He was scheduled for radical gastrectomy. Induction of anesthesia was accomplished using cyclopropane, oxygen, 75 mg. of thiopental, and 80 mg. of succinylcholine. Following endotracheal intubation, anesthesia was maintained with cyclopropane, oxygen, and intermittent succinylcholine drip. The surgery required nine hours and 45 minutes and included gastrectomy, splenectomy, partial pancreatectomy, and colon interposition. During the procedure the patient received 400 ml. of 5 percent dextrose in water, 4500 ml. of Ringer's lactate, 3000 ml. of 5 percent dextrose in Ringer's lactate, and 2400 ml. whole blood. Blood sugars by the Dextrotest method were 250 to 375 mg. per 100 ml. Acetone test by the Ketostix method showed a trace of acetone early in the surgery. Ten units of regular insulin was given intravenously when the plasma showed a trace of acetone, and again when the blood sugar ascended to 375 mg. per 100 ml. The patient arrived in the recovery room awake with stable vital signs. His insulin requirement fell progressively without adjustment of dose. On the third postoperative day he became hypotensive, with a blood glucose of 50 mg. per 100 ml. By the fifth postoperative day he was under good control with two-thirds of his preoperative insulin dose.

This patient, normally very difficult to control, fared well during his arduous procedure. During the following seven months, his insulin requirement fell steadily to 10 units per day, and fasting blood glucose levels were maintained at 100 to 120 mg. per 100 ml. It is believed that this decrease in insulin requirement probably resulted from correction of the surgical disease and surgical removal of the glucagon-producing portion of the pancreas.

Case 2.—A 39-year-old white man who had been a known diabetic for two years needed surgical correction of a herniated lumbar disc. He had a long history of chronic pancreatitis with a sprue-like syndrome and epigastric pain. He had good control of his diabetes with N.P.H. insulin, 25 units per day.

Fasting blood sugar on admission was 148 mg. per 100 ml. without sugar or acetone in the urine. Insulin was withheld the morning of surgery.

The initial blood sugar by Dextrotest was less than 100 mg. per 100 ml. and by Testape was more than 65 mg. per 100 ml. The laboratory determination on the sample showed 86 mg. per 100 ml. Anesthesia was induced with 75 mg. of thiopental, nitrous oxide, oxygen, and 80 mg. of succinylcholine. Following endotracheal intubation, anesthesia was maintained with nitrous oxide, oxygen, 50 mg. of meperidine, and an intermittent succinylcholine drip. No intravenous insulin was begun. A subsequent determination revealed 150 mg. per 100 ml. of sugar and was acetone-free. Still no insulin was given. The patient received 500 ml. of 5 percent dextrose in water and 1000 ml. of 5 percent dextrose in Ringer's lactate. Surgery lasted two hours.

In the first 24 hours postoperative, the patient received 20 units of insulin and 100 gm. of glucose in 2 liters of fluid. The morning after surgery the blood sugar was 76 mg. per 100 ml. Thereafter the patient could take his diet, and control returned to preoperative levels.

This patient had sufficient residual insulin activity to assure a relatively smooth anesthetic and operative course. The story might have been different had he been given empirical doses of insulin subcutaneously or intravenously.

Case 3.—A 25-year-old white man has had known diabetes since age five, when he had his first of several comas. He subsequently had had several hypoglycemic episodes with grand mal seizures. At age 15 he required corrective glasses, but his visual acuity decreased because of diabetic retinopathy. His diabetes had been controlled recently with 25 units of protamine zinc insulin in the morning plus 10 units of regular insulin in the evening. Ophthalmology and neurosurgery consultants recommended pituitary stalk section to arrest the progress of his retinopathy. Immediately before scheduled surgery his urine showed four plus sugar and no acetone. Anesthesia was induced at 7:40 a.m. with 140 mg. of thiopental and maintained with nitrous oxide and halothane. Intermittent positive-negative pressure respirations reduced intracerebral pressure. The blood sugar at 10:25 was greater than 400 mg. per 100 ml. and plasma acetone was moderate. Fifty units of insulin were given in increments between 11:30 and 12:30. Samples taken at 30-minute intervals remained unchanged until 1 p.m., when acetone disappeared from the plasma although blood sugar remained above 400 mg. per 100 ml. He received 100 gm. of glucose and 100 mg. of hydrocortisone hemisuccinate during the procedure, which lasted three hours and 35 minutes. Postoperatively he was given cortisone, insulin, and pitressin for the onset of diabetes insipidus, which was manifest by 2.5 to 3 liters of dilute urine output daily.

This is a case of severe diabetes in which the control of hyperglycemia was impractical either before, during, or after the surgery, so enough insulin was given to prevent acidosis as measured by plasma acetone.

Case 4.—A 69-year-old white man has had diabetes mellitus for 12 years. Two months before this admission the patient had a cholecystostomy at another hospital where, in spite of 40 units of lente insulin per day postoperatively, tests showed strongly positive acetones and four-plus urine sugars. Gradually the acidosis was corrected, but the diabetes remained difficult to control.

Upon admission the patient had his cholecystostomy tube in place and was having urinary retention. He was confused and uncooperative, and was diagnosed as having "chronic brain syndrome." His blood sugar was 147 mg. per 100 ml. and urine was free of acetone and sugar. An indwelling urethral catheter was inserted and the patient was scheduled for a transurethral resection.

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Surgery was performed without difficulty three days after admission. A blood sample drawn before anesthesia showed sugar of less than 45 mg. per 100 ml. by Testape reaction. The laboratory analysis on the same sample was 35 mg. per 100 ml. Five percent dextrose in water was infused throughout the surgical procedure, and by the time the patient arrived in the recovery room his blood sugar was 200 mg. per 100 ml. and his "chronic brain syndrome" had virtually disappeared. Over the following two days, his insulin dose was reduced to 33 units, 25 in the morning and 8 in the evening.

Four weeks after surgery the patient returned for a cholecystectomy. His diabetes was reasonably well controlled. The initial blood sugar by Dextrotest was less than 100 mg. per 100 ml.

The patient received 1000 ml. of 5 percent dextrose in lactated Ringer's solution. Insulin was not begun until completion of surgery, at which time the blood sugar was 400 mg. per 100 ml. with a trace of acetone in the urine. The patient received 22 units of regular insulin in 2000 ml. of a 5 percent dextrose-containing solution in the subsequent 24 hours.

In the second postoperative day he was again placed on 33 units of lente insulin per day (25 and 8) and was maintained in his usual state of fair control.

SIMPLIFIED LABORATORY TESTS

Dextrotest (Ames Company, Inc.) is provided as a complete kit. One milliliter of whole blood is deproteinated by the addition of an appropriate tablet. Using filter paper and funnel provided in the kit, a clear protein-free filtrate is prepared. Next, the blood sugar level is determined by adding a second tablet and comparing the resulting color of the filtrate with a color chart. (The second tablet is identical with the Clinitest tablet for determination of sugar in the urine.) The level of blood sugar can be read at 100, 150, and 200 mg. per 100 ml. Additional readings to 400 mg. per 100 ml. may be made if half the original quantity of blood is used. Dextrotest is not accurate below 100 mg. per 100 ml., and its accuracy is poor in the range of 200 to 400 mg. per 100 ml. Giesecke and associates[4] have compared the Dextrotest to standard Folin-Wu determinations.

Testape (Eli Lilly and Company) is supplied in a roll. There is an accompanying color chart for comparison and interpretation. Details of this test and its value have been reported by Giesecke and associates[4] and by Seltzer.[11]

Acetest and Ketostix (Ames Company, Inc.) are used essentially in the following manner: A small pile of Acetest powder or a Ketostix reagent strip is placed on a clean piece of filter paper. One drop of undiluted plasma is added to the powder or strip. If acetonia is present, the drop will turn purple in one minute. For a report on this technique, see Duncan, G. G., and Gill, R. J.: Clinical Value of Simple Qualitative Test for Plasma Acetone in Diabetic Coma, *Diabetes*, 2:353 (Sept.-Oct.) 1953.

Discussion

The authors' technique for diabetic management during surgery differs slightly from that presented in most current references[1, 2, 3, 6, 8, 9, 13] in the following manner:

1. Preoperative insulin dose is eliminated for the following reasons:
   A. Hyperglycemia without acidosis, dehydration, or ketosis is not harmful, whereas hypoglycemia must be prevented.
   B. Duration of the procedure cannot be predicted in all cases and intravenous infusions do not always function properly.
   C. The amount of activity from prior insulin dosage cannot be predicted nor can time or type of insulin always be ascertained in emergencies.
   D. Misjudged insulin cannot be removed but more can be added if necessary.
   E. Insulin requirement may fall during surgery and in the immediate postoperative period, for example, following abscess drainage or removal of gangrenous appendix.
   F. Anesthetic agents, especially cyclopropane and ether, cause a resistance to the effects of exogenously administered insulin.[5, 10]
   G. Hypoglycemic shock is difficult to distinguish from other causes of shock without a blood sugar determination.
2. Small doses of 10 to 20 units are added to the bottles of the intravenous solution. Insulin is never given by any route except intravenously during surgery and recovery because:
   A. In the presence of altered peripheral perfusion, uptake of insulin from subcutaneous or intramuscular injection cannot be accurately predicted.
   B. When given in the intravenous solution if the source of glucose is interrupted the administration of insulin is also halted.
3. Blood sugars are measured at intervals to assess alterations in insulin requirement which may increase with anoxia, anesthesia, and stress or decrease after childbirth, amputation of gangrenous extremity, or drainage of an abscess. The authors do not rely on urine determination because of the lag of appearance of glucose in the urine and the ever-present possibility of a significant fall in urine output during surgery.
4. Blood acetones are measured at intervals because ketosis can ensue in spite of reasonable diabetic blood sugar control.
5. The measurement of hourly urine volumes enables us to establish the onset of an osmotic diuresis and take corrective action.

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Clinicopathologic Conference

R. C. Morrison, M.D.; G. C. Curry, M.D.; J. P. Sanford, M.D.;
D. W. Seldin, M.D.; P. C. Peters, M.D.; and B. D. Fallis, M.D.

This is the seventh in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School. Dr. C. T. Ashworth, professor and chairman of the Department of Pathology, serves as guest editor for the series.

Participating in this conference are Dr. R. C. Morrison, resident in medicine, Dr. G. C. Curry, resident in radiology at Parkland Memorial Hospital. Drs. J. P. Sanford and D. W. Seldin are from the Department of Medicine, Dr. P. C. Peters, Department of Urology, and Dr. B. D. Fallis, Department of Pathology, at the medical school.

Case Presentation

DR. MORRISON: The patient was a 77-year-old white woman who was admitted to Woodlawn Hospital on May 2, 1964, with fever, chills, and a right pleural effusion.

Prior to February, 1964, the patient was in fair health and was being treated by her local osteopath for high blood pressure of several years' duration. In mid-February, however, the patient developed fever and a cough. Antibiotics were prescribed for approximately two weeks, but she did not improve. The patient was then hospitalized at a local hospital where a large pleural effusion on the left and a small right-sided effusion were found. She was treated with several antibiotics, and a drainage tube was placed in the left chest for one week. The drainage fluid was serous in type, negative on routine culture, and cytologic study for tumor cells was negative. Specific gravity was reported as "high," but the protein content was not determined. Chloramphenicol caused the fever to disappear and was continued for three or four weeks. Two blood transfusions were given "for a hemoglobin of 9.0 g.%." The patient was digitalized with digitoxin. The pleural effusions appeared to decrease. An indwelling Foley catheter was inserted into the bladder because of incontinence, but this was discontinued when the patient was discharged to a nursing home.

She remained in the nursing home for approximately three weeks, where digitalis plus diuretics were continued. Two days before admission to Woodlawn Hospital the patient began to have chills and fever to 101 F., and she seemed disoriented. She complained of frequency and burning on urination and of "foul-smelling" urine. She was started on chloramphenicol again, and the following day she was sent to the Parkland Emergency Room for observation. Chest x-ray at that time revealed a right pleural effusion, so she was admitted to Woodlawn Hospital. There was no recent history of cough, hemoptysis, chest pain, night sweats, or back pain. Further past history revealed that the patient had "stomach ulcer trouble" in the 1930's. There was no history of bleeding or abdominal pain, and she was not currently taking antacids. She denied having renal stones, but had had transient hematuria for one day two years previously. There was no previous history of surgery or fractures.

Physical examination revealed an elderly white woman with a flushed skin. Blood pressure was 160/80, respirations 20, pulse 88, and temperature 99 F. The pupils were regular and reacted to light and accommodation. Fundi revealed arteriolar narrowing with arteriovenous nicking. The neck was supple and no lymphadenopathy were found. Neck veins were slightly distended at 90°. The chest was kyphotic, and there was no bone tenderness. Dullness to percussion at the right base with moist rales were found, and breath sounds were clear in other areas. The left border of cardiac dullness was determined at the midclavicular line. The heart rhythm was regular and there were no murmurs. The abdomen was distended, and there was mild left lower quadrant tenderness. Bowel sounds were normal. No hepatomegaly, splenomegaly, or costovertebral angle (CVA) tenderness were detected. Pelvic and rectal examinations were negative. A trace of edema was found in the lower portions of both legs. No neurologic abnormalities were elicited.

Presented is a case of a woman with congestive heart failure and edema, who developed uremia and had radiologic and clinical signs indicative of urinary tract infection.

After admission the patient had a frank shaking chill. Temperature rose to 101.6 F. and blood pressure was 210/90. Blood cultures were obtained, and the patient was continued on chloramphenicol. There was no cough nor sputum production. The next day her temperature spiked to 104 F. and blood pressure fell to 95/60, but two hours later it rose spontaneously to 140/80. The urine output fell to 70 cc. over a 10-hour period, and the blood urea nitrogen (BUN) rose from 64 to 86 mg. per 100 cc. Urinalysis revealed pyuria with many gram-negative rods, and a pH of 7.5. The abdomen became more distended and tenderness increased. A KUB film of the abdomen revealed a calcific density in the right upper quadrant and gaseous distention in the large and small bowel. She was begun on Coly-Mycin and kanamycin. On May 4 she was transferred to Parkland Hospital.

At the time of transfer the blood pressure was 188/90, respirations 36, pulse 110, and temperature 101 F. Sclerae appeared icteric, her neck veins were distended at 90 F., and right CVA tenderness was noted. A grade two ejection murmur was present at the apex without radiation. Two-plus sacral edema was noted. Neurologic examination revealed cogwheel rigidity of the left arm, a questionable left facial weakness, and negative Babinskis. The patient spontaneously moved all extremities. Proctoscopic examination was negative. Four quadrant taps of the abdomen by a surgical consultant were negative. Lumbar puncture revealed mildly xanthochromic fluid with no cells. Urine culture yielded growth of a *Proteus* organism which was sensitive only to kanamycin. The patient was seen by a urologic consultant on May 5, and retrograde pyelography was done. A catheter was passed beyond a large right ureteral stone and into the right renal pelvis, where gross pus was obtained. Urine from the left kidney was clear. The ureteral catheter on the right was left in place. Urine output the following day was only 250 cc. and blood cultures were reported as positive for *Proteus mirabilis*. Penicillin in high doses was added to the therapy.

On May 7, the BUN had risen to 141 mg. and peritoneal dialysis was started. The patient appeared agitated and confused. Urine output from the left kidney was 625 cc., while from the right kidney it was 75 cc. On May 8 the patient had a sudden episode of hypotension with blood pressure of 50/28 which responded to vasopressors. For the next several days the blood pressure was maintained by vasopressors. The BUN was lowered to 58, but urine output remained low, with the right kidney putting out only 6 cc. and the left kidney 500 cc. On May 10 she began to have frequent grand mal seizures and these continued despite phenobarbital, oxygen, and Dilantin. Peritoneal dialysis was discontinued. The patient remained edematous. She was unresponsive except to deep pain. On May 13 the patient was having intermittent seizures. She had been intermittently hypotensive for the previous three days, requiring constant vasopressors. The right kidney produced no urine and the left kidney excreted 500 cc. per day. Her respirations were labored, blood pressure fell despite vasopressors, and she died.

Radiologic Findings

DR. CURRY: The first chest films are dated May 2, with follow-up on May 4. The significant findings on these two films are cardiomegaly, generalized pulmonary vascular congestion, and bilateral pleural effusion. The etiology of the effusions, at least from the radiographic standpoint, is compatible with pulmonary vascular congestion. The chest film of May 10 was made in the supine position and is difficult to compare with the initial upright film, but probably shows an increase in the pleural effusion, at least on the right side. There is density in the right base which can also be seen faintly on the KUB film of May 12. This may represent an area of pneumonia or a pulmonary infarct, but it

TABLE 1.—*Summary of Laboratory Findings.*

Urinalysis:

	May 2	May 7	May 7	
			Rt. Kidney	Lt. Kidney
pH	7.5	4.5		
specific gravity	1.011		1.011	1.012
albumin	+1		Trace	Trace
sugar	neg.		neg.	neg.
acetone	neg.		neg.	neg.
RBC (per HPF)	4-6		275	200
WBC (per HPF)	12-15		15-30	10-20

Casts Occasional hyaline and occasional granular
Bile and porphobilinogen negative.

Bacteriologic Studies:

Blood Culture: May 4, *Proteus mirabilis,* sensitive to kanamycin and chloronovo-biocin. Slightly sensitive to penicillin.

Urine Culture: May 5, Rt. kidney—*Proteus mirabilis,* 35,000/ml. Sensitive to penicillin. Left kidney—sterile.

Spinal Fluid Culture: May 5, Staphylococcus, coagulase-negative.

Stool Culture: Negative for pathogens.

Pleural Fluid: Negative smear for acid-fast organisms; negative routine cultures.

Blood Chemistries:

Total Proteins: May 6, albumin/globulin 2.5/3.1.

Serum Amylase: May 6, Less than 320 Somogyi units.

Urea Nitrogen: May 2, 64 mg. per 100 cc.; May 5, 86; May 7, 141; May 10, 58.

Pleural Fluid: Yellow; specific gravity 1.010; RBC 4,000 per cu. mm.; leukocytes 603 with 333 lymphocytes, 270 polymorphonuclear leukocytes; protein 3.0 gm. per 100 cc.

Lumbar Puncture: Xanthochromic fluid; opening pressure 24 cm. water; no cells; protein 16 mg. per 100 cc.; sugar 120 mg. per 100 cc. with blood sugar 170 mg. per 100 cc.

Stool: Guaiac negative.

Electrocardiogram: Low voltage; 1st degree A-V block; nonspecific ST-T changes.

Venous Pressure: 18 cm. water.

is probably a real finding since it is present on two different days. Our first film of the abdomen was May 4, and shows a generalized accumulation of bowel gas consistent with a nonmechanical type of ileus. The density in the right upper quadrant is a calculus in the renal pelvis. A retrograde pyelogram was done on May 5 and a second on May 12. The calculus, of course, is quite evident. The calculus has been moved by the catheters into the upper portion of the renal pelvis. On the left, no significant abnormalities were noted. The changes in the right kidney are very interesting and consist of a ragged appearance of the calyces associated with some dilatation of the calyceal systems. This could well represent, besides the known pyelonephritis, changes of renal papillary necrosis. The film of May 12 shows very similar findings, with an area in one upper pole calyx where there has actually been some sloughing of the renal papillae (Figs. 1 and 2).

In summary, radiological diagnoses are cardiomegaly with pulmonary vascular congestion and a left lower lobe density suggesting pulmonary infarction, a right renal calculus, and dilatation of all the right calyces and changes highly suggestive of renal papillary necrosis.

Clinical Discussion

DR. PETERS: The collecting system of the right kidney is not as ectatic as one would anticipate from the protocol presented. So, in view of the fact that the pyelogram looks as good as it does, while the patient was doing so poorly, I think we certainly have to suspect some severe disease in the kidney parenchyma in addition to that alluded to by the radiologist. I am thinking of bilateral disseminated foci of suppuration due to hematogenous infection or parenchymatous inflammation, and not merely a unilateral papillary or medullary necrosis.

The pyelographic appearance is that of a renal pelvic stone rather than a ureteral stone. I prefer to pass a ureteral catheter by an impacted stone in a patient actually ill with urinary tract infection to establish drainage, but if the catheter cannot be passed by the stone, or if the patient does not improve rapidly, I would then go in and remove the stone. A septic lesion can drain very well through a ureterotomy, while occasionally it will not drain well through a No. 4 or 5 ureteral catheter.

DR. SANFORD: In discussing this patient, I should like first to take up certain features to be considered in arriving at a diagnosis, and second, to make some general comments relating to the pathogenesis and management of the problem.

One of the early clinical features was the development of bilateral pleural effusions. Are the pleural effusions directly related to the later course? In considering the etiology of bilateral pleural effusions in patients over the age of 50, statistically the most common etiology is congestive heart failure.[7] This patient presented other features suggestive of congestive heart failure: cardiac enlarge-

ment, neck vein distention, and a trace of ankle edema. Another common etiology of pleural effusion is neoplasm. Neoplastic pleural effusions are seldom bilateral, and in at least one-third, malignant cells can be demonstrated by cytologic techniques. The pleural fluid protein content of 3 gm. per 100 cc. is in the range usually considered to indicate an exudate rather than a transudate. In one report dealing with the characteristics of pleural fluid, however, two of eight patients with pleural effusion due to congestive heart failure had pleural fluid protein contents in excess of 3 gm. per 100 cc.[2] Thus, I would attribute the pleural effusion to congestive heart failure, probably on an arteriosclerotic basis.

The congestive heart failure was indirectly related to her demise. The patient acquired the most common and serious nosocomial infection encountered, hospital-acquired urinary tract infection as a consequence of congestive heart failure. Specifically, an indwelling Foley catheter was inserted for an undescribed interval. It is reasonable to assume that the catheter was in place for at least 48 hours. If it had been allowed to remain for 48 to 72 hours, the chances are greater than 90 percent that the patient would have acquired bacteria within the bladder. It is conceivable that she had previous bacteriuria, either causing or secondary to the renal calculus. Subsequent to catheterization, she developed a frank shaking chill. This was associated with hypotension, and while hospitalized her temperature rose to 104 F. At the time of admission, there were certain laboratory findings which might prove helpful in making a clinical estimate of the bacteriologic diagnosis. Urinalysis revealed many gram-negative rods and a urine pH of 7.5. It is probable that urea-splitting organisms, that is, *Proteus* species, were present to produce this alkaline pH. This may be of significance, in that it is often necessary to treat a patient empirically on clinical grounds prior to the time that the laboratory can report the results of cultures and sensitivities.

When transferred to Parkland, she had a blood pressure of 188/90. At this time, her respirations were 36 per minute. The oc-

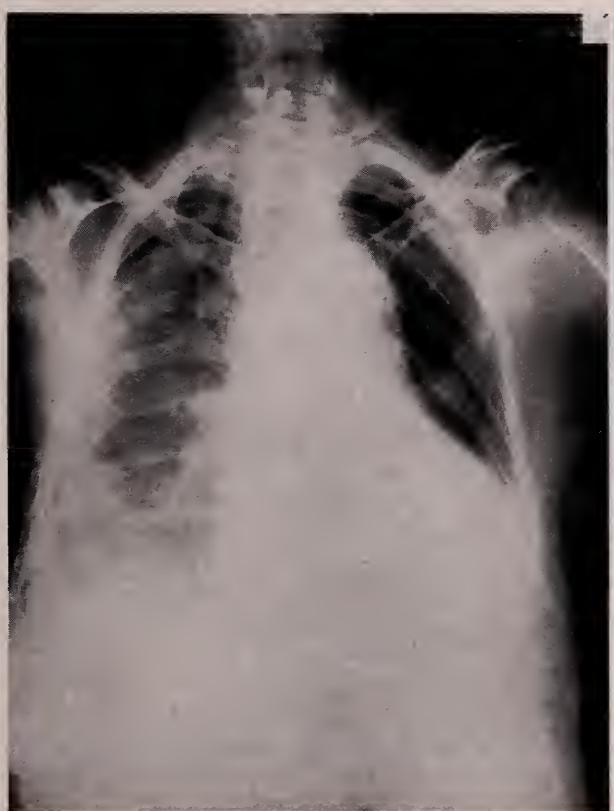


Fig. 1. Chest film (AP) of May 10 demonstrating right pleural effusion and very faint density in the right base.

Fig. 2. Retrograde pyelogram on May 5 demonstrating normal left collecting system. On the right there are generalized caliectasis and a ragged appearance of the calyces. The right renal calculus is obscured by the contrast material.

currence of hyperventilation has been reported as one of the earliest clinical manifestations of gram-negative rod bacteremia.

The patient was noted to be icteric and had right costovertebral angle tenderness. It should be emphasized that right costovertebral angle tenderness is not pathognomonic of renal infection. Patients with hepatic lesions, hepatitis, or liver abscesses may demonstrate right costovertebral angle tenderness.

She subsequently developed neurologic problems proceeding from cogwheel rigidity in her left arm progressively to grand mal seizures. These changes may have followed decrease in cerebral blood flow resulting from hypotensive episodes which compounded existing arteriosclerotic cerebrovascular disease.

At this point one might consider an esoteric diagnosis. The patient had renal disease, liver disease, central nervous system involvement and thrombocytopenia. The diagnosis of thrombotic thrombocytopenic purpura might well be considered. There is, however, one major feature against this diagnosis, namely, there was no evidence of hemolytic anemia. Therefore, I do not believe she had thrombotic thrombocytopenic purpura.

I would agree with the ward impression that the patient had urinary tract infection involving primarily the right kidney. This involvement may have been severe pyelonephritis or necrotizing renal papillitis, either of which might account for the anuria. Secondary to the urinary tract infection due to *Proteus mirabilis,* there occurred gram-negative rod bacteremia. I would explain the icterus by the antecedent episodes of hypotension.

A brief discussion of the pathogenesis and management of gram-negative rod bacteremia may be in order. This is not an uncommon occurrence, and is now becoming far more common throughout the country than staphylococcal bacteremia. It accounts for more deaths than does staphylococcal disease. Every week we encounter several patients with gram-negative rod bacteremia at Parkland Memorial Hospital. This patient presents most of the typical clinical features of this disease.[9] She had a source of infection, specifically the urinary tract, which is the most common source. The onset was abrupt with a frank shaking chill, followed by hypotension. Approximately 25 to 30 percent of patients with gram-negative bacteremia have hypotension. For a consideration of the clinical features, I refer you to the editorial in the *American Journal of Medicine* by Dr. Waisbren.[8] He discusses evidence which suggests that shock in gram-negative rod bacteremia is not the consequence of the endotoxemia, as it is commonly held to be.

There are six basic approaches to therapy in this patient. Dr. Peters has mentioned the renal calculus and the importance of draining confined pus. This is true in any infectious process. In addition to draining the site of infection, the other therapeutic approaches to be considered are antibiotics, steroids, vasopressor agents, plasma volume expanders, and adrenergic blocking agents.

We have already said that on clinical grounds one might have predicted the implication of a *Proteus* organism. Basically, there are two types of *Proteus* species: the so-called indole-negative and the indole-positive species. This differentiation is of clinical significance because the indole-negative *Proteus,* such as *Proteus mirabilis,* is more common and tends to be sensitive to kanamycin and chloramphenicol. In addition, these organisms are often sensitive in vitro to penicillin G. The amount of penicillin G required is fairly large, but some 85 percent of strains of *Proteus mirabilis* are sensitive to 20 units per ml.[1] How can this blood level be achieved? As a rough guide, for every 1 million units of intramuscular penicillin G, a blood level of 1 unit per ml. will be attained. Actually, on a dosage of 20 million units per day, blood levels are in the range of 50 units per ml. This patient received 35 million units of penicillin G the first day, then 15 million units a day. This should have been adequate to inhibit the organism. In addition, she received kanamycin, which is most likely the agent of choice. Colistin was initially employed because of the possibility of infection due to *Pseudomonas* species, and its use was very logical. The reported mortality rate in patients with *Proteus* bacteremia and shock is in excess of 90 percent, even when appropriate therapy has been administered.[9]

The question of adrenal steroids is usually raised. This patient did not receive steroids. There is no good evidence that steroids make any difference in the course of gram-negative rod bacteremia in man.[9] They may prevent the development of tachyphylaxis to

vasopressor agents. The use of steroids is based upon observations in experimental animals that pretreatment with steroids will afford protection. Once an animal has received the endotoxin, no protection is afforded. In general, we have used steroids in most patients with gram-negative rod bacteremia associated with shock; however, there is little evidence that they make any real difference. We have seen no indication that they have been detrimental. In the pediatric and obstetric literature there is a suggestion that steroids may predispose to the generalized Shwartzman reaction.[4] This has not been observed in nonpregnant adults.

The use of vasopressor agents such as metaraminol (Aramine) or levarterenol (Levophed) has been argued. I think most would agree that in the patient who has hypotension, to the degree that cerebral blood flow and renal blood flow is not maintained, vasopressors are indicated. Once large doses are required, or if the patient is maximally vasoconstricted as manifested by a pale cold skin with pilomotor response, the use of vasopressors is usually without effect. Again, we have generally used vasopressor agents in patients with gram-negative rod bacteremia with shock when there are accompanying disturbances in cerebration and diminution of urinary output.

Some investigators have suggested the use of plasma volume expanders such as plasma. These agents have a role only in patients who have a loss in plasma volume. Major decreases in plasma volume are not characteristic of endotoxin shock, at least in the experimental animal. The patient with peritonitis may have loss of fluid into the bowel or into the abdominal cavity, in which case plasma volume expanders are indicated to replace the volume.

Lastly, there is the question of the use of adrenergic blocking agents such as dibenzyline or chlorpromazine. Both of these agents have been shown to be effective in decreasing the mortality in experimental animals which are given endotoxin.[6] There has been, however, very little clinical experience with these agents. Nickerson and co-workers have used sympatholytic agents in hypotensive patients and reported an increased survival rate.[5] Administration of these agents is very difficult. If administered too rapidly, more severe shock than already present may ensue. At this time, I would suggest that the use of adrenergic blocking agents represents an experimental procedure, and unless one is prepared to carry out the appropriate control measurements, these therapeutic measures should not enter into the routine management of patients with gram-negative rod bacteremia.

Finally, I would like to close by emphasizing again the importance of this type of hospital-acquired infection. Martin and coworkers at Seton Hall[3] have pointed out that the use of a triple-lumen catheter and the irrigation of the bladder with some type of antibiotic or antibacterial solution, for example, 0.25 percent acetic acid or a combination of neomycin and polymyxin, has been effective in decreasing the frequency of postcatheterization bacteriuria and bacteremia. At the Jersey City Medical Center, when they adopted this as a routine procedure, the number of patients encountered with gram-negative rod bacteremia was significantly reduced from 33 to 5 patients per year. Extrapolation of these results to a nationwide estimate results in very striking conclusions. The American Hospital Association estimates there are 20,600,000 admissions per year to hospitals. Indwelling catheters are utilized in approximately 10 percent of all admissions. Accepting that the frequency of bacteremia can be decreased from 33 to 5, Martin and others estimate that there are between 8,000 and 31,000 preventable deaths which occur following catheterization. These numbers are comparable to the number of deaths annually due to tuberculosis and automobile accidents, respectively.

DR. SELDIN: This patient apparently had generalized edema early in the course. There are two causes for edema which are pertinent here. In the first place, the issue of congestive heart failure has been mentioned by Dr. Sanford. The protocol is somewhat skimpy and confusing in its description of the features associated with the edematous state. On physical examination the heart was of normal size, a finding which is not confirmed by x-ray. I think the evidence, taking everything together, is convincing that the patient must have been in congestive failure. The venous pressure was high; the heart was dilated; there was pulmonary congestion by x-ray. Secondly, it should be borne in mind that the patient had a moderately severe hypoalbuminemia. The serum albumin is recorded as 2.5 gm. percent; therefore, the chances are good

that, in addition to congestive failure, the edematous state was contributed to by hypoalbuminemia. The development of hypoalbuminemia of this degree probably means that she had been ill and starving for a long time.

Hypoalbuminemia alone can, of course, produce edema, pleural effusions, and ascites; however, it never produces pulmonary congestion, pericardial effusions, an elevated venous pressure, or cardiac dilatation. The facts, then, that the patient had pulmonary congestion by x-ray, that the heart was dilated, and that the venous pressure was high, indicate unequivocally that congestive failure must have been the principal cause of the edema, and that hypoalbuminemia was only a secondary contributory factor.

The second point I would like to make is that the patient had grand mal seizures at a time when she was lapsing into uremia. Convulsions are probably not a feature of uncomplicated uremic encephalopathy. Whenever a patient who is uremic convulses, some factor other than uremia should be suspected. The most common cause of convulsions in such patients is hypertension. The distinction between hypertensive encephalopathy and uremic encephalopathy is a very critical one. It was originally drawn by Oppenheimer and Fishberg who pointed out that hypertension per se can give rise to an encephalopathy characterized by convulsions, amaurosis, aphasia, headache, and hemiplegia. These findings are not usually encountered in uremia unaccompanied by hypertension. Consequently, the convulsive process in this patient probably could not have been due to renal failure alone. The patient did not have severe hypertension according to the evidence available; therefore, she must have convulsed for some other reason. She could have had, as others have suggested, profound cerebral ischemia caused by reduced cerebral blood flow. If there were a background of arteriosclerotic narrowing of the cerebral vessels, it is not unreasonable to suppose that she may have convulsed on this basis. Furthermore, it would not be surprising if cerebral ischemia had expressed itself in some degree of infarction.

The final point I want to make concerns the renal failure itself. This patient had a rather marked and progressive azotemia. Any degree of azotemia requires the presence of bilateral disturbance of renal function. The destruction of one kidney alone will not generate detectable azotemia if the contralateral kidney is normal and the circulation intact. In this patient, one kidney had virtually no renal function; the catheter drainage amounted to only 5 cc. a day. But this by itself would hardly account for the mounting azotemia that characterized the patient's course, and we have to say that the other kidney was not functioning normally. This could have been due to two circumstances. First, the circulation to the kidney may have been compromised. This might have resulted from gram-negative shock. Even though arterial blood pressure is normal, renal blood flow may be strikingly reduced when marked compensatory vasoconstriction is responsible for the maintenance of systemic blood pressure. It is possible, therefore, that the contralateral kidney was functioning poorly because its circulation was markedly reduced.

It is also possible, however, that the patient had bilateral renal disease. To be sure, on one side the renal disease was overwhelming, but on the other side, the side which was capable of elaborating 500 or 600 cc. of urine daily, parenchymal renal disease of more modest proportions might have been present. The fact that the magnitude of renal failure was so asymmetrical on the two sides is strong evidence against an evenly distributed diffuse bilateral intrinsic renal disease as the only pathologic process. I would, therefore, favor the hypothesis that the patient had a very severe pyelonephritis on one side which had progressed to the point of an acute medullary necrosis and was responsible for unilateral anuria. It is possible that there was some degree of chronic renal disease on the contralateral side, but the most attractive hypothesis, to me at any rate, is that she had an impaired cardiac output due to gram-negative shock, that this was responsible for impaired renal blood flow, and that some degree of acute tubular necrosis, mild to be sure, developed in both kidneys.

DR. PETERS: We have not seen the specimen yet, but I notice that one diagnosis mentioned in the protocol was pyonephrosis. To a urologist, pyonephrosis usually implies that there has been an infectious or inflammatory process of long standing with dilatation of the collecting system. The collecting system is often fixed at a given stage of

dilatation by scarring and fibrosis. This sort of collecting system usually becomes secondarily infected. Often there is an associated stone. A kidney that is acutely blocked by a stone in the ureter or at the ureteropelvic junction, and then becomes infected secondary to manipulation or to bacteremia, is usually classified by us as an infected hydronephrosis rather than a pyonephrosis.

One must not forget the occasional association of stone in the kidney with squamous cell neoplasm. In one study, 50 percent of the squamous cell neoplasms of the kidney were accompanied by large renal pelvic or staghorn calculi. Squamous cell neoplasm should at least be thought of when one observes a large, apparently silent (asymptomatic) stone in an infected kidney.

Pathologic Findings

DR. FALLIS: At the time of necropsy the body was that of a severely obese, aged, white female. There was slight subcutaneous edema. Our major attention in this case was directed to two systems, the urinary tract and the lungs. The right kidney weighed 140 gm., the left, 150 gm. The external surface of each kidney was finely granular due to a moderate degree of arteriolar nephrosclerosis, as might be anticipated in an elderly hypertensive patient. In addition, there were broad, irregular, depressed scars indicating chronic pyelonephritis. These scars were bilateral, although more numerous on the right, but were not extensive, as you might conclude from the fact that the kidneys were of almost normal total weight. In the pelvis of the right kidney was a large, ovoid, black calculus (Fig. 3). The black surface of the stone indicates that its cortical layers, at least, are predominantly calcium oxalate. Calcium oxalate itself is white, but these stones are usually quite hard with a rough, irregular surface. They abrade the mucosa of the urinary tract leading to minor hemorrhages, and the blood pigments then stain the stone, so that a urolith with an oxalate cortical surface is usually black or brown. The renal pelvis on the right was very little, if any, dilated, and the pyramids minimally blunted; we did not make the diagnosis of hydronephrosis on either side. The right renal pelvis was hyperemic, thick, and opaque, indicating the presence of both an acute and chronic pyelitis. In one of the lower pyramids there was papillary necrosis. This, however, involved only the extreme tip of one renal papilla and was probably not a major factor in the patient's death. The right ureter was dilated and was, in addition, acutely and chronically inflamed. The right ureter had probably been obstructed in the past, but there was no stone in it at the time of necropsy. Probably the ureter had been obstructed during passage of either another renal stone or a fragment of the stone found in the renal pelvis. The history reveals the interesting fact that the patient suffered an episode associated with hematuria several years prior to death, and it is very likely that a renal stone was passed at that time, probably lodging temporarily in the lower segment of the ureter and causing hydro-ureter associated with chronic infection. Probably chronic ureteritis caused enough scarring to provoke permanent hydro-ureter. The urinary bladder was hyperemic, and the patient had, as Dr. Sanford anticipated, infection in the lower urinary tract as well as in the upper tract. The acute and chronic urethritis and cystitis were probably caused, as discussed by Dr. Sanford, by the indwelling catheter.

Fig. 4 shows the single renal pyramid involved by papillary necrosis. The tip of the papilla was smudgy and necrotic. This was a very minimal change and involved no more than a millimeter at the tip of the renal papilla. It is very doubtful that such a lesion could be detected radiographically.

Fig. 5 shows the substance of the right kidney which was severely and diffusely involved by acute pyelonephritis. Dilated renal tubules were filled with purulent exudate, and there was an extensive interstitial infiltrate of inflammatory cells, chiefly neutrophilic leukocytes. There were small foci of chronic pyelonephritis in both kidneys, but the acute pyelonephritis was restricted to the right kidney. The papillary necrosis was caused by a combination of obstructive uropathy due to the renal stone and severe acute pyelonephritis.

The acute uremia, as Dr. Seldin predicted, was due not to the pyelonephritis (although this was certainly a contributory factor), but to the concomitant presence of an acute tubular necrosis or lower nephron nephrosis (Fig. 6). The tubular lesion was doubtless caused by shock, which the patient had had on several different occasions. In many foci the tubular epithelial cells were necrotic and had partly desquamated to form a granular

debris in the center of the tubular lumen. The tubular necrosis was focal or patchy, involving small segments of individual tubules, in various parts of both kidneys as is characteristic of lower nephron nephrosis. The acute tubular necrosis was the cause of uremia.

The spleen, weighing 200 gm., was slightly enlarged for a woman of this age, due to a subacute splenic hyperplasia in response to renal infection over the last several weeks of her life.

Summarizing the lesions in the urinary tract, the patient had a right renal stone and had evidently passed other stone material through the right ureter leading to some chronic ureteritis and hydro-ureter on the right. She also had focal bilateral chronic pyelonephritis. Her terminal course was precipitated by the indwelling catheter and was characterized by urethrocystitis and ascending acute pyelonephritis in the right kidney complicated by bacteremic shock. This in turn produced lower nephron nephrosis, which in combination with the severe unilateral pyelonephritis caused uremia.

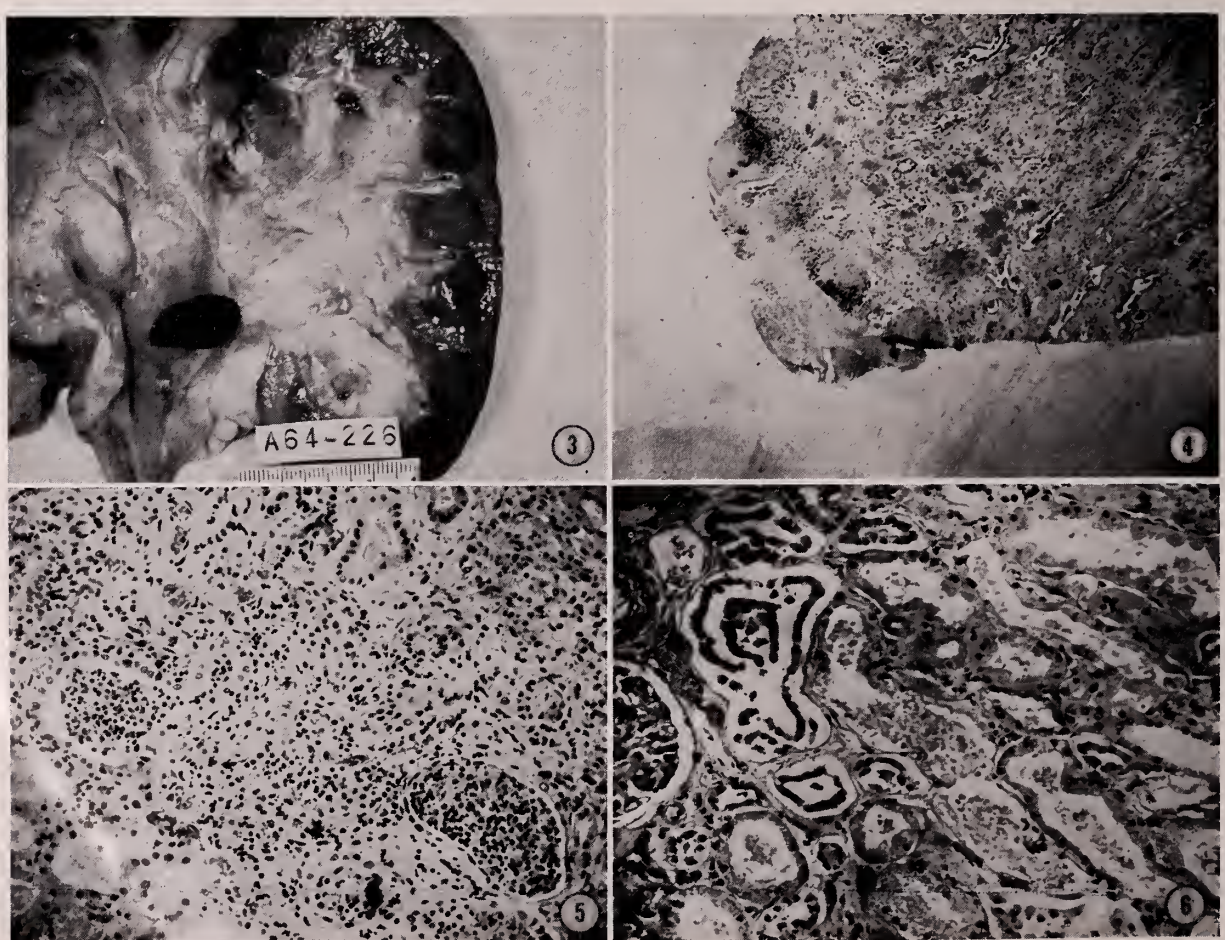
The immediate cause of death was in the lungs. There were bilateral pleural effusions (300 cc. left, 400 cc. right). The lungs together weighed 900 gm. There were several thrombotic emboli in both lower lobes, and there were two recent infarcts in the lower lobe of the right lung; one of these infarcts was secondarily infected with early suppuration. A large thrombotic embolus completely filled the right main-stem pulmonary artery. Microscopic examination of the lungs revealed multiple small thrombotic emboli of varying ages. These emboli were present in

Fig. 3. Right kidney. Note the stone in the renal pelvis.

Fig. 4. Renal papilla, right kidney (very low magnification). The tip of the papilla is smudgy and necrotic (renal papillary necrosis).

Fig. 5. Right kidney (low magnification). Dilated tubules contain neutrophilic leukocytes.

Fig. 6. Left kidney (high magnification). Note the tubule in the center; its epithelial cells are necrotic and have been shed into the tubular lumen as granular debris.



both lungs, and many were organizing and had been present for several weeks. The recurrent pleural effusions in this case were probably caused by recurrent pulmonary emboli and infarcts. Probably the patient had been in some degree of congestive heart failure. She had an enlarged heart, which weighed 400 gm., and was said to have been hypertensive. Presumably hypertension was the cause of her heart failure. There were no marked anatomic changes of heart failure at the time of necropsy, and her other diseases had obviously succeeded heart failure as her major problem. Probably she had been in some degree of heart failure, and the pulmonary emboli were the result of deep vein thrombi in the legs, which were engendered by the venous stasis of heart failure and perpetuated by ill health accompanying her urinary tract infection.

The diagnostic problem of pulmonary embolism is well exhibited in this case. A large pulmonary embolus in a reasonably healthy person will usually produce fairly distinctive signs and symptoms. Unfortunately, the initial pulmonary emboli are usually small, even miliary in size, and they usually occur in a patient clinically ill from other diseases, so that the initial embolism is usually submerged in the overall clinical picture and gives very nonspecific symptoms and signs, to which no definitive diagnosis can be fixed. Such was apparently the case here, although in retrospect the radiologist tells us that perhaps he can demonstrate the right lower lobe infarcts.

Anatomic changes in the other viscera were nonspecific. There was a large acute gastric ulcer which had bled slightly just before death. There was a healed childhood type pulmonary tuberculous lesion involving the upper lobe of the right lung and one of the hilar lymph nodes to produce a typical calcified Ranke complex. Such calcified lesions are relatively common in older adults and represent a residuum of tuberculous infection in early life. In addition, this patient had an active, although not severe, tuberculous pericarditis. The epicardium and pericardium are adherent by rather dense fibrous adhesions, and microscopic study revealed fibrosis and several hard tubercles. No acid-fast organisms could be stained in the sections, and the infection was relatively inert although obviously not completely inactive. The pericarditis presumably resulted from lymphatic spread of organisms from some of the involved hilar lymph nodes at some time in the past. There was no evidence of pericardial constriction or tamponade.

There was severe generalized atherosclerosis and the major cerebral arteries were significantly narrowed. Sections of the brain revealed no anatomic lesion within the brain, and the neurologic symptoms were probably the result of a combination of hypotension and cerebral arterial narrowing; the resultant cerebral vascular insufficiency was sufficient, presumably, to induce the various neurologic symptoms that appeared during her terminal course. The changes in the remaining viscera did not contribute to her death.

In summary, the patient was probably in congestive heart failure on a hypertensive basis, had recurrent pulmonary emboli, and also had a severe urinary tract infection which provoked gram-negative bacteremic shock, lower nephron nephrosis, and azotemia; the immediate terminal event was the impaction in the right pulmonary artery of a large pulmonary embolus.

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NEWS

New Sections, College-Medical School Articulation, 1965 Legislation Discussed by Executive Board

PROPOSALS to establish scientific sections in urology and nervous and mental diseases during the Texas Medical Association Annual Session; conferences to improve communications between medical school faculties, college premedical advisers, and practicing physicians; and legislation to be considered by the Texas Legislature during 1965 were among items discussed by the Executive Board at its fall meeting on Sept. 20 in Austin.

The Committee on Cardiovascular Diseases and the Council on Scientific Advancement also asked for consideration of a section on cardiovascular diseases, which is to be reviewed by the Council on Annual Session.

Registration Fee Set for Nonmembers

The Executive Board adopted the recommendation of the Board of Trustees that a $10 registration fee be charged nonmembers for attendance at the Annual Session. This was based on a belief by the Trustees that Association physicians should be entitled to benefits which are not made available to nonmembers. Interns, residents, and nonmember physicians participating in the Annual Session will be exempt.

Trustees also received approval of their recommendation that a parking lot for 27 additional cars be constructed behind the existing parking lot behind the Headquarters Building. This is expected to relieve at least part of the parking congestion during meetings at the building.

Approved also by the Executive Board was a decision by the Board of Councilors that a physician may be a member of the county medical society which has jurisdiction over his place of legal residence when he does not devote 60 percent of his time in practice in any one location.

Careful Review Asked Of AMA Speakers, Articles

A resolution approved by the Board for presentation to the American Medical Association was one submitted by the Texas Delegates to the AMA and Tarrant County Medical Society calling for participants in AMA-sponsored programs to be selected with great care to avoid any impression that the American Medical Association is furnishing a rostrum for any dissident individual or group. It was suggested in the resolution that the Executive Committee of the Board of Trustees be available on short notice at any time for consultation about the desirability of any proposed program participants. It was further asked that the editorial staff of every AMA publication be expected to consult with the Executive Vice-President before publishing any article or launching any program that might be expected reasonably to result in controversy.

Another resolution submitted by Dr. James H. Sammons asked that the Executive Board instruct the Executive Secretary to forward immediately following its meeting a statement of grave concern to the AMA Judicial Council over certain national publications lauding members of medical organizations, not cleared by the particular medical organizations.

In other action, the Executive Board granted the Texas Delegates to the AMA the authorization to invite the AMA to hold its 1967 Clinical Meeting in Houston.

Dividend To Be Distributed

Approved at the recommendation of the Council on Medical Service and Insurance was the acceptance of an offer by Lumbermens Mutual Casualty Company to give a $194,595 dividend on the disability insurance program for members insured during the policy year Nov. 1, 1963, to Oct. 31, 1964, and a $10,050 dividend on the major medical program for members insured during the policy year April 1, 1963, to March 31, 1964. It was agreed that the dividends should be distributed pro rata to members insured in these groups, respectively.

Report of House Action Reviewed

The Board took notice of action passed by the House of Delegates during the 1964 Annual Session which pertained to a correction requested by the Committee on Mental Health and the Council on Scientific Advancement. The item was in reference to a recommendation of passage of a privileged communications bill in Texas. The Board voted to request that the *Texas State Journal of Medicine* carry a story noting that the House

of Delegates at its 1964 Annual Session inadvertently passed a recommendation approving such passage because of a report published in the "Handbook for Delegates" by the Council on Scientific Advancement and a recommendation of the Reference Committee on Scientific Work.

Mention of the item was included originally in the 1964 Handbook report of the Council on Scientific Advancement (page 54) which had indicated that it accepted "for information only the Committee's recommendation that a privileged communication bill be passed in Texas." The Reference Committee on Scientific Work then submitted to the House of Delegates the statement that it approved "the recommendation of the Committee on Mental Health which recommends that a privileged communications bill be passed in Texas." Report of the House action was later summarized in the *Journal*, in the *TMA Daily Bulletin*, and carried in full in the Transactions of the 1964 Annual Session.

Oral Cancer Pamphlet To Go Out

Distribution to members of the Texas Dental Association and the Texas Medical Association of a proposed pamphlet entitled, "Instructions for Collection of Cytologic Smears for Detection of Oral Cancer" was approved by the Executive Board. The American Cancer Society will bear the expense of the distribution.

New Scientific Sections Considered

In the action proposing new scientific sections, the Executive Board took cognizance of the Council on Annual Session's desire for a scientific section on urology. The Texas Urological Association has been asked to sponsor or assist in formation of such a section. The Executive Board referred to the Council on Constitution and By-Laws the Council on Annual Session's recommendation for a section on nervous and mental diseases. This is to be prepared and presented to the House of Delegates for action at the 1965 Annual Session.

Premedical Counselors Conferences Approved

The Committee on Liaison with Medical Schools was designated to arrange a conference with medical school faculties, college premedical advisers and practicing physicians to establish better communications between the groups. The conference had been the Committee's suggestion and was approved by the Council on Medical Education and Hospitals. Also recommended were conferences at the county level with high school counselors and premedical advisers, however, it was suggested that these not be implemented until the medical school and college premedical advisers establish the patterns they want to follow in premedical education. The Committee on Medical Careers would be responsible for those conferences.

Dues Statements To Be Prepared

The Executive Board agreed that the Texas Medical Association should make available to county medical societies a dues statement which would include a place for a voluntary contribution in the amount of $25 for AMA-ERF.

Kerr-Mills PR Program Urged for County Societies

The Board concurred with the Council on Medical Jurisprudence that the Texas Medical Association assume the responsibility for organizing a positive public relations program to be executed at the local medical society level in support of the extension of the Kerr-Mills benefits to the aged who need assistance with catastrophic illnesses and who are not covered under the existing first phase of Kerr-Mills.

Reemphasized in the Council's report was the TMA endorsement of Texas Constitutional Amendment Number Three which will be submitted to Texas voters in November and which will allow the Legislature to act on the second phase (Medical Assistance for the Aged) for medical care for the elderly who are not now covered by Old Age Assistance.

The Texas Department of Public Welfare was congratulated for its administration of the first phase of the Kerr-Mills program in Texas.

Kilgore Commended

The Executive Board expressed its appreciation to Congressman Joe Kilgore, who is retiring from Congress at the end of this session, for his outstanding service rendered while a member. Regret was expressed that he was retiring and the Board extended its best wishes to him.

Record Crowd Attends PR Conference

SIX HUNDRED fifty-eight, a record number of members, their wives, and guests, attended the tenth annual Public Relations Conference in Austin Sept. 19.

They took part in council and committee meetings, heard panel discussions, and listened to an all-day array of outstanding speeches. One hundred thirty-eight physicians attended the Association's Orientation Program, held simultaneously with the Conference. The day ended with a hospitality hour at the Driskill Hotel and the Texas-Tulane football game at Memorial Stadium.

One of the high points of the day was the presentation of plaques to Drs. M. O. Rouse of Dallas and George R. Herrmann of Galveston naming them 1964 recipients of the Texas Medical Association Distinguished Service Award.

Speakers included Dr. Max E. Johnson, San Antonio, President of Texas Medical Association; Guy D. Newman, Th.D., Brownwood, president of Howard Payne College; Dr. Alfred M. Popma, Boise, Idaho, chairman of the AMA Voluntary Health Agencies; Dr. Morris Fishbein, Chicago, editor of *Medical World News;* Dr. Donald E. Wood, Indianapolis, chairman of the board of directors of the American Medical Political Action Committee; and Dr. Walter H.

Judd, Minneapolis, statesman and former congressman.

The morning panel discussion was entitled "Working Together for Better Community Health Care." Presiding was Dr. Robert Mayo Tenery of Waxahachie. Participants were Crawford A. McMurray, D.D.S., Ennis, secretary-treasurer of the Texas Dental Association; C. B. Francis, Austin, president of the Texas Nursing Home Association; Mrs. Lillian E. Taubert, R.N., San Antonio, executive director of the Texas Nurses Association; W. R. Whitten, Fort Worth, director, Texas Pharmaceutical Association, and George B. Pearson, Tyler, past president of the Texas Hospital Association.

During the afternoon a panel on "How To Preach What You Practice" was held, with introductions by Dr. Glenn D. Carlson of Dallas. Panelists were James R. Hickox, Chicago, director of the AMA Program Services Department, Communications Division; Dr. Sam A. Nixon, Jr., Floresville, chairman of the public relations committee of Gonzales County Medical Society; Dr. C. Mac Geyer, Wichita Falls, chairman of the public relations committee of Wichita County Medical Society; and Dr. B. B. Markette, San Antonio, chairman of the public relations committee of Bexar County Medical Society.

Those attending the Orientation Program heard addresses by Dr. Byron P. York, Houston, chairman of the TMA Board of Trustees; C. Lincoln Williston, Austin, Executive Secretary; Dr. A. Rex Kirkley, Dallas, chairman of the Committee on Health Insurance; Dr. James H. Sammons, Highlands, secretary to the Board of Councilors; Dr. C. E. Fuste, Jr., Alvin, vice chairman of the Board of Councilors; Donald M. Anderson, Austin, Assistant Executive Secretary; Dr. Walter Walthall, San Antonio, chairman of the Board of Councilors; Dr. Milton V. Davis, Dallas, chairman of the Committee on Military and Veterans Affairs; and C. Dean Davis, LL.B., Austin, Assistant General Counsel to TMA.

Hostesses for the Conferences were members of the Woman's Auxiliary to the Travis County Medical Society. They were assisted by members of the Future Nurses Club of Austin.

The hospitality hour and bus transportation to the football game were arranged by the Charles O. Finley Co.

Dr. Johnson

Dr. Max Johnson, President of Texas Medical Association, called upon doctors to present a united front and to be not only good doctors, but good men and good citizens.

He outlined several ways in which doctors could present a strong united front: by working to strengthen the county medical societies by increasing attendance and by participation in their activities and those of the state and national associations, by conducting good public relations programs and enlisting the aid of the allied professions, patients, and friends, by using the press to tell their story through regular articles of public interest and paid advertisements stating the county society's position on important issues, by using radio and television, by establishing speaker's bureaus with speakers who are well informed on the issues, and by establishing liaison committees to work with allied professions.

He called on physicians to act now and at the election polls in November to uphold their "great common responsibility this year to the medical profession, to our patients, and to the private enterprise system."

Dr. Newman

Citing man's knowledge of how to unleash nuclear power, Guy D. Newman, Th.D., president of Howard Payne College at Brownwood, noted that in this, history has not repeated itself.

Speaking on "The World Drift and our Western Alternative," Dr. Newman noted that in face of the struggle for survival, man is debating whether he has chosen the right road to freedom and human dignity.

There is lack of understanding of the nature of our world drift, he said, and the intellectuals have become enamored of the concepts of materialism, including socialism.

He noted some of the great advances which have occurred in the United States—the greater longevity of life and the greater production of material goods, and noted that "socialism is failing every place on earth and it will fail here if we try it."

Pointing out that socialism offers no stimulus, he said that when everyone owns everything, no one takes care of anything.

He called on his audience to stop blaming Washington for the type of leadership we get and start blaming ourselves. "We get the leadership we deserve," he said.

Dr. Popma

Because the American people have become acutely conscious of many of the problems of health and disease and are becoming increasingly aware of the many methods used by scientists to prevent and combat disease and to further public health, many voluntary health organizations have been developed, reported Dr. Alfred M. Popma, chairman of the AMA Council on Voluntary Health Agencies.

The AMA, he said, is actively interested in and concerned with the free growth of these organizations, many of which are composed primarily of lay people who must look to doctors for guidance.

"It therefore becomes of prime importance for the doctor to have an understanding of his responsibilities in working with groups of lay people, to formulate medical policy along lines of established science and proper medical ethics," he said.

The individual physician who holds membership in his medical society, plays four separate but related roles when he becomes affiliated with a voluntary health agency, he added: he may be delegated to speak for his society, he may be called on by an agency to serve as a physician in ways suggested by his specific professional interest, he may seek membership on his own accord and become active in an organization's program because of his own interests, and he may use a health agency as a medium for public service as a private citizen in his community, and regardless of his profession.

A physician identified with such a group, he cautioned, should identify himself with all of the activities of the organization. He should know the needs of the group, its faults and weaknesses, as well as its virtues, and he will then be in a better position to give advice and counsel.

"As a physician who is cognizant of the steady encroachment of state and federal government upon the private practice of medicine, he will be in an excellent position to offer guidance to any new or old program which in any way affects the practice of medicine."

He cited public education in re-

Dr. Milford O. Rouse of Dallas (left) and Dr. George R. Herrmann of Galveston (right) share honors as the recipients of the first Distinguished Service Awards given by the Texas Medical Association for meritorious achievement in medical science, public service, and service to the medical profession. In a special ceremony Sept. 19 in Austin, the plaques were presented by Dr. Walter Walthall, San Antonio, chairman of the Board of Councilors, and Dr. Tom M. Oliver, Waco, Councilor for the Twelfth District. In acceptance, Dr. Rouse said, "The appreciation and love of you fellow Texans means more to me than any other honors." He expressed thanks to his fellow doctors, his partners, and to Mrs. Rouse. Dr. Herrmann said, "I accept the award with gratitude, and with full knowledge that I owe it to many others—my parents, teachers, preceptors, colleagues, and students, as well as my wife and family."

gard to problems of diseases, their diagnosis, and possibly prevention and treatment as one of the important programs of voluntary groups. All public information should be checked for scientific accuracy by competent physicians, he reported as the belief of the Council he represents, although much material can be prepared and written very well by qualified lay people. He cited qualified physician speakers before lay audiences as an important phase of county medical society efforts. Here, he cautioned the physician speaker to be sure to use language which his audience would understand. A happy medium in expressing hopes for cure of diseases is also important, he said.

"Nothing is more disconcerting than to have a doctor appear before a lay audience and present false hopes of cure for some incurable disease. Likewise, it is also unwise to be too helpful."

Well person examinations should also be encouraged at such meetings, he said.

Dr. Fishbein

Describing the modern charlatan as a person of striking appearance and a list of degrees which were not conferred by a recognized group, Dr. Morris Fishbein illustrated his talk on "Quackery" with brief case histories of some quacks and charlatans whom he has known.

He cited Paracelsus as the first great quack and at the same time a great scientist—a mystery, he said. He pointed to a modern parallel of a physician who has been in the news in recent years.

History repeats itself, he said, and men do not learn.

Victims of charlatans are not always the most ignorant people, he observed; many times they are members of the intelligentsia in a community. People want to believe in miracles at times of stress.

A number of medical cults have been absorbed into medicine, he noted, and there were more that should be absorbed into the scientific way of thinking.

Frequently in the past, great discoveries have provided impetus for quacks. Claims have been made that they could cure many diseases. He pointed to such claims surrounding the discovery of uranium.

In concluding his talk, he cau-

Panelists who discussed "How To Preach What You Practice" are (standing left to right) Dr. C. Mac Geyer, Wichito Falls; Dr. B. B. Morkette, San Antonio; (seated center) James R. Hickox, Chicago; and (seated right) Dr. Sam A. Nixon, Jr., Floresville. Dr. Glenn D. Carlson (seated at left) introduced the group.

tioned his listeners always to be skeptical and to inquire.

Dr. Wood

"You *Are* In Politics, Doctor!" This point was made clear throughout the address of Donald E. Wood, M.D., Indianapolis, Ind., chairman of the board of directors of the American Medical Political Action Committee.

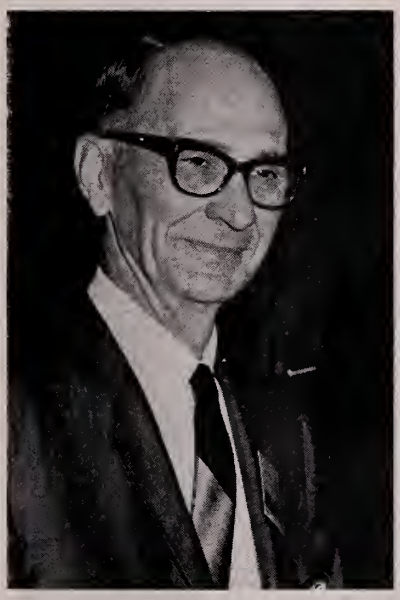
Citing the fact that whenever politicians consider legislation which affects the health of citizens, the doctor is automatically "dealt in" whether he asks for a hand or not. Dr. Wood explained that this realization led to the formation of the political action movement in medicine. The AMPAC and state PACs are bipartisan; a candidate's party label isn't half so important as his personal beliefs.

"This year," said Wood, "the country will elect a President, a Vice President, 435 members of the House, 34 out of 100 Senators, and 26 out of 50 governors." Now, about 48 percent of the members of the House vote with the opinions expressed by the AFL-CIO political leaders, and about 52 percent lean toward conservatism in their voting. "Suppose this year's elections bring about that shift of a little more than 1 percent?" Wood asked. In answer, he said that in the 87th Congress there were 24 key issues voted upon. A third of these issues would have gone the other way by a swing of 3 to 10 votes, and over half would have been changed by a swing of from 3 to 15 votes.

"How did we get into this situation of hanging on by our fingernails?" Wood traced the rise of the labor movement and its influence in politics from about 1935 to 1947, the year that the Taft-Hartley Law barred the unions from making contributions to political campaigns. The problem was solved quite simply: The unions created, as separate entities from themselves, political action groups composed of union members who "voluntarily" contributed money to support candidates.

Such an organization is COPE; it provides "money that makes the mare go" during a campaign, and manpower for the work that must be done. COPE works as a political education group—disseminating voting records, discussing issues, and so forth. Under this aegis it conforms with the law which forbids the use of union

Dr. Morris Fishbein, Chicago, whose witty remarks seasoned his talk on "Quackery," provides Dr. Howard O. Smith of Marlin with a hearty laugh.



Guy D. Newman, Th.D., president of Howard Payne College, Brownwood, told physicians, "When everyone owns everything, no one takes core of anything."

Dr. Donald E. Wood of Indionopolis got into the matter of politics ond how it relates to the medical profession. He is board of directors chairman of AMPAC.

The first and last speakers of the day get together for a few words. Dr. Max E. Johnson, left, San Antonio, first speaker, entitled his talk "United We Stand." Dr. Walter H. Judd, Minneapolis, statesman and former Congressman, spoke late in the afternoon on "The Doctor's Place in Public Affairs."

Down from the far northwestern state of Idaho, Dr. Alfred M. Popma, left, who spoke on voluntary health agencies, hears about Texas medicine from Dr. Harvey Renger of Hallettsville.

dues for the support of candidates, and corporate monies contributed for similar purposes. In short, labor has created a powerful political force, Dr. Wood said.

Physicians, because they are politically inexperienced, sometimes are outraged when asked to give money for support of political candidates, particularly at the national level. They overlook the fact that at the national level, legislation would affect medical practice in all states. Also, the speaker said, if physicians expect legislators to help with medical legislation, they must cooperate by giving direct candidate support.

AMPAC is legally constituted to perform this function if money is provided by voluntary contributions. Its corporate dollars, provided by the AMA, are used for overhead and for every other purpose the law allows, but its effectiveness depends on having contributed funds available for candidate support. The autonomous state PACs do these things: (1) Collect and assess political information and pass it to AMPAC. (2) Operate on the theory that only physicians can effectively raise money from other physicians. (3) Provide the bulk of candidate support funds raised by the profession, with help from AMPAC only when necessary. (4) Strive for individual efforts as well as money to support their candidate and his party. (5) Support candidates on the basis of their principles, not their party labels. However, once committed to a candidate's cause the PACs are completely partisan in his behalf.

Outlining the effectiveness of medical PACs, Dr. Wood said, "In 1962, our candidates won in 70 percent of cases, compared with COPE's 59 percent." Now, AMPAC is concerned with the forthcoming elections. "We solicit your help and your wholehearted support for the medical profession's political action movement. It is, we think, our best—and perhaps our only—hope for the future."

Dr. Judd

Scientific discipline and experience with human beings as they really are has given physicians cool heads, warm hearts, and the courage to make decisions and carry them through, and these are qualities the nation needs in its leaders today.

Speaking on "The Doctor's Place

Moderator for the panel that discussed "Working Together for Better Community Health Care" was Dr. Robert Mayo Tenery, Waxahachie (upper left). Members of the panel are Crawford A. McMurray, D.D.S., Ennis (upper center); George B. Pearson, Tyler (upper right); C. B. Francis, Austin (lower left); Mrs. Lillian E. Taubert, R.N., San Antonio; and W. R. Whitten, Fort Worth.

in Public Affairs," Dr. Walter H. Judd, Minneapolis, former Congressman, reminded his listeners that the profession has constantly evolved to meet the needs of the times. Now, he said, the doctor must be more active in public affairs to ensure his professional future, to improve environmental conditions that affect his patients adversely, and to ensure his future as a human being.

"We talk much about what effect our government's actions may have on us. The time has come when we must give more thought to what effect we can and must have on our government," Dr. Judd declared.

Most doctors oppose socialized medicine, but we have failed to show the public that we oppose this form of medical practice not because it would hurt us, but because it would hurt the public, Judd said. After all, the superior doctor will get ahead under any system; the average M.D. would be as well off financially under socialized medicine as under free competition, and the inferior physician would fare better.

Hitting hard at the profession's failure to tell the public what is at stake, Judd commented that most people still think they can get more and better medical care at lower cost under socialism than under free enterprise. The difference in viewpoint is that doctors want higher quality of care, but laymen take high quality for granted and are more concerned with more equitable distribution. "It is not enough for us to be right," he said. "We must tell and sell our reasons to the public far better than we have."

Judd compared Communism with a malignancy, pointing out that it cannot be malignant toward one area and benign toward another—it is either malignant or benign. As in performing an autopsy, in government there is a need to study the mistakes of the past, admit them openly, and learn from them. In dealing with a cancer, doctors have learned that there can be no end to the struggle until it or the body prevails. It must be isolated, and the strength of the healthy portion of the organism must be built up.

To underestimate the extent to which Communism has spread in our nation and in the thinking of the American people could be fatal. Of equal importance is that the basic soundness of the American system and its appeal to oppressed millions abroad not be underestimated.

"What the world generally *wants* most from the United States is our wealth, our goods, our tools. But these are not what it *needs* most—for these are all results. What the world needs most from us is the secret that led to those results."

The secret, Judd said, is our economic system which provides maximum opportunity and incentive for men to get ahead. It came from a political philosophy based on the right of the individual, which, in turn, came from a religious faith—the belief that there is a Creator, and that every man is endowed by his Creator with certain inalienable rights arising from his importance and worth as a child of God.

Judd asserted that physicians must work hard in the groups that are dedicated to the causes in which they believe, and this includes political groups. "If you can't change the mind of your Congressman," said Judd, "then work to change your Congressman."

Doctors should become willing candidates for public office, the

former Congressman believes, and emphasized that this involves hard work and sacrifice. "There are many bruises in this business," he concluded, "but freedom is worth it."

Panel on Community Health

Representatives of five allied health professions presented the problems and viewpoints of their groups to doctors during a panel discussion on "Working Together For Better Community Health Care."

Dr. Robert Mayo Tenery of Waxahachie, past president of TMA, moderated the panel.

Presenting viewpoints of the dental profession was Crawford A. McMurray, D.D.S., of Ennis who is secretary-treasurer of the Texas Dental Association. He cited the dentists' belief in the importance of community health programs and said that too often health programs were "crash" programs. The medical field must take leadership in longer-term health programs, he advised, and pointed to such programs now being undertaken by dentists. These include dental care for the homebound, oral cytology examinations for oral cancer, dental health programs in schools, dental care for the indigent in dental clinics or in private offices, mouthguard programs for those engaged in contact sports, speaker's bureaus on dental health subjects, promotion of science fair projects on health subjects in local schools, fluoridation of community water supplies, and an education program on the effects of fluoride contaminated water in areas with too much fluorine.

He suggested that coordinated effort by all professions interested in an effective health program should be developed in every city and town in the state.

Mr. C. B. Francis of Austin, president of the Texas Nursing Home Association, said that nursing homes should play an important role in the community health team. Recent dynamic developments in this field, he said, are bringing nursing homes into the light and out of the shadows. With the support of doctors, he added, nursing homes can make a genuine contribution to health care.

Pointing to some of the serious problems of nursing homes, he cited the need for low cost nursing home services. Nursing homes have a serious deficiency of income, he noted, and without sufficient income they cannot do their best. He said there was encouraging progress toward raising standards of nursing homes and that now all states have licensing laws on sanitation. In addition the American Nursing Home Association and the American Medical Association are working to establish a national council on nursing homes for better patient care. Two important needs are public understanding and leadership from medical associations.

Mrs. Lillian E. Taubert, R.N. of San Antonio, Executive Director of the Texas Nurses Association, spoke of the changing role of the professional nurse in the hospital, of the problems of the nursing profession financially, competition with other professions in which women could have well-compensated employment, and the nursing shortage.

She called for mutual sharing of expectations doctors and nurses have of each other so that they can operate effectively and efficiently as a team on behalf of the patient.

"The changing and ever-expanding character of the hospital requires a far different type of nursing practice than the relatively simple organization in which nursing was practiced a few years ago," she said.

Greater flexibility is called for, she said, and increasingly, procedures formerly performed by physicians have been shifted to nurses because doctors devote their time to larger numbers of patients.

Some of the problems of direct service to patients, she said, are how the professional nurse can best use her time, which duties she can relegate to others and whether the delegations will be accepted by hospitals, physicians, and the public.

She cited a need for more adequate education programs in which nurses learn not only technical skills but depth of understanding so that they can deal with ever-changing and increasingly complex responsibilities.

Fort Worth pharmacist W. R. Whitten who is also a director of the Texas Pharmaceutical Association and a member of the Texas State Board of Pharmacy noted that the root of most misunderstandings was lack of communication. He praised cooperative programs as a good way to help communication.

Mutual problems between physicians and pharmacists were topics of his discussion, including physician-ownership of pharmacies and lack of pharmacies in hospitals.

The State Board of Pharmacy has started a program of routine checking of pharmacies on unauthorized refilling of prescriptions and the over-the-counter sale of prescription drugs. He asked doctors to report any violations to the State Board of Pharmacy and called upon them to cooperate with pharmacists when they are called for refill authorizations. The pharmacist is merely cooperating with the law by calling, he said.

He cited one area in which pharmacies could be of help in health education by providing a stand for public health literature, which is placed there by local physicians.

A past president of the Texas Hospital Association, Mr. George B. Pearson of Tyler, pointed to three specific community health problems: the continuing and growing shortage of trained personnel in health related fields, the need for intelligent and farsighted planning of health facilities in Texas, and the need for some means to control utilization of hospital admissions.

He said that additional schools are needed to train new students, adequate salaries are necessary to encourage young people, and they must be encouraged to consider the health professions even at the grade school levels. Adequate working conditions are also necessary to keep trained personnel, he said.

On hospital planning, he warned that if the health fields fail to make a concerted effort to promote voluntary local planning of facilities, before too long state and federal agencies will be creating bureaus to do such planning.

He called upon doctors to solve the problem of utilization of hospital admissions, including such areas as diagnostic procedures that can be done in offices instead of hospitals and excessive stays in hospitals. He cited the tremendous cost to a community to provide additional hospital beds, now estimated at $30,000 per bed.

Panel on Public Relations

Medicine's Basic PR Program.— "Public relations is not a mystical thing. It is not much more than sound management plus a jigger

of old fashioned horse sense. It has only one other major requisite as far as the medical profession is concerned—it must always have the best interest of the patient in mind."

Dusting the mystery off public relations, James R. Hickox of Chicago explained the master plan of the AMA; it is available to state and county societies for the asking. Hickox is director of the Program Services Department of the AMA's Communications Division.

The plan, he said, outlines eight areas of activity which, if understood and practiced by most doctors, will immeasurably strengthen medicine's overall public relations stature. Three of these are rooted in basic policy decisions—a sound emergency call system, a grievance committee, and medical care for all regardless of ability to pay. The others require policy decisions, but are learned techniques; they consist of developing effective media relations, developing a speakers' bureau, orientation of new members, initiation of public service projects, and citizenship activities.

Hickox commended Texas for the overall excellence of its programs, and made three recommendations. First, a survey should be conducted at the county society level to determine what condition the society is in now. "You will probably be amazed at the number of doctors who have somehow evaded a working knowledge of the PR policies, and the obligations they have to insure the success of same," he said. Second, leaders of each society should evaluate the survey, and set forth a definitive set of policies as outlined in the AMA's basic PR manual. Third, every doctor should be educated regarding the plan and encouraged to participate in some way.

Hickox then mentioned the following aids and services provided by the AMA:

1. Bulk copies of *Today's Health* available to county societies without charge.

2. Feature stories on items of medical interest for use by house organs.

3. Films, recordings for radio, and TV film material on many subjects. General information on how to use TV advantageously. Films and speech materials for schools.

4. A stable of about 150 speakers throughout the country. Speech material on any subject. To help doctors improve their abilities as public speakers, a one-day seminar can be arranged for local groups.

5. *PR Doctor*, a publication devoted to programs and helpful ideas, for all PR committees.

6. Materials to promote interest in medical careers.

7. Packets of information on virtually any aspect of public relations.

8. Materials to help educate medical assistants, posters on general medical themes, pamphlet racks and pamphlets, and "To All My Patients" plaques available at nominal cost.

9. Newspaper ads. Complete helps for "Operation Hometown."

10. For freight charges only, exhibits ranging in size from tabletop to 30 feet in length.

11. Help on every aspect of health fairs.

12. For suitable purposes, the editor of *JAMA* will make available any art, photographs, or type that has been used in AMA publications.

13. To help medical executives answer charges frequently leveled at the profession, a PR Memo is sent to executive secretaries; it will be sent also to chairmen of PR committees on request.

14. To help make local opinion leaders aware of the activities and views of medicine, physicians may ask that such laymen be placed on the mailing list for *AMA News*.

15. The AMA provides news media with regular releases on both scientific and legislative matters of medical interest.

The Small County Society.—In small societies, doctors may be few and far between, but public relations is not. It is a daily part of the doctor's life and usually is at the person-to-person level.

This is the main difference between PR efforts in large and small societies, said Dr. Sam A. Nixon, Jr., Floresville, chairman of the Gonzales County Medical Society's committee on public relations.

Emergency calls can be handled by any central point which is open 24 hours a day. It may be a hospital if there is one, or a nursing home, or a druggist or another physician by mutual consent. The important thing is that the doctor keep his answering service informed of his whereabouts, and that he let his patients know who or where to call if emergencies arise.

"We have a grievance committee," Dr. Nixon said, "but it has never heard a case." Patients either complain directly to the doctor and grievances are worked out, or patients complain to friends or relatives but never to the doctor himself. The former is most often the case, for rural practitioners are not only doctors, they are friends of human beings and as such are deeply involved in the life and activities of the community.

"Good press relations and speakers bureaus are informal and natural in small societies," Dr. Nixon commented. Editors are always glad to print an item about medicine, especially if there is a local connection. We do ask that our names not be used in connection with anything medical without our express permission, to prevent such items as "Mr. Smith died yesterday; Dr. Nixon was treating him." As for speakers bureaus, all one has to do is say "Yes" when a civic club program chairman is faced with an empty slot day after tomorrow. The question and answer periods that follow are good fun, and give the speaker excellent exercise in parrying questions designed to get free medical advice.

"We may not be formal or go strictly by the book, but we get things done just the same," Dr. Nixon concluded.

The Medium Size County Society.—The public relations programs outlined in the AMA's manual are adaptable to societies of any size, said Dr. C. Mac Geyer of Wichita Falls, chairman of the Wichita County Medical Society's committee on public relations.

He emphasized the need for training, enthusiasm, and experience in public relations committees, and recommended that terms should run from two to four years and that the chairman should have a period of indoctrination in the field.

For each year, the committee should have a written plan for a positive program; it may include only one or two projects, but these should be realistic and aimed toward self-perpetuation so that over a period of years much can be accomplished, he said.

"The best PR project is one which provides the medical society with a chance for service plus publicity," Dr. Geyer said. He emphasized that publicity must be carefully planned and must be aggressive. As an example of the importance of this aspect, he told

of his society's active efforts to provide excellent publicity about its Sabin vaccine program, and to assure that all reports mentioned the medical society's sponsorship. An informal poll late in the program revealed that a substantial number of people still thought that the program was being sponsored by "the government." Efforts were redoubled to clarify the issue.

Other areas in which Wichita County Society has given service and promoted good public relations include obtaining a medical examiner, welfare problems, a 24-hour pharmacy service, and sponsoring a program to provide proper equipment and adequate training of crews for the city's emergency vehicles. "The drivers and crews were especially grateful," he said. "Many of them told us that they had felt inadequate and almost helpless in certain emergency situations, and they were eager to learn proper procedures and techniques."

"The recent encephalitis and mosquito problem in Texas provides an opportunity for PR activity by medical societies," Dr. Geyer said. Where local health units and government have done a good job in vector control, they should be publicly commended by physicians, and if programs are less than adequate, physicians can make this known and help provide facilities and personnel to improve the situation.

The Large County Society.— "Public relations takes time and it takes people who understand it, are interested in it, and are willing to give their time to do the plain hard work which it involves." Dr. B. B. Markette of San Antonio added that Bexar County Medical Society is fortunate enough to have a full-time executive secretary and the part-time services of a public relations expert, as well as a budget for PR programs.

"Our worst enemies are apathy and complacency; we have to sell our members the idea that PR is needed," he said.

In establishing good press relations, doctors must realize that newspapers are in business, that reporters have a job to do and deadlines to meet, and that they can do a good job of medical reporting only when physicians make themselves available to answer questions and provide accurate information.

"We have made it a point to get to know the people whose business is getting and giving news to the public," Dr. Markette said. "We also buy ads in the local papers—the amount of free help and publicity that we have received in return has been astonishing," he added.

Regarding cooperation with reporters, Dr. Markette had this to say: "When a reporter calls or comes to me for information he doesn't demand it, he asks for it. But I treat it as if it were a demand. If I don't know the answers I say so, but I offer to find out, or suggest someone else who might help. The only thing you have to do is tell the truth. It is really easy."

130 Auxiliary Members Attend Austin Meetings

The Woman's Auxiliary to the Texas Medical Association had a total attendance of 130 at its fall executive board meeting Sept. 17 and School of Instruction on Sept. 18. "Advice by the Pound" was the theme, emphasizing weighing the usefulness of Auxiliary activities.

Mrs. W. D. Nicholson, Lake Jackson, president, presided over the executive board meeting at which Dr. A. O. McCary, chairman of the TMA Advisory Committee to the Woman's Auxiliary, addressed the group.

A dinner for the executive board members was held at Green Pastures on Thursday evening. Arrangements for the dinner and entertainment were made by Mesdames Eugene P. Schoch, Jr., Donald E. Pohl, R. N. Snider, Albert Vickers, and J. R. Rainey. "Flair for Fun and Fashion," a fall style show, was presented by a local store.

The School of Instruction was directed by Mrs. Paul Klinger, San Antonio, chairman, and Mrs. Donald E. Pohl, president of Woman's Auxiliary to Travis County Medical Society, cochairman. Reports were presented in pantomime, and new materials for use by county auxiliaries were displayed. A "Pound of Advice" was given by Dr. Max Johnson, San Antonio, President of TMA. Other speeches concerned "An Ounce of Prevention" by Dr. Morris Fishbein, Chicago, editor of *Medical World News;* "The Full Pound" by Kenneth Pepper, D.D., Fort Worth; and "Conservative Measure" by Mrs. Lee Ann Elliott, Chicago, AMPAC representative. An informative talk was given by C. Lincoln Williston, Executive Secretary of TMA, on legislation and Texas medicine's positive programs.

An interesting skit entitled "Sleeping Beauty, Don't Be Weighed and Found Wanting" was presented. A demonstration on "Resusci-Annie" and instructions on mouth-to-mouth resuscitation were given by Dr. Robert Umstattd, Austin anesthesiologist.

Round table discussions were followed by film presentations of "Counter-Attack" and "Pulse of Life."

Postgraduate Courses

Cardiovascular Disease, Houston, Dec. 7-9—The Division of Continuing Education of the University of Texas Graduate School of Biomedical Sciences at Houston will present a course on "Recent Advances in Cardiovascular Diseases," in the Texas Medical Center. Dr. Victor A. McKusick, professor of medicine, Johns Hopkins University School of Medicine, Baltimore, will discuss genetics, heritable disorders of connective tissue, and cardiovascular sounds and murmurs.

For further information physicians may write: Division of Continuing Education, the University of Texas Graduate School of Biomedical Sciences at Houston, 102 Jesse Jones Library Building, Texas Medical Center, Houston.

"No thanks. Not after all of the trouble I had quitting."

Out-of-State Speakers, Plans Told For 1965 Annual Session in Alamo City

SOME PLANS for the Annual Session in San Antonio April 28-May 2, 1965, have been released by the Council on Annual Session. Chiefly, these are revisions made in the format adopted last year and the announcement of 21 prominent out-of-state guest speakers obtained to date.

The Granada Hotel has been designated scientific sessions headquarters, along with the nearby Villita Assembly Hall. The St. Anthony Hotel will serve as headquarters for the House of Delegates members and members of boards, committees, and councils. El Tropicano Hotel will serve as headquarters for the Woman's Auxiliary.

With regard to housing, the San Antonio Chamber of Commerce has agreed to coordinate housing for the meeting. Physicians and guests will direct their communications listing three preferences to the TMA Housing Bureau in care of the Chamber of Commerce which will forward requests to the facility of choice. A list of San Antonio hotels and motels will be published in a later issue of *Texas Medicine.*

The Council on Annual Session, having agreed that the Association should undertake the sponsorship of a luncheon on Sunday, has gone on record that the Committee on Medicine and Religion might most appropriately arrange a program for the luncheon with a nationally-known clergyman as guest speaker. The luncheon has been set for 12:30 to 2 p.m.

For 1965 the Sunday meeting of the House of Delegates has been scheduled for 8 instead of 8:30 a.m.

The Memorial Service has been scheduled for 10:15-10:45 a.m. Sunday to allow physicians and their wives to attend 11 o'clock church services should they wish. The Memorial Service will be held on the Alamo grounds.

The Blue Cross-Blue Shield meal will be a breakfast on Friday at 7:30 a. m., a change from Friday, 6 p.m.

In order to allow those of the Seventh Day Adventist faith to attend the Orientation Program, the Council has agreed to schedule the Program on Friday from 8 a.m. to 12 noon rather than on Saturday morning.

Golf and tennis events will be held on Thursday.

The Curbstone Consultations have been rescheduled from Friday at 8 p.m. to Friday from 5 to 6:30 p.m. The Consultations will follow immediately the scientific section meetings which adjourn at 5 p.m., and it is hoped the rescheduling will enhance attendance. Tickets will be necessary for admittance; however, there will be no charge for tickets. Beer, coffee, and soft drinks will be served.

Since there are no meeting room facilities suitable for closed-circuit television presentations, there will be none offered in 1965.

The Texas Academy of General Practice Seminar will be held Thursday from 12:30 to 6 p.m. The Symposium on Alcoholism to be sponsored for a second year by the Texas Commission on Alcoholism and the University of Texas will be Thursday at 8 p.m. with Dr. Marvin A. Block, New York, as guest speaker. The Committee on Nutrition will sponsor a Symposium on Nutrition on Friday from 8:30 to 9:45 a.m.

The Symposium on Transportation Safety sponsored by the Transportation Safety Committee will be Saturday from 10 a.m. to 12 noon.

A Friday luncheon from 12:30 to 2 p.m. has been set for a "Teacher-Practicing Physician Relationship" program sponsored by the Committee on Liaison with Medical Schools.

The Committee on Nuclear Medicine has indicated its wish to sponsor and to participate in several programs.

Twelve additional guests will be secured shortly to complete the roster of out-of-state speakers for the 1965 Annual Session. The following speakers have been obtained:

Dr. Marvin A. Block, assistant clinical professor of medicine, University of Buffalo Medical School, Buffalo; Dr. Frederick C. Blodi, associate professor of ophthalmology, State University of Iowa, University Hospital, Iowa City; Dr. John E. Bordley, otolaryngologist-in-charge, Johns Hopkins Hospital, Baltimore; Dr. Frank P. Brooks, associate professor of medicine and physiology, University of Pennsylvania School of Medicine, Philadelphia; Dr. David S. Carroll, clinical professor of radiology, Department of Radiology, University of Tennessee College of Medicine, Memphis, Tenn.;

Dr. Israel Davidsohn, director, Department of Pathology, Mount Sinai Hospital, Chicago; Dr. Giulio Gabbiani, research assistant, Institute of Experimental Medicine and Surgery, University of Montreal; Dr. Samuel B. Johnson, professor and head, Division of Ophthalmology, University of Mississippi School of Medicine, Jackson, Miss.; Dr. Robert R. Kierland, head of section of dermatology, Mayo Clinic, Rochester, Minn.; Dr. Averill A. Liebow, John Slade Ely professor of pathology, Yale University School of Medicine, New Haven, Conn.;

Dr. Max M. Novich, attending orthopaedist, Doctors Hospital, Newark, N. J.; Dr. William J. Oliver, associate professor of pediatrics, University of Michigan Medical School, Ann Arbor, Mich.; Dr. Joseph H. Pratt, chief, section of surgical gynecology. Mayo Clinic, Rochester, Minn.; Dr. Hyrum R. Reichman, assistant clinical professor of surgery, University of Utah College of Medicine, Salt Lake City, Utah; Dr. William H. Remine, assistant professor of surgery, Mayo Foundation, Rochester, Minn.

Dr. David C. Sabiston, Jr., chief of surgery, Duke University, Durham, N. C.; Dr. John J. Shea, Memphis Otologic Clinic, Memphis, Tenn.; Dr. Wilson T. Sowder, state health officer of Florida, Jacksonville, Fla.; Dr. Donovan F. Ward, President, American Medical Association, Dubuque, Iowa; Dr. Thomas H. Holmes, professor of psychiatry, University of Washington, Seattle, Wash.; and Dr. Christian Hermann, Jr., associate professor of medicine and neurology, Division of Neurology, California Medical Center, Los Angeles.

Trustees Receive Mrs. Thompson's Estate

Cash and securities valued at about $873,000 were placed in the hands of the trustees of Texas Medical Association on Sept. 28 by executors of the estate of Mrs. Sam E. Thompson.

Mrs. Thompson's bequest, the largest the Association has ever been given, also includes 661 acres of ranch land in Kerr County, valued at nearly $70,000. This property will become available to the Association sometime in the future.

A past president of the Woman's Auxiliary and the wife of the late Dr. Sam E. Thompson of Kerrville, Mrs. Thompson died on Feb. 13, 1963. In her will, she directed the trustees of TMA to invest the monies and to pay sums of $5,000 annually to her two sons and to her daughter-in-law during their lifetimes. The remaining income from this trust estate is to be used for the purpose of operating and maintaining the Memorial Library.

The Board of Trustees has for several years administered the Sam Thompson Student Loan Fund, with total assets now exceeding $1,000,000. This fund was established by Dr. Thompson's legacy. Eventually, proceeds from the sale of the Kerr ranch will be added to the Loan Fund, Mrs. Thompson's will has stipulated. Presently, the ranch is in the possession of W. H. Fleming of Kerrville to whom Mrs. Thompson willed it during his lifetime.

Voters To Consider Kerr-Mills MAA

The MAA (Medical Aid for the Aged) phase of the Kerr-Mills program goes before Texas voters on Election Day Nov. 3 in the form of a proposed State constitutional amendment.

Passage of this amendment is required before the MAA phase can be implemented in Texas. The amendment would empower the legislature to authorize vendor payments for medical care for needy individuals 65 years of age and over who are not Old Age Assistance recipients and who are unable to pay for needed medical services.

The amendment, which will be the third one on the November ballot, has the official support of the Texas Medical Association.

At present, more than 229,000 Texas OAA recipients are covered by a Blue Cross-Blue Shield health insurance policy purchased by the State of Texas with funds made available by the Kerr-Mills program. It is for those Texans 65 and over who are not covered by the OAA program and who still need and want outside help in meeting health care costs that the MAA phase is designed.

This is the way the proposal will appear on the ballot:

"FOR the Amendment to the Constitution giving the Legislature the power to authorize vendor payments for medical care on behalf of the needy individuals sixty-five (65) years of age and over who are not recipients of Old Age Assistance, and who are unable to pay for needed medical services; providing for the acceptance of funds from the Federal Government for the purpose of paying such medical assistance; and providing that the amounts paid out of state funds for such purposes shall never exceed the amount that is matchable out of Federal funds; provided further, however, that such medical care, services or assistance shall also include the employment of objective or subjective means, without the use of drugs, for the purpose of ascertaining and measuring the powers of vision of the human eye and fitting lenses or prisms to correct or remedy any defect or abnormal condition of vision."

"AGAINST the Amendment to the Constitution giving the Legislature the power to authorize vendor payments for medical care on behalf of needy individuals sixty-five (65) years of age and over who are not recipients of Old Age Assistance and who are unable to pay for needed medical services; providing for the acceptance of funds from the Federal Government for the purpose of paying such medical assistance; and providing that the amounts paid out of state funds for such purposes shall never exceed the amount that is matchable out of Federal funds; provided further, however, that such medical care, services or assistance shall also include the employment of objective or subjective means, without the use of drugs, for the purpose of ascertaining and measuring the powers of vision of the human eye and fitting lenses or prisms to correct or remedy any defect or abnormal condition of vision."

Memphis To Host Southern Medical Meet

The 58th annual meeting of the Southern Medical Association will be held Nov. 16-19 in Memphis.

The scientific program will include three special symposiums: one on "Drug Therapy, Clinical Pharmacology and Reaction to Drugs in Hospital and Office," one on "Current VD Problems," and a third on "Problems of Adolescents."

There will be 22 section programs, and the following conjoint societies will have programs: American College of Chest Physicians, Southern Chapter; The College of American Pathologists; Flying Physicians Association; The Radiological Society of North America; Southern Electroencephalographic Society; and Southern Gynecological and Obstetrical Society.

Dr. Robert D. Moreton of Fort Worth is president of SMA.

For additional information concerning the meeting, physicians may write to Mr. R. F. Butts, executive secretary, Southern Medical Association, 2601 Highland, Birmingham 5, Ala.

Therapy of Shock Study To Be in Oklahoma

A symposium on therapy of shock of interest to specialists and practitioners and jointly sponsored by the Civil Aeromedical Research Institute and the University of Oklahoma Medical Center is scheduled for Nov. 19-21 at the Center for Continuing Education, Norman, Okla. This program will stress therapy of shock based on laboratory experiments and the clinical experience of the 25 guest participants.

Preceding the symposium, the guest speakers will gather in Oklahoma City and conduct research projects according to their specific interests. This "Shock Tour" will be limited to the guest researchers on Nov. 16, 17, and 18 at the Civil Aeromedical Research Institute. Results of the three days laboratory work will be presented and discussed with current therapeutic measures being stressed at the symposium.

Information may be had from Office of Postgraduate Education, University of Oklahoma Medical Center, 801 Northeast 13th St., Oklahoma City.

Arboviral Encephalitis

1964 Texas Outbreak

Houston and, to a small extent, other Texas cities experienced outbreaks of sleeping sickness during the past summer. The Houston outbreak reached epidemic proportions. Presented are two accounts of appearance of the disease, one from the City of Houston Health Department and the other from the State Department of Health.

Encephalitis in Houston

CHARLES A. PIGFORD, M.D., M.P.H.

The City Health Officer of Houston describes the events surrounding the detection and control of a St. Louis encephalitis epidemic there this summer in which there were 712 suspected cases and 32 deaths believed due to the disease.

DURING the summer of 1964 the City of Houston and Harris County experienced an epidemic of St. Louis encephalitis (SLE). This was superimposed upon the usual summer seasonal incidence of disease caused by entero-viruses, which in some cases produce central nervous system effects variously described as aseptic meningitis, encephalomyelitis, and related terms. The first recorded appearance of arbovirus encephalitis in Houston was postulated by epidemiologic methods and confirmed by laboratory studies. The epidemiologic postulations were by the Public Health Department of the City of Houston, and the initial laboratory confirmations were by the Texas State Department of Health laboratories. Subsequently, tremendous contributions in epidemiology, laboratory work, and control were made by the Texas State Department of Health and by the Communicable Disease Center of the United States Public Health Service. The clinical pathology and virology departments of the Baylor University College of Medicine and Ben Taub Hospital provided data and virologic studies on a large percentage of the initial cases reported and studied. Mosquito control was carried out under the supervision of the City of Houston Civil Defense Department.

The term *arbo* comes from the first two letters of the words "arthropod" and "borne." This is now accepted nomenclature for this group of diseases. Within this classification Casals and Reeves list 28 human disease entities.[6] They generally fall into two main serologic subgroups, A and B. The three types of encephalitis that have produced significant human disease in the United States are western equine encephalitis (WEE), eastern equine encephalitis (EEE), and St. Louis encephalitis (SLE). As indicated by the term *arbo*, all are arthropod borne, or transmitted by insects. The most important vector is the mosquito. Western equine and eastern equine belong to serologic group A; St. Louis belongs to group B, as does yellow fever and dengue fever.

Background

St. Louis encephalitis (SLE) was first intensively studied in 1933, when the virus got its name from an outbreak of the disease in St. Louis County, Mo. The same disease had occurred in Cincinnati, Ohio, and Paris, Ill., during the summer of 1932. It reappeared in St. Louis in 1937.[8] In 1954 an epidemic appeared in the Rio Grande Valley with an attack rate of 163.9 per 100,000.[1] Since then cases have been reported annually from that area. In 1956 there occurred an outbreak of WEE in the South Plains of Texas which gave me first-hand experience with arbovirus encephalitis.[4] Subsequently SLE has been reported from both Lubbock and Hale counties. The last recorded outbreak of epidemic proportions in the U. S. was in St. Petersburg, Fla., in 1962, with 566 suspects of which 192 were confirmed and 39 presumptively confirmed. There were 28 deaths, 14 confirmed, and 14 presumptively confirmed.[3] The virus is from 20 to 33 mμ. in size and can be cultivated. It is principally found in birds, which are the main host reservoir. The most important vector is the mosquito, although the chicken mite, *Dermanyssus gallinae*, has been implicated.[8] The incubation period in man appears to be in the range 7 to 21 days,[8] although 5 to 15 days is frequently stated.[5] There is no specific therapy, but most patients do well on supportive care during the course of the self-limiting disease. There is no commercially available vaccine. The only effective control measure is control of the mosquito vector. In Houston, the principal vector is *Culex quinquefasciatus*, and control was achieved predominantly by killing the larvae and by fogging.

Man, as with other mammals, is only incidentally or accidentally infected. Mammals are not required in the maintenance of the virus, since the usual cycle is bird-mosquito-bird, etc. Mammals apparently show transient viremia of a low

order, and therefore, are not regarded as of first importance in passing the virus to the mosquito. Fig. 1 shows, in the lower left hand corner, the usual bird-mosquito-bird cycle and indicates that man is incidentally infected by mosquito bite.[7] SLE does not affect horses, so it was not incorporated in the chart by the original author. However, with the exception of the horse infection, it fits into the diagram well. Fig. 1 shows the relationship of other arbovirus diseases, in which man is accidentally infected by the bite of an insect.

"Arm Chair Epidemiology"

The development of the SLE encephalitis epidemic in Houston demonstrates the well known fact that morbidity reporting is relatively unreliable and incomplete, and that health agencies must scrutinize carefully mortality statistics. Deaths *must* be reported, and mortality statistics are a valuable tool. On the morning of Aug. 13, morbidity reports indicated that there had been a total of six encephalitis or aseptic meningitis cases in Houston, three in July and three in August. That morning, however, three death certificates indicating encephalomyelitis were received. Since this would be unexpected with the usual summer incidence of central nervous involvement by the enteroviruses, further examination was indicated. A review of all death certificates revealed a total of 12 deaths reported since July 16, which could be suspected as encephalitis. Two of these had been in children. A telephone call to a general hospital revealed recollection by the house staff of some 25 children and 35 adults who had "encephalitis-like" disease. Thus, as shown in Table 1, there were then about 60 suspected clinical cases and 12 deaths suspected from encephalitis. With the exception of the children reported, the suspected deaths were in older age groups. Arbovirus encephalitis was immediately suspected.

At first, it would have been easy to implicate EEE rather than WEE or SLE because of the relatively high death to disease ratio among the suspects. However age distribution in the suspected death group resembled the patterns in SLE in St. Petersburg,[3] and suggested SLE. Serologic studies were vital to the issue. Eight paired sera were obtained and immediately sent to the Texas State Department of Health laboratories in Austin. On Aug. 18 that laboratory reported tentative results indicating arbovirus encephalitis, type and cause undetermined. On Aug. 20, four of the eight cases were reported as having antibody titer rise (CF) for SLE. Two were among the 12 deaths. Thus, Houston was at some point in the course of an epidemic of St. Louis Encephalitis (SLE). Much clinical, pathologic, laboratory, and epidemiologic study would have to follow to develop the details. This evaluation was concurred in by the Texas State Department of Health and the Communicable Disease Center of the United States Public Health Service. With the knowledge and concurrence of the chairman of the Public Health Committee of the Harris County Medical Society, this knowledge was provided to Mayor Louie Welch at 2:30 p.m., Aug. 20, 1964.

Executive Action

Mayor Welch decided to make public announcements concerning the disease through all media. Upon being informed that mosquito control was the only effective means of combating the epidemic, he or-

OCCUPATIONAL EXPOSURE OF MAN TO ARBOVIRUS INFECTION

Work, T. H. 1964. Arbovirus impingement on the natural history of man.

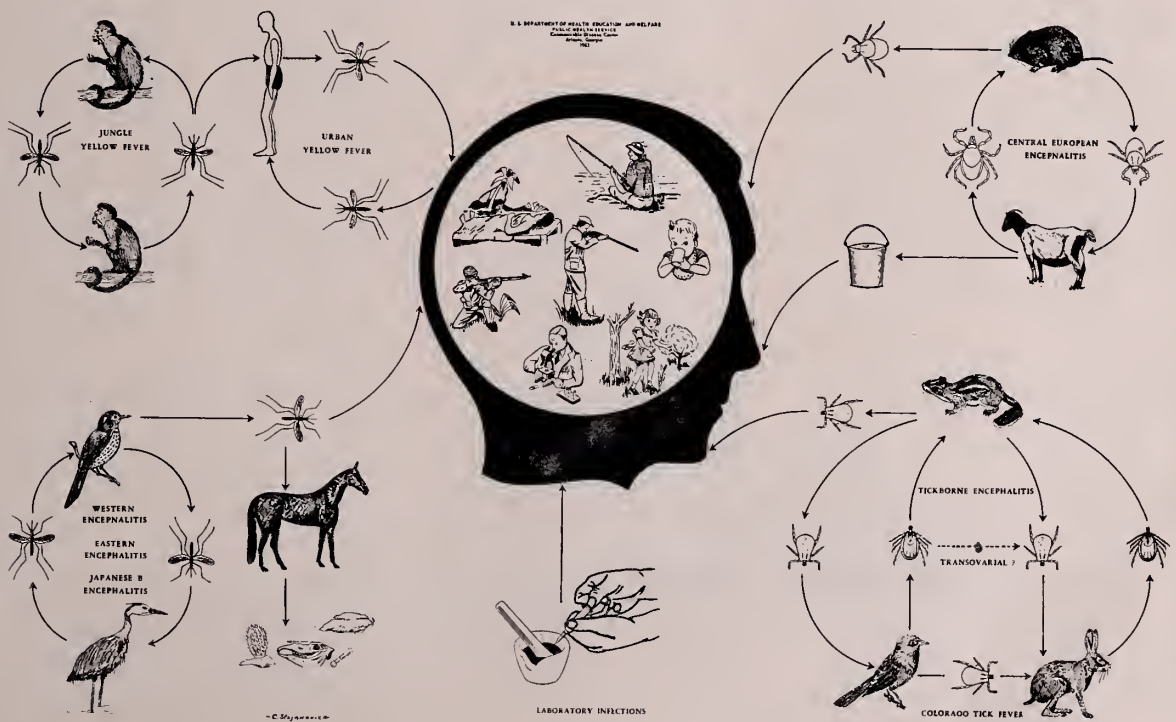


Fig. 1.

TABLE 1.—*Reported Cases and Deaths of Suspected Encephalitis.*

Hr.	Date	Case Reports	Death Certificates
10 a.m.	Aug. 13, 1964	6	12
4 p.m.	Aug. 13, 1964	60	12

dered the Director of Civil Defense to start a major control effort. Within a matter of hours, the pest control industry of the city had mobilized, and abatement operations were begun. Fire Department apparatus was used to apply insecticides. Fifteen power spraying units were diverted by the State Department of Health to Houston from an *Aedes aegyptii* program under way in San Antonio. Four fogging machines of the Houston Health Department were put into action. In ten days the city was covered completely and efficiently. The use of Civil Defense to execute and act as communications center for the control program was a master stroke, since it developed into a live wire operation and concurrently relieved the Health Department of all but technical advice and responsibilities in this area, allowing full time to epidemiologic pursuits.

"Shoe Leather Epidemiology"

On Aug. 21, teams of physicians, epidemiologists, entomologists, biostatisticians, and various other specialists from the State Health Department and the CDC Public Health Service arrived and plunged into work. A great deal of "shoe leather" epidemiology had to be done to find suspected cases, talk with physicians, and do laboratory work upon specimens obtained from the sick, the deceased, the mosquito vectors, and the bird population. Mosquitoes had to be trapped, identified, and processed in the laboratory to isolate the virus. All species of birds had to be captured, studied, and serologic and virus isolation tests made upon their blood. Case records had to be carefully reviewed; blood and stool specimens had to be obtained for serologic and virologic studies to screen cases of SLE from other virus diseases.

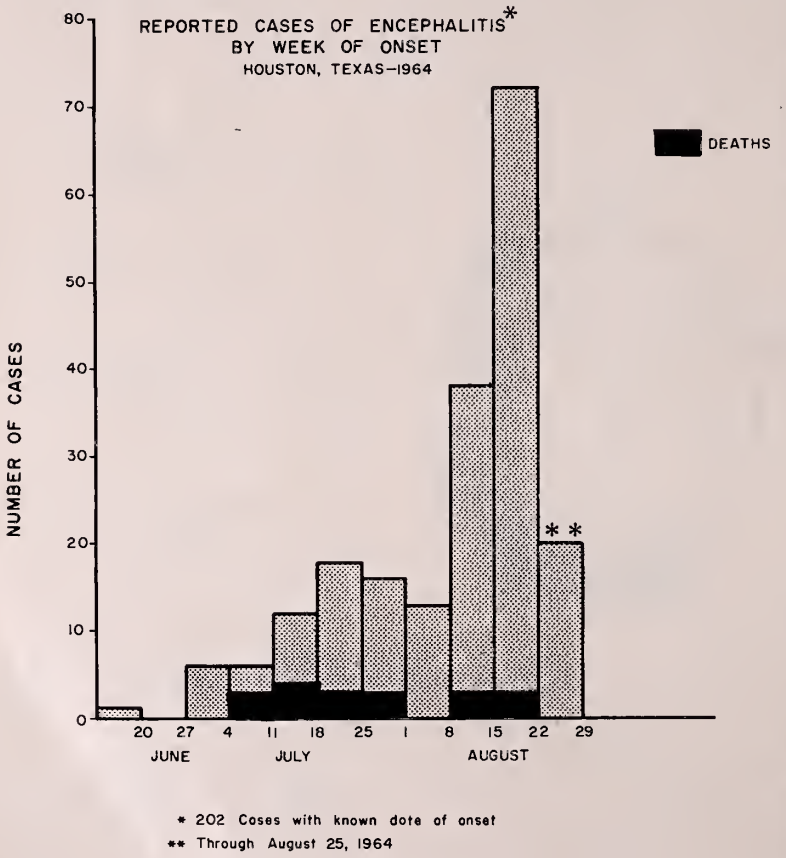


Fig. 2.

All this required a major effort beyond the capacities of the city and which could not have been accomplished without outside help from state and federal health agencies.

Letters were sent immediately to all practitioners of medicine and osteopathy providing information on SLE and requesting telephone and postcard reports of suspected cases, early and late sera for serologic studies, and stool samples for enterovirus isolations. Included was an information sheet reproduced from the "Report of the Committee on the Control of Infectious Diseases" with the permission of the American Academy of Pediatrics.[5] The response was virtually total and began to provide epidemiologic pictures within 24 hours. These will be shown and discussed in sequence as follows:

August 25

By Aug. 25 we had 215 suspected cases and 22 deaths suspected of being due to SLE. This began to provide a picture indicating that the disease might have commenced as early as June and that there was a steady increase through the week ending Aug. 22. The slope of the histogram, shown in Fig. 2, indicated that the peak of the epidemic was yet to come. Parenthetically, the local and national news media requests for information and predictions became a considerable load, necessitating additional communications equipment and the pressing into service of individuals of the Health Department as television and radio speakers and as interviewees. Under such conditions, news media demands are heaviest just at the time the epidemiologic and other health requirements are heaviest upon the Health Officer and his assistants.

August 31

The week ending Aug. 29 showed a peak in suspected cases—but a relative decline in suspected deaths, as shown in Fig. 3. As of Aug. 29 there had been 408 suspected cases with 19 deaths suspected due to SLE. The slope of the histogram was not as acute on Aug. 30 and 31, and many cases then being reported had dates of onset in July and early August. This had been apparent on Aug. 25, when the statement was made that the peak of the epidemic had been reached. This was not concurred in by some and there was a resultant flurry of controversy in

the local and national press. However, that was the way the picture looked and the Health Department reaffirmed the opinion.

September 5

The close of the reporting period on Saturday, Sept. 5, provided the histogram in Fig. 4. As of Sept. 5 there had been 585 suspected cases with 19 deaths suspected due to SLE. The peak during the week ending Aug. 29, previously predicted, was now indisputable. The question then became "how long will it last." This is yet to be answered with exactness, but the advent of cooler weather and "northers" brought a noticeable elimination of cases. The first very cold weather was predicted to assure the termination of vector transmission, at least for this year.

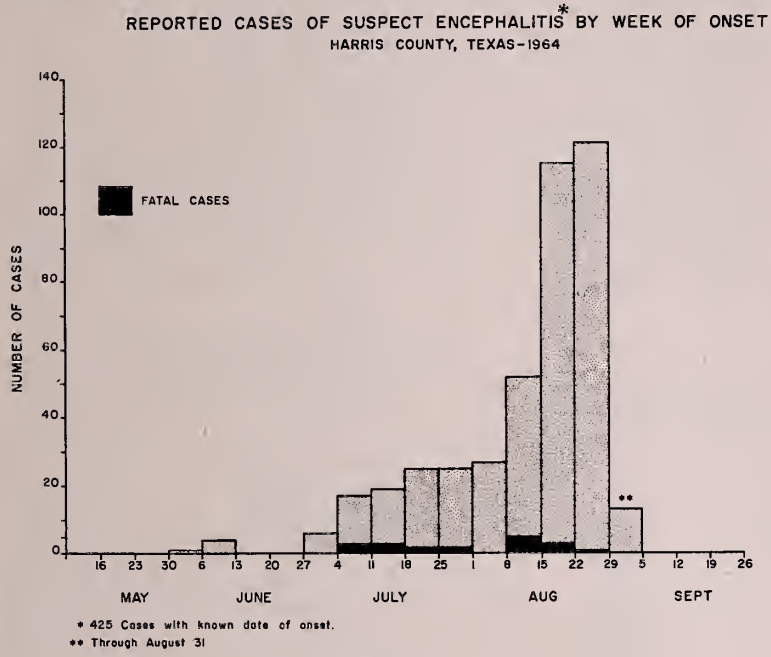


Fig. 3.

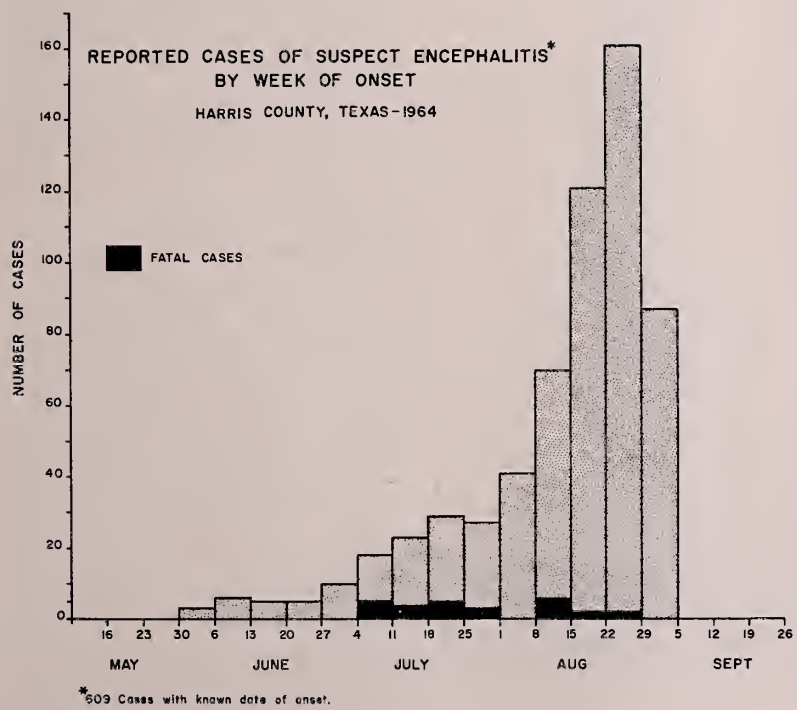


Fig. 4.

September 12

The week ending Sept. 12, shown in Fig. 5, showed a continuation of the downward slope of the curve. There were 673 suspected cases and 30 suspected deaths. The number of suspected deaths did change appreciably because several additional suspected cases were found in a short period of time, through careful analysis of mortality reports—most of them old. This again merely sharpened the epidemiologic picture—it did not materially change the previous data and interpretations. It should be noted that in each histogram the total number of cases for every week changed. This was the result of "shoe leather" epidemiologists running down data on suspected cases and deaths and adjusting the figures for week of onset to reflect the most accurate data. Duplications needed to be eliminated. Corrected diagrams had to be posted. All of these efforts sharpened the data and provided progressively meaningful tables and histograms. This procedure, of course, had to be and must be continuous. Each week the data became better.

September 19

The histogram for the epidemic through the end of the week from Sept. 19 is shown in Fig. 6. It reflected 684 suspected cases and 31 deaths. While readjustments in the previous weeks' data are noted, the overall epidemiologic picture, as discussed previously, did not change materially. It was apparent that we were approaching the end of the epidemic. This was announced in the public press Sept. 20-21.

Laboratory

Serologic changes detected by appropriate laboratory tests provide confirmation of diagnosis in suspected cases of SLE—as they do for the other arboviruses. These changes depend upon the development of antibodies in the blood. Fig. 7 presents a clear picture of the development of viremia and the subsequent rise of titer of three antibodies — neutralizing, hemagglutination (HI), and complement fixation (CF).[7] The HI antibody often appears in two or three days after the onset of symptoms, while the CF antibody develops later. De-

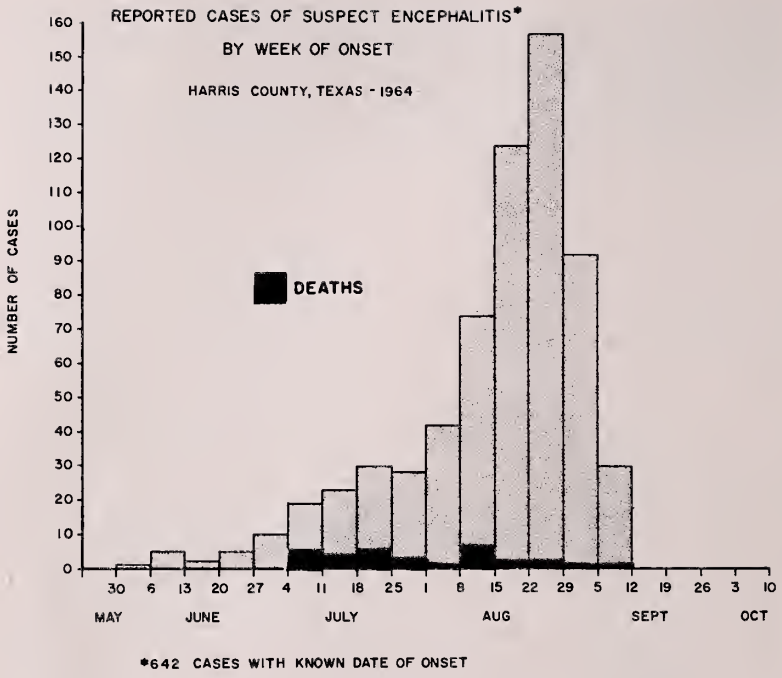


Fig. 5.

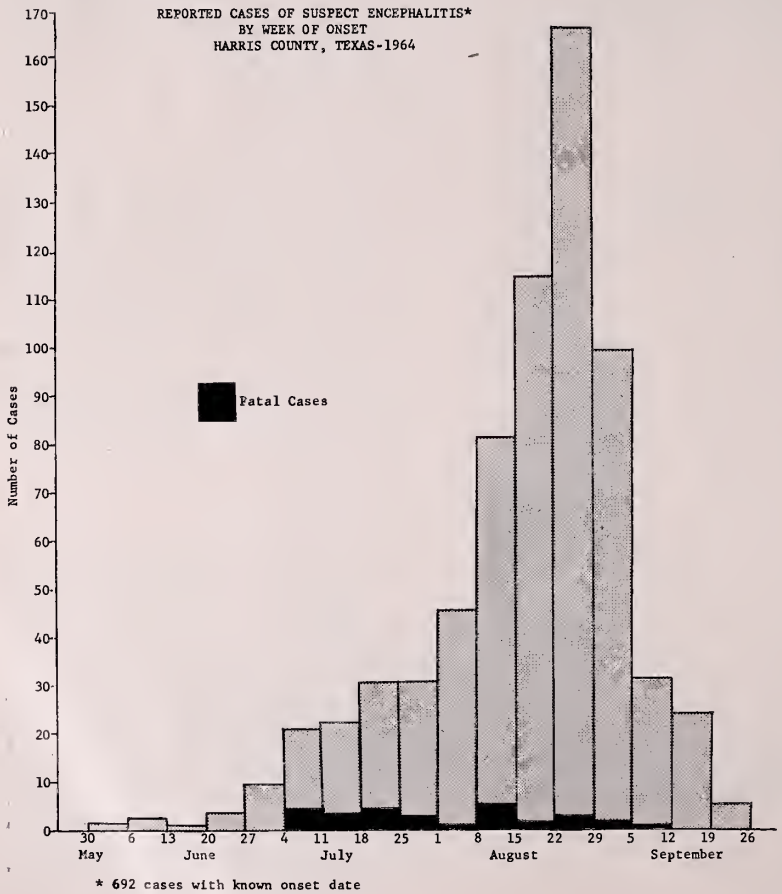


Fig. 6.

tection of a rise in titer, or during epidemic conditions of a single elevated titer in a suspected case, by HI gives a presumptive diagnosis. Variable results require consideration of several possible interpretations, so that the CF tests must be performed on sera collected early (first three to four days) and later (two to four weeks) after onset of illness. A rising CF titer in paired sera, supporting a previously noted rising or elevated HI test provides final confirmation.[2]

In some instances, the SLE virus may be recovered from the brain tissue of fatal cases, from pools of mosquitoes, or from the blood of birds. From Houston, the virus had been isolated from the blood of one blue jay bird and from seven pools of mosquitoes *(Culex quinquefasciatus)* by Sept. 26. No isolations from human brain had been reported.

These laboratory tests are difficult and time consuming. The initial tests were performed by the Texas State Department of Health Laboratories under the direction of Dr. J. V. Irons whose work confirmed initially Aug. 18 the epidemiologic deduction that arbovirus encephalitis was present in Houston. He confirmed it as SLE two days later on Aug. 20. Subsequently, the tremendous laboratory work load was shared by the Communicable Disease Center of the U. S. Public Health Service in Atlanta and by the laboratories of the Department of Virology and Epidemiology of Baylor University College of Medicine.

Much laboratory work remains to be done by all these laboratories to complete the picture.

Conclusions as of September 26, 1964

The histogram for the week ending Sept. 26 is shown in Fig. 8. At this time there had been 712 suspected cases of St. Louis Encephalitis, of which 21 had been confirmed positively and 188 presumptively by rising HI and CF titers. There had been 32 deaths suspected as encephalitis on clinical grounds. From these, the children previously mentioned and several adults had been excluded after thorough clinical, epidemiologic, and pathologic review. Of these 32 deaths, 22 had been confirmed positively by laboratory means, and 10 had been presumptively confirmed.

Virus had been isolated by mouse brain innoculation and recovery in

HYPOTHETICAL DIAGNOSTIC FEATURES OF THREE ARBOVIRUS INFECTIONS OF MAN

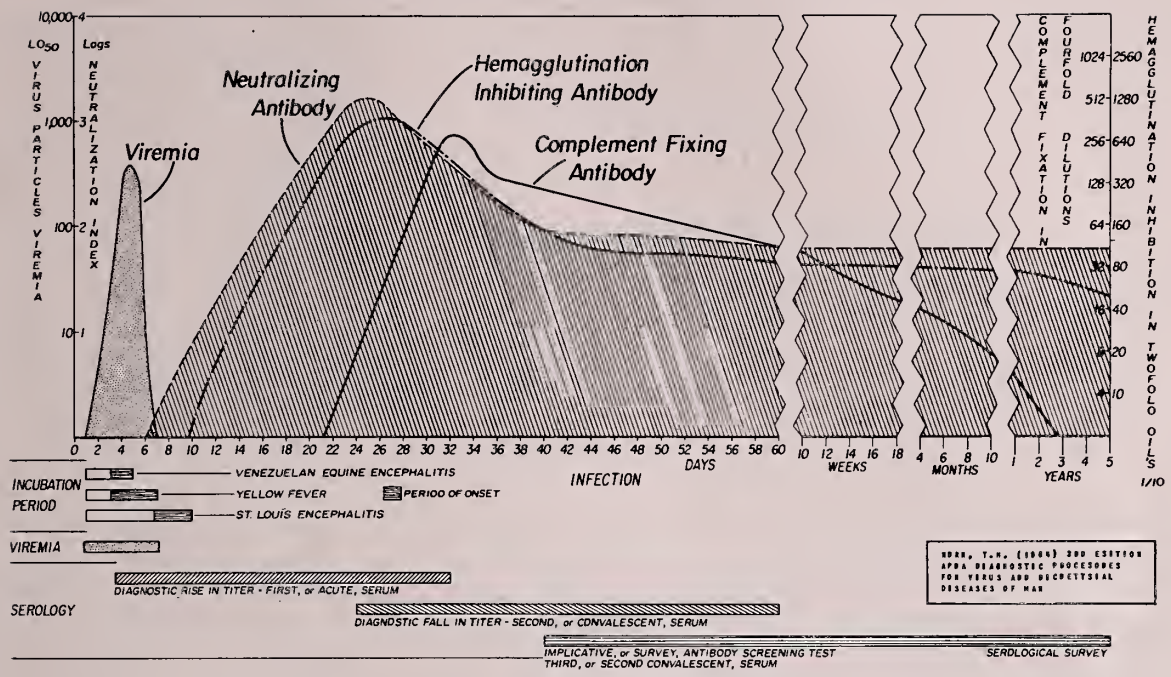


Fig. 7.

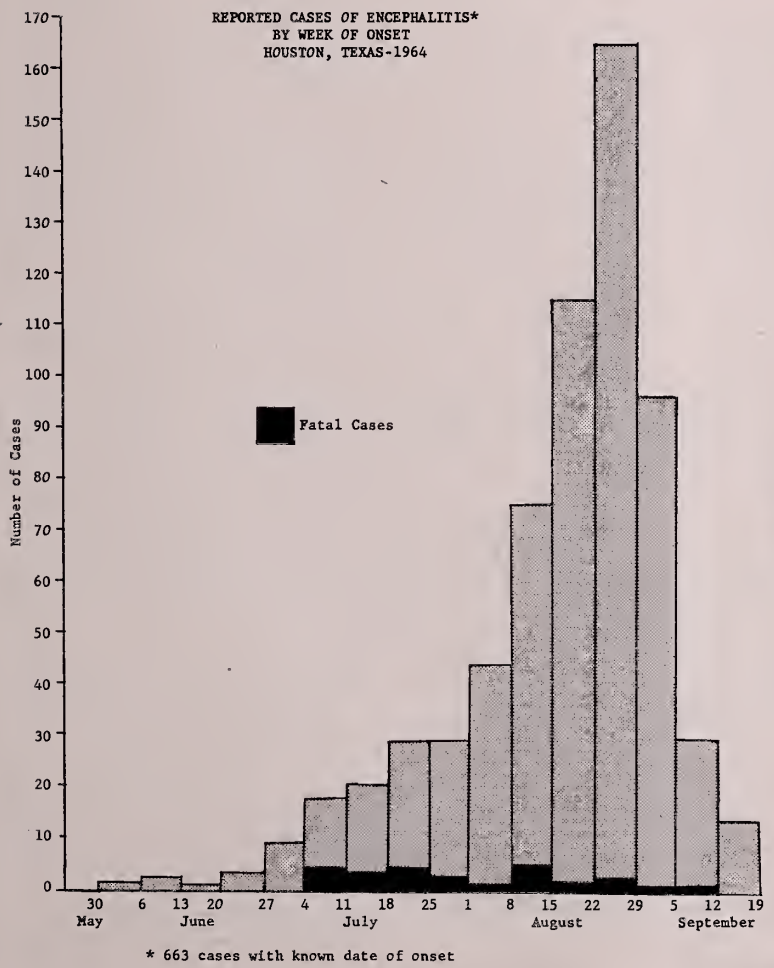


Fig. 8.

one bird, a blue jay, and from seven pools of *Culex quinquefasciatus* mosquitoes. No virus isolations from human brain had been reported, although studies were under way.

The age incidence and attack rates are shown in Table 2. Incidence by sex is shown in Table 3. Incidence by race is shown in Table 4. There was an overall attack rate of 57.3 per 100,000 in the population.

Epidemiologic, clinical, pathologic, virologic, and other studies continue. While the epidemic is almost over, much study and work remains.

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◗ Dr. Pigford is City Health Officer of Houston.

TABLE 2.—*Reported Cases and Deaths of Viral Encephalitis by Age Group, Harris County, 1964.*

Age	Total Cases	Deaths	Cases per 100,000*
0-9	107	0	35.0
10-19	98	0	48.9
20-29	138	0	82.3
30-39	95	2	48.1
40-49	70	0	44.5
50-59	68	7	60.5
60-69	62	10	98.4
70+	66	13	167.0
Unknown	8	0	-----
Total	712	32	57.3

*Based on 1960 Harris County Population.

TABLE 3.—*Reported Cases and Deaths of Viral Encephalitis by Sex, Harris County, 1964.*

Sex	Total Cases	Deaths	Cases per 100,000*
Male	376	23	61.8
Female	335	9	52.8
Unknown	1	0	-----
Total	712	32	57.3

*Based on 1960 Harris County Population.

TABLE 4.—*Reported Cases and Deaths of Viral Encephalitis by Race, Harris County, 1964.*

Race	Total Cases	Deaths	Cases per 100,000*
White	537	14	54.0
Negro	147	18	58.9
Unknown	28	0	-----
Total	712	32	57.3

*Based on 1960 Harris County Population.

New Hospital Completed in Houston

Recently completed at a cost of $5,000,000 is Rosewood Memorial Hospital and Convalescent Center in Houston.

Dr. Donald M. Gready was elected president of the medical staff, and other officers are Drs. Jesse Walter Hofer, vice president, and Harvey B. Koch, secretary. Administrator of the hospital is Bill L. Hamilton.

The medical staff executive committee is composed of the officers and six chiefs of service: Dr. Elmer K. Sanders, surgery; Dr. Loren B. Goss, obstetrics and gynecology; Dr. James M. Skelton, medicine; Dr. Ethel E. Erickson, pathology; Dr. David M. Earl, radiology; and Dr. Audrey E. Posey, anesthesiology.

The nonprofit charity hospital complex is owned and operated by the Wallace E. and Alma E. Johnson Foundation, of Memphis, Tenn.

Rosewood Hospital provides the full range of medical, obstetric and surgical services, in addition to complete hospital operations.

Rosewood Convalescent provides medically-oriented facilities for patients suffering from nonacute, noncontagious illness and disability, including the aged.

Personals

Dr. J. Arnold Bargen of Temple will speak at the Interstate Postgraduate Medical Association in Pittsburgh Nov. 9-12.

Dr. R. R. Lovelady of Coleman has retired from practice.

Guest speaker at the September meeting of the Hill County Medical Society was *Dr. Max E. Johnson* of San Antonio, President of Texas Medical Association.

Dr. Everett C. Fox of Dallas has been elected vice president of the American Dermatological Association.

Robert L. Innes, former assistant administrator of the Ontario Crippled Children's Center, has been appointed administrator of the Galveston unit, Shriners Burn Institute of Shriners Hospitals for Crippled Children.

Chairman of the Southern Medical Association section on gynecology is *Dr. John A. Wall* of Houston. The gynecology section program is to include appearances of *Drs. Joseph R. Feste, Alan L. Kaplan, Philip T. Hudgins*, and *Wall* of Houston; *Dr. Peggy J. Whalley* of Dallas; *Dr. Jack L. Turner* of Fort Worth; and *Dr. William J. McGanity* of Galveston.

Dr. Max A. Stoner, Dr. Lewis A. Leavitt, and *Dr. Willis C. Beasley* of Houston will participate in the section meeting on industrial medicine and surgery at the meeting of the SMA.

Dr. Terry D. Allen of Dallas will be one of the speakers at the Southern Medical Association section meeting on medicine.

Appearing before the section on neurology and psychiatry at the Southern Medical Association meeting will be *Drs. William Kemp Clark* and *Don P. Morris* of Dallas.

Drs. G. C. Nabors of Dallas and *William J. McGanity* of Galveston are named on the program of the section on obstetrics at the SMA meeting.

Dr. Philip A. Deffer of Fort Sam Houston will be a discussant at the meeting of the section on orthopedic and traumatic surgery at the SMA meeting.

Drs. Max A. Stoner and *Lewis A. Leavitt* of Houston are listed on the program of the section on physical medicine and rehabilitation of the Southern Medical Association meeting.

Dr. Gerald D. Barton of Dallas is chairman of the SMA's section on preventive medicine.

Encephalitis Reports from over the State

JAMES E. PEAVY, M.D., M.P.H.
J. V. IRONS, Sc.D.

Officials of the Texas State Department of Health report on findings of two forms of arboviral encephalitis in Texas.

IN THE United States eastern equine encephalitis (EEE), western equine encephalitis (WEE) and St. Louis encephalitis (SLE) are widely distributed.[5] Eastern type infections have been mainly confined to the Atlantic and Gulf coast states, while WEE infections have occurred mainly in states west of the Mississippi River to the Pacific coast. SLE outbreaks also have occurred in areas west of the Mississippi, and in the central and southeastern states including Florida. As indicated by the names, EEE and WEE viruses frequently affect equines but SLE equine illnesses rarely have been reported. Infection occurs principally as a result of bites of mosquitoes which acquire virus from birds or sources other than man or equine. Therefore, these are described as arthropod-borne or arboviral infections. Culicine mosquitoes are believed to be the important vectors of SLE and WEE in man.

"Sleeping sickness" in horses long had been known in Texas and human infections had been suspected. Clinical and serological evidence of the probable concurrent activity of all three of these arboviruses was found in 1941[3] in the Lower Rio Grande Valley. The following year the Texas State Health Department and the Hooper Foundation, University of California, San Francisco, conducted entomologic and serologic surveys[4] in the vicinity of San Benito, Cameron County, and limited serologic surveys in several areas, but very little evidence of viral activity was found.

In the 15-year-period, 1949-1963, as shown in Table 1, 2,030 cases of infectious encephalitis were reported to the State Health Department. Certainly many of these cases were post-infectious or unspecified, but in some of these years it was shown that the St. Louis or western equine viruses were responsible for most of the illnesses. Increasingly, reports of arboviral encephalitis are based on laboratory confirmation.

One of the largest recorded SLE outbreaks occurred in Hidalgo County in the Lower Rio Grande Valley in 1954.[2,7,10] This outbreak was investigated by the State Department of Health and the U. S. Public Health Service. Records of 373 suspected cases were collected, but it was estimated that there were more than 1,000 cases. Most of the cases occurred in late August and early September. The SLE virus was recovered from the brain of one of the fatal cases. One hundred and twenty cases[1,11] of St. Louis encephalitis occurred in Cameron County, Texas, in the summer of 1957. On the basis of serological tests of persons who remained well at the end of the epidemics, inapparent infections evidently were common. Many illnesses were mild. While the mortality rate[1,7] was hardly more than 2 percent there were many serious illnesses. Several pools of *Culex quinquefasciatus* mosquitoes collected during the outbreaks showed the presence of SLE virus. Infected mosquitoes also were found in January, 1958, well after the end of the second epidemic.

An outbreak[8] occurred in the summer of 1956 in the Texas High Plains with the heaviest concentration of cases in the Plainview-Lubbock area. Both SLE and WEE cases were serologically confirmed, unlike the lower Valley outbreaks when only SLE was found. Cases of WEE or SLE have been confirmed in the Texas High Plains[9] nearly every summer in recent years. WEE virus[6] has been recovered repeatedly from pools of *Culex tarsalis* but less often from *Culex quinquefasciatus* mosquitoes in this area. During the summer and early fall of 1963,[9] more than 70 suspected cases of encephalitis

TABLE 1.—*Reported Incidence of Infectious Encephalitis in Texas (1949-1963).*

Year	Cases	Year	Cases
1949	48	1957	196
1950	59	1958	148
1951	63	1959	161
1952	82	1960	116
1953	104	1961	93
1954	336	1962	95
1955	96	1963	87
1956	346		

occurred in or near Hale County. Thirty of these were serologically WEE laboratory presumptive or confirmed. Twenty-nine cases were of unknown etiology, but clinically indistinguishable from WEE cases.

Laboratory Diagnosis

Laboratory tests[9,10] are required to confirm suspected cases of encephalitis in view of the overlapping clinical manifestations and wide range of severity of illnesses which often makes clinical diagnosis extremely difficult. The more severe cases characterized by stupor, tremors, convulsions, coma, and even death are more easily recognizable but the specific etiology can be learned only by application of laboratory procedures. In less severe cases differential diagnosis of nonparalytic polio, aseptic meningitis due to enteroviruses, mumps virus, or other problems may arise.

In suspected arboviral infections, three blood specimens are usually required. Five to 10 ml. of clotted blood or sterile serum should be drawn. The first should be taken early after onset of illness and serves as a baseline for measuring significant titer increases later. The second should be drawn one to two weeks later for possible significant increase in hemagglutination—inhibition (HI) titer. A third blood drawn four to six weeks after onset of illness frequently is required for possible significant increase in complement fixation (CF) titer. An early HI titer is of presumptive diagnostic value and should be confirmed by demonstration of an eight-fold or greater rise in HI or four-fold or greater rise in CF titer. A 1:320 HI titer or 1:16 (4+) CF or greater titer on a single optimal serum may be accorded diagnostic significance. The recovery and identification of the specific virus should be attempted in fatal cases. Portions of fresh brain tissue should be submitted in the frozen state for this test. Where a probability of aseptic meningitis due to an enterovirus is suspected, stool and spinal fluid specimens are required for virus culture since serological tests are usually impractical.

Arboviral Encephalitis Findings in 1964

A few physicians in the Texas High Plains requested aid in diagnosis of encephalitis in June, but the first laboratory confirmed WEE patient, from Hale County, became ill in mid-July. The first proved SLE patient in this area, from Lubbock County, became ill a month later. Unless there is a late increase in SLE cases, which seems unlikely at present, WEE cases will predominate as during recent years. As in recent years it appears that a high percentage of suspected cases will lack laboratory confirmation. It is important in correct reporting of infectious encephalitis that laboratory confirmation of suspected cases be obtained since it is difficult to assess effect of control measures unless adequate data on prevalence and distribution of WEE and SLE cases are known. As of mid-September the results of serological tests on suspected cases are shown in Table 2. Undoubtedly confirmation of several additional cases will be obtained.

TABLE 2.—*Suspected Encephalitis in 1964 in Texas High Plains Based on Serological Findings.*

County	Confirmed SLE	Confirmed WEE	Presumptive SLE	Presumptive WEE	Inconclusive
Bailey			1		
Hale		5	1	8	Several
Hutchinson					1
Lamb			1		
Lubbock	1	2		4	Few
Potter					2
Wheeler				1	
Others*	1		5		

*Angelina, Bowie, Galveston, Howard and Washington.

As in past years, the State Health Department, the Disease Ecology Section of the U. S. Public Health Service, the Hale and Lubbock County Health Departments and others have continued investigation and attempted control of the outbreak. As in the past, attack rates appear to be highest in Hale County. This area of the Texas panhandle is flat and vegetated, undrained playa lakes are numerous. Irrigation practices, too, seem to render control of culicine mosquitoes difficult.

St. Louis encephalitis apparently had not previously occurred in Houston and was first suspected when the City Health Department learned of an unusual number of serious illnesses with several deaths. Complement fixation tests of a few pairs of sera in the Laboratories of the State Health Department confirmed that a SLE outbreak indeed was in progress. Meanwhile the Communicable Disease Center of the U. S. Public Health Service, the State Health Department and the City Health Department mobilized resources aimed at trying to bring the outbreak under control.

By mid-September laboratory confirmation of numerous SLE cases had been obtained in Houston or Harris County. It appeared likely that a few cases in nearby counties had acquired infection in Houston. This possibility pertained to a confirmed SLE case in Nueces and one suspect each in Angelina, Galveston, and Washington counties and Columbia, Miss. Obviously several additional confirmations will be obtained. Confirmatory serologic tests are in progress both in the Laboratories of the Department of Virology and Epidemiology, Baylor University College of Medicine, and the State Health Department.

Several pools of *Culex tarsalis* mosquitoes collected in Hale County in late July and early August were found harboring WEE virus. SLE virus has not yet been recovered from mosquitoes collected in Hale or Lubbock Counties in 1964. It should be added that among live mosquitoes received for virus tests from this area *Culex tarsalis* predominated. In Houston, however, *C. quinquefasciatus* was the principal culicine mosquito collected late in August and submitted for virus tests. Among the first of these mosquitoes, pools of *C. quinquefasciatus* were found harboring SLE virus.

It should be emphasized that the only effective way to break the chain of transmission is to attack the mosquitoes. Experience has shown other approaches to be ineffective. Control of epidemics depends upon their early recognition and institution or intensification of control measures.

Summary

Outbreaks of St. Louis encephalitis (SLE) occurred in the Lower

Rio Grande Valley in 1954 and 1957, in the Texas High Plains centering in the Hale-Lubbock Counties in 1956 and in Harris County in 1964. Western equine encephalitis (WEE) also is endemic or enzootic in the Texas High Plains. Eastern equine encephalitis (EEE) and WEE infections in horses have occurred but EEE infections in man rarely have been reported in Texas. Laboratory diagnosis depends primarily upon the results of serologic tests of paired sera in which hemagglutination (HI) and complement fixation (CF) procedures are utilized. The former is primarily a rapid presumptive test. Confirmation depends upon tests of paired sera showing significant rise in CF antibody titer. A significant increase in titer in SLE frequently is not obtained in less than three or more weeks and in WEE less than four or more weeks after onset of illness. *Culex tarsalis* is believed to be the principal mosquito vector of WEE while *Culex quinquefasciatus* is thought to be the principal vector of SLE. Control depends upon effective mosquito control measures.

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◗ Dr. Peavy is Texas State Commissioner of Health and Dr. Irons is Chief, Laboratory Section, State Health Department, Austin.

M. D. Anderson Announces Ninth Annual Clinic Conference

RECENT Advances in the Diagnosis of Cancer" will be the subject of the ninth annual clinical conference to be held at the University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Nov. 6-7. Twenty-six scientists and physicians will participate in the program, presenting recent developments in nuclear medicine, pathology and cytology, medicine and clinical pathology, and diagnostic radiology. Cosponsor of the conference is the Division of Continuing Education of the University of Texas Graduate School of Biomedical Sciences at Houston.

Partial support for this year's conference has been contributed by the American Cancer Society, Texas Division; a Community Cancer Demonstration Project Grant from the Cancer Control Program, United States Public Health Service; and the Doubleday Fund.

The first session on Friday morning will be on nuclear medicine, and among papers to be presented are "Use of Isotopes and Advances in Scanning Techniques" by William C. Dewey, Ph.D., associate physicist; "Localization of Labeled Rabbit Antihuman Fibrin Antibodies in Human Tumors" by Dr. David Marrack, associate pathologist; and "Use of Radioactive Phosphorus in the Diagnosis of Esophageal, Gastric, and Rectal Lesions" by Dr. Robert S. Nelson, internist, all of the M. D. Anderson Hospital staff.

The second session will be on pathology and cytology, and papers to be presented will include "The Cryostat: Use in Diagnosis of Neoplasia" by Dr. William O. Russell, pathologist, M. D. Anderson Hospital; "Exfoliative Cytology: Its Role in the Diagnosis of Cancer" by Dr. Emmerich von Haam, professor and chairman, Department of Pathology, Ohio State University College of Medicine, Columbus, Ohio; "Recent Advances in Cytological Techniques for Diagnosis of Cancer of the Lung" by Dr. Suk Chul Chang, associate pathologist at Anderson Hospital; and "A Re-evaluation of the Significance of Circulating Cancer Cells in the Peripheral Blood" by Dr. William M. Christopherson, professor and chairman, Department of Pathology, University of Louisville School of Medicine, Louisville, Ky.

The session Friday will specifically apply to the progress in medicine and clinical pathology. Dr. Felix Wroblewski of the Department of Medicine at the Memorial Center for Cancer and Allied Diseases in New York City, will discuss "Enzymes in the Diagnosis of Cancer." Several papers will follow including "An Evaluation of Recently Proposed Diagnostic Tests" by Dr. Douglas H. Sprunt, professor of pathology at The University of Tennessee Medical School, Memphis, Tenn.; "Immunoelectrophoresis and Immunodiffusion Techniques in Diagnosis of Neoplastic Diseases" by Dr. Philip J. Migliore, assistant pathologist; "Clinical Applicaton of Immunological Studies in Multiple Myeloma" by Dr. Daniel E. Bergsagel, associate internist, both of M. D. Anderson Hospital; and "The Influence of Recent Research on Cancer Diagnosis" by Dr. Warren Henry Cole, professor and head of the Department of Surgery at the University of Illinois College of Medicine, Chicago.

Saturday's program will begin with a series of papers on diagnostic radiology including the following: "Lymphangiography" by Dr. Sidney Wallace, instructor in radiology, Jefferson Medical College, Philadelphia; "Mammography" by Dr. Robert L. Egan of Methodist Hospital in Indianapolis; "The Role of Angiography in the Diagnosis of Tumors" by Dr. Mordecai Halpern, assistant professor of radiology at Cornell University Medical College, New York City; "Thermography" by Dr. Jacob Gershon-Cohen, director of the Department of Radiology, the Albert Einstein Medical Center, Philadelphia; and "Remote Control Cineradiography of the Esophagus, Stomach, and Duodenum" by Dr. Gerald D. Dodd, Jr., clinical professor of radiology at Jefferson Medical College, Philadelphia.

The final topic under consideration at the conference will be cancer diagnosis *in futuro*. The first paper will be "Application of Probability Theory to Medical Diagnosis" by Dr. Clyde M. Williams, associate professor of radiology at

the University of Florida College of Medicine, Gainesville, Fla. Following will be "Service of Karyology in Cancer Diagnosis" by T. C. Hsu, Ph.D., biologist, and "Electron Microscopy of Human Leukemia" by Dr. Leon L. Dmochowski, virologist and electron microscopist, both of M. D. Anderson Hospital.

Chest Disease Talks Arranged for Houston

The first annual TB Christmas Seal Symposium on Chest Diseases will be conducted by the Houston-Harris County TB Association at the Warwick Hotel in Houston, Nov. 20, Dr. Howard T. Barkley, medical advisory committee chairman, has announced.

Speakers and subjects for the symposium will be Dr. William F. Miller, University of Texas Southwestern Medical School, Dallas, "Inhalation Therapy and the Management of Pulmonary Insufficiency"; Dr. Robert R. Shaw, University of Texas Southwestern Medical School, Dallas, "Pulmonary Carcinoma and Traumatic Surgery of the Chest"; Dr. Morris Ziff, University of Texas Southwestern Medical School, Dallas, "Pulmonary Fibrosis and Collagen Disease"; Dr. Arthur C. Beall, Jr., and Dr. Denton Cooley, Baylor University College of Medicine, Houston, "Vascular and Embolism Surgery"; Dr. H. William Harris, Woman's Medical College of Pennsylvania, Philadelphia, "Recent Concepts in the Diagnosis and Management of Tuberculosis"; Dr. Robert R. Wright, University of California Medical Center, San Francisco, "Pathology of Pulmonary Emphysema"; Dr. Watts R. Webb, University of Texas Southwestern Medical School, Dallas, "Transplantation of the Lung and Surgery of the Solitary Nodule"; Dr. Roger S. Mitchell, Webb-Waring Institute for Research, Denver, "Pulmonary Emphysema and Chronic Bronchitis"; Dr. John H. Seabury, Louisiana State University, New Orleans, "Diagnosis and Management of Fungus Disease of the Lung"; and Dr. Daniel Jenkins, Baylor University College of Medicine, Houston, "Atypical Tuberculosis."

The Symposium is endorsed by the Harris County Medical Society; Division of Continuing Education, University of Texas Graduate School of Biomedical Sciences at Houston; Baylor University College of Medicine; Texas Tuberculosis Association; Texas Thoracic Society; and the Harris County Academy of General Practice.

Society on Aging To Meet in Austin

The Texas Society on Aging will hold its annual conference Nov. 15 at the Austin Hotel in Austin.

Speakers will include Sen. Walter Richter, chairman of the Governor's Committee on Aging; Dr. Charles Gaitz, Houston State Psychiatric Institute; Mrs. Emily Welty, Texas State Library; Donald P. Kent, Ph.D., Office of Aging, Health, Education and Welfare; and Dr. Francine Jensen of the Houston City Health Department.

Reports will be given about current activities including the Governor's Committee on Aging, Committee on Comprehensive Mental Health Planning, and Rehabilitation Teamwork in Nursing Homes by three local communities. Dr. Kent will discuss poverty and the aging.

Texas Cancer Group To Meet in San Antonio

The American Cancer Society, Texas Division, Inc., will hold its 19th annual meeting Nov. 18-19 at the Granada Hotel in San Antonio.

The annual dinner speaker will be Dr. F. J. L. Blasingame, Executive Vice President of the American Medical Association.

"It says: 'Take two aspirins and go to bed.'"

Recent Deaths

DR. G. E. GRININGER, JR.

Dr. George E. Grininger, Jr., died Aug. 15 in a light plane crash near Beeville. The Houston physician's mother and two of her friends also were killed in the crash.

Dr. Grininger, a general practitioner, was born in St. Paul, Minn., June 1, 1925. He attended

DR. G. E. GRININGER, JR.

school in Wichita Falls, received a B.A. degree from Rice Institute, and earned his M.D. degree from Baylor University College of Medicine in 1950. He interned at Jefferson Davis Hospital in Houston.

A former Rice football player, Dr. Grininger apparently was piloting the plane when the crash occurred. The plane in taking off from the Beeville airport failed to clear an embankment on the other side of a road at the end of the runway.

Dr. Grininger was a member of Saint Michael's Catholic Church in Houston.

He belonged to Harris County Medical Society and the Texas Medical Association.

He is survived by his brother, Dr. Donn Robert Grininger, now in practice in Hawaii.

DR. H. P. SAMMONS

Dr. Howard P. Sammons of Hubbard died at his home Aug. 12 after an extended illness. He was 73.

He was born July 16, 1891, in Hubbard and was the son of J. H. and Sarah Ruth (Middleton) Sammons. He attended school in Albany and went to Stamford College, Stamford, Tex., and Southwestern University, Georgetown, Tex. He received his M.D. degree from the University of Texas Medical Branch in 1919.

Following internship at Scott and White hospital in Temple, he returned to Hubbard to practice. He had lived there ever since.

Dr. Sammons married the former Mabel Kinslow of Appleby, Ark., while she was teaching in the Hubbard schools. They were married on his birthday in 1923.

Dr. Sammons was a charter member of the Hubbard Lions Club, a former member of the school board, a steward in the Methodist church, and city health officer. He was a member of the Texas Medical Association through the Hill County Medical Society.

He is survived by his wife of Hubbard; a daughter, Mrs. William P. Saylor, Tampa, Fla.; a granddaughter, Suzanne Saylor, Tampa, Fla.; a sister, Miss Nell Sammons, Albany; two brothers, Hansel Sammons, Carlsbad, N. M., and Henry Sammons, Portland, Ore.

DR. J. B. LUCAS

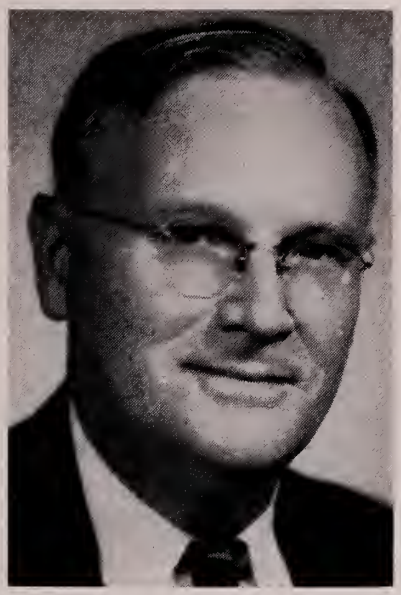
Dr. J. Beeman Lucas, Houston general practitioner, died Aug. 14 of a heart attack. He was 64.

Dr. Lucas was born in Augusta, Tex., Sept. 9, 1899. He attended Sam Houston State Teachers College at Huntsville, the University of Texas, and Texas A & M. He received his M.D. degree in 1926 from the University of Texas Medical Branch at Galveston. He interned at Herman Hospital in Houston.

Before entering private practice, he worked for the Texas State Department of Health.

He was a member of the Harris County Medical Society, Texas Medical Association, and the American Medical Association.

Dr. Lucas is survived by his



DR. J. B. LUCAS

wife, Mrs. Lucille Lucas; two daughters, Mrs. Patricia Hartley of Annapolis, Md., and Miss Jean Lucas of Houston; a son, Charles Lucas of Houston; two sisters, Mrs. Pearl Musick of Houston and Miss Jennie Lucas of Augusta; and three brothers, Sam Lucas of Augusta, Frank Lucas of Rusk, and Clarence Lucas of Galveston.

DR. B. H. REINARZ

Dr. Berthold Henry Reinarz, a resident of Victoria, died there Aug. 6. He was an allergist and

DR. B. H. REINARZ

dermatologist.

He was born June 8, 1903, in New Braunfels, the son of Otto and Anna (Ebert) Reinarz. He attended New Braunfels High School, the University of Texas, and received his M.D. degree from the University of Texas Medical Branch in 1927. Following internship at John Sealy Hospital in Galveston, he served a residency in internal medicine at Scott and White Hospital in Temple. Through the years he took a number of postgraduate courses.

During World War II he served in the U. S. Air Force as a lieutenant colonel in the medical corps from 1942 to 1946.

Dr. Reinarz practiced internal medicine and allergy in San Antonio from 1931 to 1942. He practiced allergy in Austin from 1948 to 1952 and allergy and dermatology there from 1955 to 1960. He had been in Victoria since 1960.

He was a member of the Victoria-Calhoun-Goliad Counties Medical Society, the Texas Medical Association, American Medical Association, American College of Allergists, Southwest Allergy Forum, and Academy-International of Medicine.

He was an Episcopalian.

He married Elaine Francis Weber, June 5, 1928.

During the 1930's, Dr. Reinarz was one of San Antonio's highest average bowlers, winning numerous championships in both ninepins and tenpins.

He is survived by his widow; two sons, Dr. Allen Reinarz, San Antonio, and Ronald Reinarz, Austin; one brother, Richard Reinarz, San Antonio, and a sister, Mrs. Mildred Reeh, New Braunfels.

DR. W. L. SOUTHER

Dr. William Lester Souther of Waco died July 26 in a Temple hospital.

He was born April 25, 1881, to J. H. and Mary (Renick) Souther in Oakland, Ky. He attended school in Bowling Green, Ky., and was graduated with honors from the University of Tennessee Medical School in 1905. His father and grandfather were both physicians.

Dr. Souther first practiced medicine in the Indian Territory with a special permit from President Theodore Roosevelt.

A registered pharmacist as well as physician, he moved to Texaco,

DR. W. L. SOUTHER

N. M., in 1907 where he practiced medicine and operated a drug store. He continued his dual occupation in Waco where he moved in 1910.

A veteran of the Spanish-American War, Dr. Souther was active in the Veterans of Foreign Wars organization. He was surgeon-general of V.F.W. for Texas for four years.

A staff member of Hillcrest and Providence Hospitals in Waco for more than 50 years, Dr. Souther retired from his practice in 1963 following a stroke.

Dr. Souther married the former Ora Vernon of Holland in Waco in 1911.

He was elected to honorary membership in the Texas Medical Association in 1957 through the McLennan County Medical Society.

He is survived by his wife of Waco; a son, William L. Souther, Jr., of Houston; a sister, Mrs. Marcus Dunnam of Alexandria, La.; and a granddaughter.

DR. C. B. SANDERS

Dr. Charles B. Sanders, Houston pathologist, died there of myocardial infarction on July 11 at the age of 64.

Dr. Sanders was born in Orange on Dec. 11, 1899, the son of G. B. and Ida Wolkarte Sanders. He attended the Orange schools and received his bachelors degree at Davidson College in Davidson, N. C., in 1922. In 1926 he graduated in medicine from the University of Texas Medical Branch. He interned at City Hospital, Louisville, Ky., and returned to the Medical Branch in Galveston for postgraduate work from 1927 to 1933. During this time he also taught in the Pathology Department there. In 1937 he was certified by the American Board of Pathology.

He worked as a pathologist in Parkland Hospital, Dallas, from 1933 to 1937 when he moved to Houston and opened a private laboratory which is still in operation. For many years he was a clinical professor of pathology at the University of Texas Dental Branch in Houston.

He was pathologist for Galveston County Memorial Hospital in La Marque and for Wright and St. Elizabeth's hospitals in Houston.

Dr. Sanders was a fellow of the American Society of Clinical Pathologists, College of American Pathologists, and the American College of Physicians. He was a member of the Harris County Medical Society, Texas Medical Association, American Medical Association, Southern Medical Association, Texas Society of Pathologists, Inc., Houston Society of Clinical Pathologists, and Phi Chi fraternity. He was a past president of the Texas Society of Pathologists, Inc.

A collector of Texana, he was a Presbyterian and a member of the Doctor's Club in Houston.

He was the author of some 15 published papers.

He and Mrs. Sanders, the former Elva McDonald of Galveston, were married in Galveston on Nov. 3, 1934. She survives as do a daughter, Mrs. Elva Ida Weatherford; a son, Charles B. Sanders, Jr., all of Houston; two sisters, Mrs. Clara Dau, Abilene, and Mrs. Edna Sureau, La Marque; and three brothers, Jules, Harold, and Arthur Sanders, all of Orange.

DR. L. BARNES

Dr. Livingston Barnes, 92, who practiced medicine in Hubbard from 1914 until 1947, died in a Waco hospital on July 24.

He was born in Irving College, Tenn., July 20, 1872, the son of a Civil War veteran. His father was Dr. William Carroll Barnes, a Confederate surgeon who had to use bandages until they wore out and had to operate without anesthesia. As a boy, he followed his father on his horseback rounds and thus developed a love for medicine. He is said to have started his career by setting broken legs for chickens.

DR. L. BARNES

He entered his father's school, the University of Tennessee, and was the youngest graduate of his medical class, receiving a gold medal for scholastic honors. Because doctors were so badly needed, he began to practice when he was only 19. His first patients were Tennessee miners, but around the turn of the century, he left this practice to come to Texas.

He stopped first at Dawson and in 1914 moved on to Hubbard where he stayed the rest of his life. In the 1930's he was twice elected mayor of the town.

He married Lemma Etter who died March 13, 1954.

Dr. Barnes was awarded the Golden "T" award by the Univercity of Tennessee for 50 years of loyal service to his community.

Dr. Barnes was elected to honorary membership in Texas Medical Association in 1951 through the Hill County Medical Society.

Survivors include two daughters, Mrs. Edna Wilhite of Hubbard and Mrs. Willetta Reaves of Elgin; one grandchild, Mrs. Edward A. Nahkunst; and two great-grandchildren, Michael Nahkunst and Carol Sue Nahkunst, all of Richardson.

COMING MEETINGS

American Medical Association, Clinical Meeting, Miami Beach, Fla., Nov. 29-Dec. 2, 1964. Annual Meeting, New York City, June 20-24, 1965. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 24-26, 1965; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

OCTOBER

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 18-23, 1964. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics, New York, Oct. 24-29, 1964. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Cancer Society, New York City, Oct. 28-30, 1964. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Gastroenterology, New York City, Oct. 18-22, 1964. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition, New York, Oct. 25, 1964. Dr. Robert A. Peterman, 3 Craig Court, Totowa Boro, N. J., Sec.

American College of Physicians, Los Angeles, Oct. 8-10, 1964. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology, Minneapolis, Minn., Sept. 29-Oct. 2, 1964. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Chicago, Oct. 5-9, 1964. Dr. R. S. Myers, 55 East Erie, Chicago, Sec.

American Heart Association, Oct. 20-26, 1964. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Medical Women's Association, New York, Oct. 16-20, 1964. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Public Health Association, Inc., New York City, Oct. 5-9, 1964. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Bal Harbour, Fla., Oct. 10-14, 1964. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Miami, Fla., Oct. 16-24, 1964. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

Association of American Physicians and Surgeons, Inc., New Orleans, La., Oct. 8-10, 1964. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

International College of Surgeons, Mexico City, Oct. 6-11, 1966. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Southern Psychiatric Association, Kansas City, Mo., Oct. 2-4, 1964. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southwestern Medical Association, Las Vegas, Nev., Oct. 22-24, 1964. Dr. Zigmund Kosicki, 1220 North Stanton St., El Paso, Sec.

Texas Academy of General Practice, Corpus Christi, Oct. 12-14, 1964. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Assoeiation for Mental Health, Houston, Oct. 22-23, 1964. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Surgical Society, San Antonio, Oct. 4-6, 1964. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Third District Society, Amarillo, Oct. 3, 1964. Dr. E. K. Jones, 604 W. 8th, Amarillo, Sec.

Oklahoma City Clinical Conference, Oklahoma City, Oct. 26-28, 1964. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

NOVEMBER

Radiological Society of North America, Chicago, Nov. 29-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Texas Dermatological Society, Waco, Nov. 8, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Division, American Cancer Society, San Antonio, Nov. 18-19, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Society on Aging, Austin, Nov. 15, 1964. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas State Board of Examiners in Basic Sciences, Fort Worth, Nov. 13-14, 1964. Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

American Academy of General Practice, San Francisco, April 12-15, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Association of Obstetricians and Gynecologists. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, New York City, June 17-21, 1965. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

Ameriean College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American Congress of Physical Medicine and Rehabilitation, Philadelphia, Pa., Aug. 22-27, 1965. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Boca Raton, Fla., Feb. 25-March 2, 1965. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society, New York, May, 1965. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Hospital Association, San Francisco, Aug. 30-Sept. 2, 1965. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Neurological Association, Atlantic City, N. J., June 14-16, 1965. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group, Chicago, Nov. 6, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Medical Society of United States & Mexico, Phoenix, Ariz., Dec. 9-12, 1964. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson. Ariz., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Southwest Allergy Forum, New Orleans, La., March 14-16, 1965. Dr. W. P. Coleman, 1828 Calhoun St., New Orleans, La., Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. Leonard R. Robbins, 3618 Yoakum, Houston, Sec.

Texas Heart Association. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association. Dr. R. R. Dugan, Continental Oil Co., Box 2197, Houston, Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Pediatric Society. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. Charles Peterson, 1333 Moursund, Houston, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10. Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Anesthesiologists, San Antonio, May 1-2, 1965. Dr. Fred P. Thomas, 2342 Maroneal, Houston, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas Society of Pathologists, Inc., Houston, Jan. 29-31, 1965. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 7-9, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, Sec.

United States-Mexico Border Public Health Association, Los Angeles, April 26-29, 1965. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 9-10, 1965. Dr. J. E. Albright, 2481 Medical Center, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

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La Leche Leaguers Lead Revival of Breast-Feeding

UNTIL RECENTLY the art and practice of breast-feeding seemed virtually to disappear from the American scene. The turning point came in 1956 when a group of young mothers, reclaiming one of the instinctive rights of motherhood—the right to feed a baby at the breast—and desiring to help other mothers master the knack of nursing, organized La Leche League.

"La leche" means "the milk," and is taken from the Spanish title for the Madonna: "Nuestro Señora de la Leche y Buen Parto"—Our Lady of Happy Delivery and Plentiful Milk.

Today, more than 140 active units of La Leche League International provide instruction and personal counseling to any mother or prospective mother who desires to breast-feed her baby. Not only may meetings be attended by physicians, but courses also are offered for medical students, house officers, and student nurses. "The Womanly Art of Breast Feeding,"* and "Nursing Your Baby,"[3] contain a wealth of information on the nature and practice of nursing.

But the task of the mother who wants to breast-feed her baby is not easy. Whispers of doubt that impose underlying negative attitudes, or countless stereotyped reasons why nursing should be rejected or discontinued, prevail at every turn. In a culture of readily available canned formulas and a wide variety of strained foods and supplementary vitamins, infant feeding programs often beyond physiological needs in early weeks of life are stressed as every bit as good, or even better, than breast-feeding.

Clinical and experimental studies, however, raise crucial questions about the wisdom of today's unorthodox feedings.[4] Indeed, contrary to these overfeeding practices, the successfully breast-fed baby seldom requires supplementary food for a period of

*Available from La Leche League International, 9606 Franklin Ave., Franklin Park, Ill. 60131.

six months. Human milk is nutritionally natural for human infants, and it meets all quality standards essential for normal human growth and development. Vitamin C and vitamin D are provided in accordance with maternal consumption. Therefore breast-feeding is to be seriously considered by parents, and to be intelligently fostered by physicians and nurses who want to feed small infants safely, conveniently, and cheaply. For the practical-minded, human milk is ready-mixed and the temperature is always right. For the perpetual nonconformist, no federal packaging regulations are necessary.

In the selection of infant feeding methods, cost (always a matter of sober interest to physicians) is of considerable importance. But convenience and cost aside, human milk is superior to bovine milk. For one thing human milk, especially colostrum, is immunologically useful to babies. It contains antibodies against *Escherichia coli* and staphylococci, not infrequent causes of serious neonatal infections, and provides resistance to enteric infection with poliovirus. Nor should data indicating that most crib deaths occur not in breast-fed but in formula-fed infants be overlooked.

In other ways human milk feedings affirm the everlasting wisdom of nature. Concerning family planning, for instance, the observation that lactation also is an effective baby-spacer should not be forgotten. Natural suppression of ovulation, however, obviously is related to the duration of successful breast feeding.[1] Since the natural instinct of motherhood is to give and to nourish, breast feeding has a significant but often individual relationship to emotional maturity. In this connection, apart from obvious species differences, the long-term results of Harlow's famous experiments with rhesus monkeys isolated at birth and raised on dummy mothers are perhaps pertinent to feeding experiences early in life. After reaching maturity Harlow's experimental subjects were mated and their behavior toward their own young observed. They were uniformly poor mothers. Not only did they neglect their offspring and punish them when they cried, they also failed to develop patterns of normal sexual behavior.[2]

Although human milk is not a solution to all human problems, many mothers nonetheless desire to breast-feed their own babies. Not all mothers are working, nor are all engaged in activities outside the home. Therefore the aims and achievements of La Leche League merit the thoughtful consideration of professional persons concerned with the kind of practices that nurture the physical and mental maturity of children and adults.

—FRED M. TAYLOR, M.D., Houston.

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Keeping Children Healthy

LONGVIEW, and perhaps as many as six other Texas communities, will launch a pilot project this year to help parents supervise the health of their children from birth through age 18.

These efforts are expected to usher in an ambitious and worthy program entitled "Keeping Children Healthy in Texas." Spearheaded by the 800,000-member Texas Congress of Parents and Teachers, the project has been endorsed by the Texas Medical Association and seven other state organizations concerned with children and their health.

Goals of the program are (1) to increase parents' awareness of the importance and value (a) of having regular periodic medical and dental checkups for their children, (b) of carrying out the physician's and dentist's recommendations for treatment or correction of defects, and (c) of having children receive immunizations and boosters against diseases; (2) to encourage the early selection of family physicians and dentists; (3) to encourage parents to keep a permanent personal health record for *each* child in the family; (4) to encourage the use of adequate school health records; (5) to support the improvement of health education programs in the community; (6) to increase parents' knowledge of the community's health resources and facilities; and (7) to provide a suitable questionnaire to assist parents in evaluating the health supervision of their children.

Organizations besides TMA and the Texas Congress of Parents and Teachers which are supporting the project are the Texas State Department of Health, Texas Dental Asso-

ciation, Texas Education Agency, Texas School Nurses Association, Texas Hospital Association, Texas Nurses Association, and Texas Association of School Administrators.

A state advisory committee has been formed including representatives of each of these groups and other consultants. It met several times in Austin during the past year, analyzing material available from national sources and tailoring this program to fit the specific needs of the people of Texas.

This project has involved a great deal of imagination, energy, and enthusiasm on the part of the P-TA, and it is going to mean a lot of "leg-work" for the local parent-teacher groups; but Texas children should benefit from their efforts and our support.

—EDWIN L. RIPPY, M.D., Chairman
Committee on School Health.

Maximum Use of Facilities In a School Health Program

IF ALL CHILDREN were normal, there still would be need for a school health service to help them stay well. Unfortunately, most children sooner or later need health counseling and guidance even if they have no gross handicaps. The complexities of modern living tax the physical and emotional resources of everyone.

Experience in the Health Service of Dallas Independent School District indicates that health problems of school children are in three groups: First are the simple ones the nurses encounter daily and dispose of in a routine manner. Second are physical problems which require constant attention and counseling with both parents and children, but which are finally resolved. Third, and most difficult, are those involving physical handicaps with emotional overlay, and those with emotional involvement that may or may not have secondary physical complications.

In Dallas, we recognized several years ago that our facilities, staff, and procedures had to be constantly improved. Experience has dictated numerous changes in our clinic facilities; nurses must have the best in clinics and equipment to attain maximum efficiency.

Besides being health counselors for the children, school nurses are also medical advisors for their principals and members of the faculty. Of our nurses, 13 percent have master's degrees, 84 percent have bachelor's degrees, and 86 percent have had public health training and experience. All have had one or more courses in education and are qualified health counselors.

School nurses work teachers' hours and also make home calls that cannot be made during school hours. With the exception of principals and home counselors, nurses have more contact with the community than any other school personnel. They are important public relations people whose service cannot be duplicated by nurses from outside agencies.

While our nurses do no formal teaching, they assist health science and homemaking teachers by furnishing supplementary resource materials and by demonstrating nursing procedures.

The health service is only one of several special services in our organization. As far as our work is concerned, the most important of these are special education, the research-psychologic service, home counselors, and the counseling and guidance service. To coordinate the work of these agencies, pupil-personnel committees have been organized under the leadership of the research-psychologic service. These committees, in elementary schools, are composed of the principal, assistant principal, home counselor, nurse, speech and hearing therapist, and selected members of the faculty. In junior and senior high schools the school counselors are included, and other personnel may be added.

Specific purposes and functions of these committees are:

1. To collect and evaluate data concerning individuals or groups of children whose conflicts and deficiencies in school life indicate need for special consideration and help.
2. To combine efforts of teachers and special service personnel within the school to help students make adjustments and improvements in accordance with individual potentialities.
3. To effect the proper referral when services beyond the building group are needed.
4. To follow up on recommendations of education-oriented personnel in the administration offices.
5. To agree upon the proper person on the committee to confer with parents when their participation is needed.

All available information is presented at the first meeting, but only in rare instances

will one discussion suffice. Further investigation may include several psychologic tests, physical examination, special reports from committee members, and reports from community agencies outside the school. A second meeting is held then, and definite conclusions are reached. The principal then appoints a member of the committee to confer with the parent.

In the pupil-personnel committees we have an effective machinery for dealing with physical and emotional problems of school children. As long as the pupil remains in school, he gets sympathetic help. Should he become a "drop-out" before the case is satisfactorily concluded, information gathered by the committee is used by the home counselors in a continuing effort to get the pupil back in school.

—GEORGE A. GRAY, M.D., Dallas.

The Forgotten Ankle

TREATMENT of ankle sprains over the past 22 years has not improved greatly in my experience. As a practicing orthopedic surgeon I have seen many so-called "severe sprains of the ankle" in which the convalescence and rapidity of recovery as well as the completeness of recovery could have been expedited much more rapidly had the true condition been recognized at the time of the original injury. While stationed at Brooke General Hospital in 1946 I was proud of a series in which only 3 of 85 consecutive patients with ankle sprains required hospitalization, all having been treated with ethyl chloride spray. In retrospect I wonder how many more of those would have been treated more radically and with less final disability had I taken the proper x-rays.

It may be thought that the rupture of the lateral ligaments of the ankle or the deltoid ligament on the medial aspect of the ankle is a relatively rare occurrence. I do not believe this to be true.

Usually the doctor is called to the hospital or sees in the office the patient with a history of the ankle having been twisted in a fall. Routine x-rays are made of the ankle, including anteroposterior, lateral, and oblique views. The x-rays are rightfully interpreted as being negative. Clinical evaluation of the patient, however, reveals objective findings and pain far out of proportion with the neg-

Fig. 1. Inversion stress.

Fig. 2. Eversion stress.

ative x-ray findings. Massive hematoma as a localized finding is seen far more frequently beneath the lateral malleolus than beneath the medial malleolus. This is because the injury apparently occurs more frequently on the lateral aspect than on the medial aspect. However, massive hematoma can occur on the medial aspect as well. This is certainly an indication to take the proper stress x-rays to determine the true state of the ligamentous structures about the ankle. It has become my practice when clinical evaluation reveals the sprain to be out of proportion with the x-ray findings to take the routine x-ray views and, in addition, to take the stress x-rays. It should be noted that this is frequently a painful condition and anesthesia may be obligatory in order to secure proper films without the spasm which can mask a ruptured ligament. These are made with the ankle held slightly internally rotated with one examining hand holding the tibia firmly and the other hand producing sharp inversion of the forefoot and calcaneus (Fig. 1). This determines the status of the lateral ligaments of the ankle. The procedure is reversed for eversion of the forefoot and calcaneus to determine the status of the deltoid ligament on the medial aspect of the ankle (Fig. 2). Far more frequently found than rupture of the medial structure is rupture of the lateral ligaments of the ankle. Interpretation of the x-rays is relatively simple by looking at the position of the talus in the ankle mortise. If the talus widens on the lateral aspect in relation to the tibia one can suspect rupture of the lateral ligaments. If it widens on the medial aspect, one should expect damage to the medial ligaments of the ankle. It is frequently a wise idea to obtain similar stress films on the intact ankle for comparative purposes against the involved ankle.

I believe that a tilt of no more than 10 to 15 degrees of the talus in the mortise away from the tibia is an indication for conservative treatment consisting of applying a cast for 3 to 4 weeks to allow time for the ankle ligaments to heal. If the tilting is more than the prescribed limit surgical repair must be considered.

Early repair of the ruptured ligaments of the ankle can be easy or can be exceedingly difficult. Frequently reconstructive procedures must be considered, particularly of the lateral structures by substitution with the peroneus brevis tendon in prescribed techniques. Aftercare is routine immobilization for about three weeks followed by intensive physiotherapy for rehabilitation.

Many so-called chronic sprained ankles can be prevented or rehabilitated by simple early investigation with proper treatment.

—WALTER W. GLASS, JR., M.D., Port Arthur.

personal opinions and views

Protective Law Needed For Battered Child Reports

UNFORTUNATELY the physician in Texas who treats an abused child is faced with this dilemma: He can report the trauma to the authorities and risk a damage suit against him by the parents, or he can overlook the probable cause of injury and thus avoid legal complications—possibly at the cost of the child's life.

Two things can be done to protect both the child and the physician. First, the courts, police, and child welfare agencies must be made cognizant of the reliability of radiological evidence in "battered child" cases. Second, legislation similar to that suggested by the Children's Bureau, U. S. Department of Health, Education, and Welfare should be enacted, making the report of abused children mandatory and also giving the person making the report immunity from legal actions.

A few states have adopted such laws; Ohio has reported that its law is successful, and other states have had their laws in force for too short a time to give fair evaluation. Success or failure of such legislation depends on its acceptance by the medical profession. The law is only valuable if physicians abide by it, thus this type of legislation could be a success in one state and a failure in another.

The argument has been made that protective legislation might keep parents from seeking medical care for abused children for fear of being reported to the police. Statistics are meager, but the Children's Division of the American Humane Association re-

ports that one of every four abused children dies from his injuries. Those who survive do so only to be mistreated again and again until someone takes steps to prevent it. It is the subsequent abuse which legislation is aimed to curb.

Under the present Texas statutes, the physician, if he chooses to take legal action for the child's protection, may institute proceedings himself to have the child declared "dependent" or "neglected," in which case he must file a petition to that effect with the district clerk of the county in which he resides and the child is located. However, this petition would be based solely on the physician's opinion and should he be unable to prove his case, the risk of liability would be increased. The physician may also contact a child welfare agency in his county, but the agency may file the petition on the doctor's word alone, again increasing the physician's risk.

For his own protection, the most desirable action the physician can take is to call the police, who will investigate the matter and, if need be, file a petition based upon their investigation. Also, if the child is in the custody of the parents, the police have authority to cope with the situation. When the evidence is purely radiological, the police may choose not to file a petition, thus leaving the child unprotected. However, the physician, at least in part, has decreased the possibility of a damage suit.

Usually the physician is required to furnish a written report containing the facts upon which he bases his opinion. He can again minimize his risk if he will remember one simple rule: *Never accuse.* He must report only what he or his associates have observed; never should he include accusations or personal suspicions. The report must be completely objective. A suit to have a child declared dependent and neglected is not a criminal one. The district or county attorney must be requested to act, either by the court or by the petitioner. His role is merely to collect evidence and present the case for the petitioner in the interest of the child.

The timeliness of reporting cannot be emphasized too much. If the child is in a hospital, a petition should be filed before the child is released. This will give the hospital the right to keep the child until a hearing takes place, thus avoiding the possibility of further abuse. If the physician treats the child in his office, or cannot otherwise hold the victim until the petition is filed, speed is imperative to insure that the child will immediately be under the court's supervision.

The battered child syndrome is not self-limiting. If not curbed, the abuse will often continue until the child is crippled or killed. Unfortunately there is no legislation to protect the physician who wants to report the situation. Nevertheless, until such laws are passed, the doctor has at least a moral obligation to report child abuse, and in the medical profession, which is dedicated to life and health, it is his professional responsibility.

—P. Larry Pascoe, LL.B., and
Carl C. Peterson, Jr., M.D., Dallas.

Physicians Commended for Support of Amendment 3

TEXAS PHYSICIANS and Auxiliary members are to be commended for their diligent work in support of Amendment 3, which was passed Nov. 3.

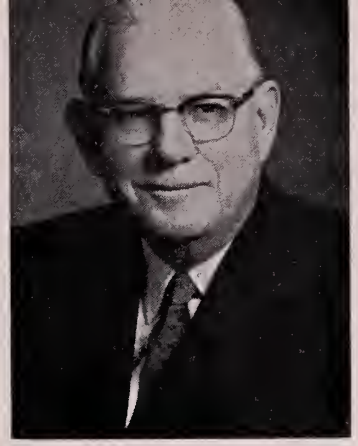
In addition to the public service of informing the voters on this issue, TMA has had the opportunity once again to be on record as favoring a positive and effective program to provide health care benefits under the Kerr-Mills Law for needy aged persons who do not receive Old Age Assistance.

The public relations effort included news releases explaining the proposal and urged its support; they were widely used by news media. Also, letters were sent to all members of the Association and the Auxiliary, and an enclosed leaflet was offered for distribution through office waiting rooms, hospitals, civic clubs, or the mails.

The flood of 1,165 requests for copies of this leaflet topped the demand for any other brochure ever produced by TMA. Four additional printings and two extra employees were required to fill these orders; all orders were mailed within 24 hours. A total of 279,028 copies was distributed.

The overwhelming vote in favor of Amendment 3 is, in itself, a fitting recognition of the medical profession's active support of worthwhile legislation to provide medical care for aged persons who need and want help.

—Byron P. York, M.D., Houston.



Extending Benefits Of Kerr-Mills Law

We in the United States enjoy the best medical care the world has ever known, and our nation has become the medical center of the world. Our people want and expect medical help as a part of a more abundant life.

Texas leads all other states in care of the aged. It truthfully can be said that most of the Texas aged who actually need and want medical help are receiving it. Surveys reveal that only 3 percent of all elderly Texans are not receiving the medical help they need or want.

The adoption of a Constitutional amendment by voters this month makes the Texas Legislature responsible for broadening the existing Kerr-Mills program to provide health benefits for those who do not qualify for Old Age Assistance, but who need and want medical help.

The Legislature also should give consideration to extending benefits under Kerr-Mills to provide financial assistance in catastrophic illnesses. Many individuals who qualify for coverage under Kerr-Mills have adequate funds to meet routine expenses, but have difficulty in financing care in major illnesses because of their modest income and reserves.

Another problem concerning the Legislature will be to establish income limits as qualification for eligibility.

The members of the Texas Medical Association and of the county medical societies should assume the lead in supporting the extension of the Kerr-Mills program to include these benefits. Active support and cooperation to make these benefits available to those who need them will be added assurance to the public that we, the physicians of Texas, are truly concerned and interested in medical care for the aged. This positive approach to such a worthwhile program also will provide another excellent opportunity to tell the Texas story of medical care for the aged.

Mat E. Johnson

President
Texas Medical Association

Immunization Against Measles

REVIEW ARTICLES

THE LICENSING of measles vaccine on March 21, 1963, was a significant event in the history of preventive medicine. It represented the culmination of nine years of outstanding contributions by many groups, beginning with the isolation of measles virus in tissue culture by Enders and Peebles in 1954 and the attenuation of the virus by Enders, Katz and Milovanavic in 1958. Subsequently, extensive field trials established the safety, potency, and efficacy of measles vaccine.

Two types of measles vaccine are available for use—a live attenuated vaccine prepared in chick embryo tissue culture and a formalin inactivated (killed) vaccine prepared in chick embryo or monkey kidney tissue culture.

Live Attenuated Measles Virus Vaccine.—A single inoculation of live vaccine, Edmonston B type, is followed by an antibody response in more than 97 percent of susceptible infants and children over nine months of age. The presence of passive antibody in younger infants may suppress the antibody response. A high incidence of fever and rash observed after injection of Edmonston B vaccine has been reduced significantly by the simultaneous inoculation of gamma globulin, 0.01 ml. per lb., at a separate site. Experience based on more than 5 million inoculations of live vaccine plus gamma globulin has confirmed the safety and efficacy of the vaccine.

A further attenuated vaccine has been developed by Schwarz. The clinical and immunogenic effects of further attenuated measles vaccine without gamma globulin are essentially the same as those observed following Edmonston B type measles vaccine plus gamma globulin. These live vaccines have the following advantages: (1) only one inoculation is required for optimum immunization; (2) no local reactions occur at the site of inoculation; (3) absence of complications such as otitis media, pneumonia, and encephalitis; (4) absence of communicability to susceptible contacts; (5) rapid immunogenic effect; and (6) solid protective capacity. The pattern and persistence of antibody response following immunization with live measles vaccine is the same as that following natural measles infection.

Inactivated Measles Vaccine.—Systemic and local reactions are rare. Three inoculations of killed measles vaccine at monthly intervals will induce an antibody response in 90 percent of susceptible infants and children over nine months of age. In infants under three months, serologic conversion is induced in less than 5 percent following three doses of inactivated vaccine given at monthly intervals. Antibody titers following inactivated vaccine are lower than those following live vaccine; in most cases they decline to undetectable levels one year or more after the last inoculation. Under conditions of natural exposure one year after vaccination, the efficacy of killed measles vaccine was 75 percent as compared with 97 percent efficacy of live measles vaccine.

Combined Inactivated and Live Measles Vaccine.—Administration of live attenuated measles vaccine one to three months after two monthly inoculations of killed vaccine induced a significant antibody response (more than 95 percent) and minimal clinical reaction in infants and children over nine months of age. The protective capacity of this combination was approximately 97 percent one year after vaccination.

Inoculation of killed measles vaccine along with diphtheria-pertussis-tetanus toxoid at age two, three, and four months did not induce a significant measles antibody response (less than 5 percent). However, subsequent administration of live Edmonston B vaccine without gamma globulin at age 12 to 14 months was followed by no significant clin-

Reviewed are the two types of measles vaccines available for use—a live vaccine and a formalin inactivated vaccine. In addition, recommendations are made for administration of vaccines.

SAUL KRUGMAN, M.D.

Dr. Saul Krugman is professor and chairman of the Department of Pediatrics at New York University School of Medicine. He originally discussed this topic before the Section on General Practice during the April, 1964, Texas Medical Association Annual Session in Houston.

ical reaction and an antibody response in 99 percent of the infants.

Recommendations for Measles Immunization.—Evaluation of the virtues and limitations of the various measles immunization regimens reveals a clear-cut superiority of live attenuated measles virus vaccine for normal infants over nine months. Until a further attenuated vaccine becomes available, it is recommended that gamma globulin be given with the Edmonston B vaccine. In view of its rapid immunogenic and protective effect after a single inoculation, live vaccine is especially indicated for susceptible infants and children who are immunized at a time when measles is either endemic or epidemic in the community.

The use of killed vaccine alone is recommended for children with leukemia, lymphomas or other malignancies. The inactivated measles vaccine prepared in monkey kidney tissue culture would be indicated for children with extreme sensitivity to egg.

When killed measles vaccine is incorporated with diphtheria-pertussis-tetanus toxoid, it will be a useful preparation for primary immunization during the first six months of life. An inoculation of live attenuated measles vaccine, Edmonston B type, without gamma globulin would be indicated at 12 to 14 months of age.

Dr. Krugman, New York University School of Medicine, New York.

COMMENTS

The high incidence of measles during the first half of 1964 indicates that the vaccine has probably not been administered as vigorously as it should have been during the past year. This may be due to a persistent attitude that measles is an inevitable, "mild" childhood disease or to some anxiety about reactions similar to those known to occur with other virus vaccines. Since the incidence of allergy to egg is relatively high among allergic children, I believe it is worthwhile to point out that the live attenuated measles vaccine was given to nine children with severe allergy to egg, and no unusual reactions occurred. Since the original report appeared,[1] the investigators administered the same vaccine to six additional egg-allergic children without allergic reactions. Furthermore, several hundred allergists were asked to report allergic reactions or other undesirable effects following administration of the live attenuated measles vaccine; to my knowledge, none have been reported. This should encourage a more vigorous campaign by practicing physicians to include measles immunization among their routine procedures. Failure to do this, it seems, leaves their susceptible patients vulnerable to a disease of unpredictable dangers.

The highest acclaims are deserved by Dr. Krugman and his colleagues for their participation in the development of this vaccine. It is an accomplishment in preventive medicine in this decade second only to the successful marketing of poliomyelitis vaccine.

—PETER B. KAMIN, M.D., San Antonio.

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Dr. Krugman, Department of Pediatrics, New York University School of Medicine, New York City.

Respiratory Emergencies in the Newborn

REVIEW ARTICLES

RESPIRATORY EMERGENCIES in the newborn are frightening and dangerous, and usually leave little time for delay in diagnosis and treatment. Some 5 percent of neonatal respiratory deaths may be due to surgically correctable thoracic lesions, and even more are due to lesions that could be corrected by appropriate mechanical and medical therapy.

Many aspects of normal respiratory physiology peculiar to this age group limit physiologic reserve or increase vulnerability to any abnormality.[6] Cartilage and ribs of the newborn are weak and flexible, leading to inefficiency, especially since respiration is primarily diaphragmatic. The trachea and bronchi are small and of soft cartilage so they are easily blocked from within or without, and the cough mechanism is extremely poor. The mediastinum is labile, and pneumothorax or effusion may result in shift and compression of the opposite lung. The respiratory rate is rapid with a tidal volume of only 15 to 30 cc., and the infant increases the rate rather than the depth of respiration. In addition, the lung alveoli often are not fully developed, so the infant has barely enough lung tissue to maintain adequate transfer of oxygen and removal of carbon dioxide, and the slightest disturbance may prove disastrous.

Clinical Evaluation.—The cardinal signs are cyanosis, dyspnea, and tachypnea in all degrees and combinations. Central respiratory depression producing apnea may be due to narcotic depression, severe asphyxia due to prenatal factors, blood loss, intracerebral damage from hemorrhage, or a cerebral anomaly. The Apgar index gives a useful guide to the severity of the infant's condition.[1] Infants who are cyanotic without dyspnea usually prove to have a brain injury or a complex congenital heart anomaly, though cardiac defects may produce dyspnea as well. Some cardiac defects such as a large patent ductus or ventricular septal defect initially may produce severe dyspnea without cyanosis.

Laryngeal obstruction is usually characterized by an inspiratory stridor and an abnormal cry or absence of crying.[4] With obstruction below the larynx, tachypnea and retraction are present but the cry is normal.

The physical examination is of extreme importance in lesions of the upper respiratory tract, but is of less value in the chest because of the widespread transmission of breath sounds. The infant in respiratory distress should have immediate upright films in at least the posterior-anterior and lateral views. Due to the rapid respiration this will require high milliamperage and minimal time exposure.[2]

Resuscitation.—Resuscitation has as its aim the immediate restoration of a patent airway, reexpansion of the lung, and the maintenance of adequate ventilation and oxygenation.

In the absence of equipment, mouth-to-mouth ventilation is effective, but care must be used not to inflate the stomach or to overdistend the lung. Preferably, an emergency kit for intubation and aspiration should be kept available at all times, and the physician concerned with infant care should master the technique of laryngoscopy with quick, gentle tracheal intubation. Use of the intratracheal airway and aspiration is lifesaving and can often eliminate or at least delay the need for tracheostomy.

The need for tracheostomy should be anticipated rather than resorted to as a terminal event. It should be done in the operating room where one is adequately prepared with trained assistants, lighting, and resuscitative equipment. Tracheostomy provides not the end but only the beginning of the need for diligence on the part of the attending and nursing staff. Only small tracheostomy tubes can be used, and these are extremely susceptible to occlusion by secretions unless adequate humidification is maintained and mucolytic agents such as

There are many causes of respiratory distress in the newborn. Because infants have limited reserves, it is important to make diagnoses and plans for treatment both quickly and carefully. Corrective measures for various lesions are outlined in this article.

WATTS R. WEBB, M.D.

acetylcysteine are utilized frequently, sometimes as often as every few minutes.[12] Gentle, careful suctioning is performed with a very soft catheter. With such care the inner cannula may be left out of the tracheostomy tube to more than double the lumen of the airway.

Technical aspects of the tracheostomy require that it be placed below the larynx but where the trachea is still superficial, such as at the second or third tracheal ring. Similarly, one should incise the tracheal rings and not remove a button lest subsequent healing produce tracheostenosis.

A pediatrics recovery room should be available with trained personnel who can recognize and correct respiratory obstruction quickly. One most important factor in reducing mortality is feeding weak newborn infants with a dropper, which eliminates the danger of aspiration associated with nipple feeding. This alone has reduced greatly the postoperative mortality in tracheo-esophageal fistula, intestinal obstruction, and other operative procedures in premature infants.

Lesions that Obstruct Upper Respiratory Tract

Some of the more common lesions in the region of the nasal or oropharynx are macroglossia, micrognathia, choanol atresia, and tumors such as teratomas, cystic hygroma or congenital goiter.[4] Congenital laryngostenosis is fortunately rare, but also frequently overlooked.[8] Infants with higher obstruction usually have loud, rough respirations indicating air movement in and out of the lungs, but in laryngostenosis there is significant impairment of air movement. Emergency therapy is the provision of an unobstructed airway, after which definitive surgery can be performed electively.

Tracheobronchial Lesions

These include both intrinsic disease of the tracheobronchial tree and extrinsic lesions which can embarrass respiration by compression.

Tracheo-Esophageal Fistulae.—The most common lesion is congenital atresia of the esophagus with or without a tracheo-esophageal fistula. In about 85 percent there is a blind upper pouch of the esophagus with a fistula from the lower esophageal segment to the tracheal bifurcation. Gastric reflux through the lower esophageal segment into the trachea causes the serious respiratory difficulty because children with complete atresia and no fistula (about 10 percent) have much less respiratory difficulty. Air in the stomach indicates a fistulous connection with the trachea or the rare tracheo-esophageal fistula without atresia ("H" fistula). The infant with a tracheo-esophageal fistula shows excessive salivation, coughing, and gasping on attempting to swallow. About one-third of the patients will be premature or have associated polyhydramnios, and about one-third will have other anomalies. Diagnosis can be established by passing gently a naso-esophageal catheter and noting the block a short distance beyond the pharynx. Often it is unnecessary to introduce radiopaque medium but if necessary, it should be a minimal amount of material such as Gastrografin or Dionosil.

Management consists of operation as soon as the patient can be rehydrated and brought into optimal condition, since the prognosis is directly proportional to the delay in diagnosis and operation.[7] If there is increasing abdominal distension, a gastrostomy may be performed under local anesthesia as the initial procedure to allay distension and minimize gastric reflux into the lungs. This also will prevent abdominal distension produced by the positive pressure breathing required during operation. The tracheo-esophageal fistula is divided and oversewn and esophageal segments are approximated with a double layer of very fine sutures.

If an immediate reconstruction is not pos-

sible, the upper blind pouch is exteriorized in the neck and gastrostomy performed for feeding purposes. When the child is approximately two years of age, the esophagus may be constructed by a colonic interposition.

Vascular Rings.—Vascular rings may be formed from many combinations of double aortic arches or from anomalous brachiocephalic vessels and the ductus or ligamentum arteriosum. These produce a localized obstruction of the trachea and esophagus to cause dyspnea or dysphagia, or both. Diagnosis is usually made by an esophagogram which shows a localized "cookie bite" deformity. Operation consists of division of the ring at whatever point will maintain optimal circulation and yet release pressure on the trachea and esophagus. This condition is frequently associated with tracheomalacia, allowing collapse unless the trachea is splinted by a tracheostomy.

Mediastinal Tumors.—Mediastinal tumors rarely cause respiratory obstruction in the newborn, but may during the first year or two, particularly when the masses are firm and rapidly growing. The thymus gland almost never gives rise to obstruction, but teratomas arising therein may be large and symptomatic. Lymphomas may become large, usually involving the central hilar nodes bilaterally to cause both bronchial and pulmonary compression. The most frequent mediastinal tumor in infants, however, is a neuroblastoma, situated posteriorly, which may have dumbbell extension through the vertebral foramen with bone erosion and neurologic signs. The treatment is excision and postoperative irradiation, but the prognosis is still poor. Neurofibromas and ganglioneuromas are benign tumors which usually can be totally excised.

Foreign Bodies.—Foreign bodies are rare in the newborn, but become common after the age of six months. Asthma is relatively uncommon and the most common cause of wheezing obstruction in infancy is secretions associated with infections or aspiration.

Croup.—Tracheolaryngobronchitis or "croup" is an acute inflammatory reaction involving the major airways, and most commonly occurs in the age group of six months to three years. It may rapidly progress to hoarseness, laryngeal stridor, and severe obstruction with dyspnea, cyanosis, and even death. The child is immediately placed in a croupette with an enriched oxygen content and high humidity. Appropriate antibiotics are given to combat secondary infection and minimal sedation with barbiturates may be used to provide rest. Constant observation is essential and the few patients who do not respond to treatment should have a tracheostomy. This should be carried out early rather than as a terminal measure, since the infant may die from exhaustion and chronic hypoxia.

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Extrapulmonary Lesions That Compress the Lungs

Diaphragmatic Hernia.—Perhaps the most common congenital lesion causing extrapulmonary compression is a diaphragmatic hernia which requires surgical correction as promptly as possible, since the big hernia in the small infant causes a high mortality rate.[11] It should be suspected in the infant who, after a relatively normal early period, develops progressive respiratory distress or apnea. The diagnosis can be confirmed by X-ray with the conflicting lesion being that of multiple cysts in the lung, which similarly require immediate operation. Most diaphragmatic hernias occur posterolaterally on the left through the foramen of Bochdalek, and can be handled easiest by an abdominal approach. Air is allowed to enter the hernial sac and the viscera are delivered into the abdomen starting with the stomach, the small intestines, and then the large intestine. On the right side, due to the presence of the herniated liver, the repair is easier through a low thoracic incision. It may be necessary to aspirate the stomach, or even the intestine, with multiple enterotomies to alleviate distension and aid in reduction. If there is not enough room in the abdominal cavity to accommodate all organs, the skin alone may be closed, with repair of the fascial defect

some weeks or months later. Frequently the lung will be hypoplastic, and great care should be taken in attempting to inflate the lung, as excessive pressure can cause a pneumothrax, even on the contralateral side. These lungs will grow, and at the end of weeks or months the X-ray will usually appear normal.

Eventration of the diaphragm implies an abnormally high and relaxed but intact diaphragm which rises to encroach upon the breathing space. Treatment consists in surgical plication of the diaphragm by the thoracic approach.

Pneumothorax.—Pneumothorax may occur in the immediate newborn period either spontaneously or as a result of vigorous resuscitative efforts. Diagnosis can be made by diminished breath sounds, tympany, and mediastinal shift. The safest method of therapy is introduction of a small intercostal catheter attached to water seal drainage with suction. At times it may be difficult to differentiate a pneumothorax from a large pulmonary cyst, which would require immediate excision. If the air leak cannot be controlled by catheters, an immediate thoracotomy may be required for repair of a ruptured trachea or bronchus, or even a congenitally open bronchus.

Most commonly, pneumothorax occurs in staphylococcal pneumonia from rupture of a small peripheral abcess with an open bronchopleural fistula. This may lead rapidly to a tension pneumothorax and death. The demonstration of a small effusion or pneumothorax necessitates immediate closed drainage.

Mediastinal Emphysema.—Mediastinal emphysema is uncommon and of itself is of little significance since sufficient pressure cannot develop within the mediastinum to produce compression of either the airway or the great veins.[13] The air ruptures into the pleural space, causing a tension pneumothorax to add further to the difficulties, or dissects into the neck or retroperitoneal spaces, where it is of little significance. Mediastinal emphysema is often secondary to interstitial emphysema, and it is the peripheral air lock around the alveoli and tiny bronchioles which is responsible for the neck vein distension, cyanosis, and dyspnea.

Intrapulmonary Compression

The usual intrapulmonary compressive lesions are congenital pulmonary cysts, enterogenous cysts, pneumatoceles, and lobar emphysema.

The signs and symptoms shown by infants with any of these are similar. The onset may be at birth with rapidly progressing tachypnea and breathlessness accentuated by feeding. More commonly the symptoms develop gradually, though a slowly progressive lesion can precipitously become a desperate emergency.

Each of these conditions entails progressive loss of functioning lung tissue by mechanical pressure on both the parenchyma and bronchi and even on the contralateral lung, as the mediastinum is shifted and the diaphragm is depressed.

Lifesaving therapy may require immediate decompression by thoracentesis or insertion of an intercostal catheter attached to water seal drainage and suction. This latter has found favor particularly in the enlarging pneumatoceles of staphylococcal pneumonia in which the pleural space is sealed by the accompanying emphysema. Surgical removal of a congenital cyst is simple, and is recommended upon demonstration to avoid the major complications of continued expansion or infection.

Lobar emphysema is usually confined to one upper lobe but can involve only a single segment. A valvular mechanism is accepted with various etiologic explanations such as bronchial folds, mucosal folds, deficiency or weakness of the bronchial cartilage or persistent patent ductus, though most are idiopathic. Differentiation from a cyst may be difficult, but the symptoms are usually less acute and X-rays may demonstrate faint lung markings throughout.

Parenchymal Lesions

Aspiration.—Aspiration is followed by atelectasis which may be either segmental, lobar, or massive of an entire lung. Treatment here is directed at immediate removal of the obstructing agent, usually mucus, by intratracheal suctioning or bronchoscopy. We have found extremely rewarding the insertion of a small percutaneous intratracheal plastic catheter and frequent injections of acetylcysteine to liquefy these plugs.

Respiratory Distress Syndrome.—The respiratory distress syndrome or hyaline membrane disease occurs primarily in premature infants, children of diabetic parents, or children delivered by caesarean section. The symptoms usually progress during the first six hours of life and plateau during the first 24 hours, after which the signs gradually disappear. This is associated with neurologic depression, pitting edema of the hands and feet, fine, moist rales, chest retraction, respiratory grunting, and extremely poor air entry. The etiology is not clear but it is thought to be associated with high histamine levels and the rapid clamping of the umbilical cord so that the full systemic pressure is transmitted directly to the unexpanded pulmonary vasculature through the patent ductus to produce capillary damage.[9] In hyaline membrane disease there is a deficiency of surfactant which has the ability to reduce surface tension of fluids within the alveoli. In its absence, a high surface tension tends to collapse the alveoli or to prevent their expansion immediately after birth. In fact, surface tension is the greatest single factor in the elastic retraction of the lung during expiration.

The chest X-ray shows a uniform fine granular opacification with a wide superior mediastinum. The abdomen frequently is distended and bowel sounds and function are diminished. A late systolic or continuous murmur of a patent ductus may be heard for several days to make the differential diagnosis difficult.

These patients have not only severe respiratory acidosis but also metabolic acidosis due to hypoxia.[10] There is an adrenocortical hyperactivity with evidence of rapid tissue destruction, weight loss, and a rise in blood levels of BUN and phosphorus. The serum proteins are low and capillary permeability is increased.

Therapy requires adequate humidity and the use of whatever concentration of oxygen is required to relieve cyanosis, though this is lowered to 40 percent as soon as improvement is evident. Some prefer to correct the metabolic acidosis with sodium bicarbonate and to give a constant infusion of glucose to provide calories and to reduce the level of circulating potassium. Perhaps the most important thing is to provide heat; otherwise, the child's metabolism and oxygen demands will rise in the attempt to maintain a normal temperature.

Ninety percent of the infants who are alive at 72 hours will survive, but muscle tone and activity improve only slowly.

Cardiac Lesions.—Congenital heart disease is beyond the scope of this paper but may be difficult to distinguish from primary respiratory disease in the newborn. The heart murmurs and electrocardiographic abnormalities remain the clinical mainstays of differential diagnosis. Accurate diagnosis is essential and many require cardiac catheterization and angiocardiography to distinguish the operable lesions.

Summary

Many lesions of the upper respiratory tract, the mediastinum, lung, diaphragm, heart, and great vessels may produce acute respiratory distress. In most instances, a careful history and physical examination and well-planned roentgenograms will lead to the diagnosis. Many of these lesions are correctable with prompt, well-planned surgery. Due to the limited physiologic reserve of infants, careful pre- and postoperative care often proves an equally important factor in success or failure.

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Pyrogenic Reaction Following Tonsillo-adenoidectomy

BEN T. WITHERS, M.D., and BEN M. NAIL, JR., M.D.

RESEARCH & INVESTIGATION

IN AN EARLIER study,[8] a method was presented whereby post tonsillo-adenoidectomy (T & A) hemorrhage was decreased from an initial 10 percent to 0.6 percent of cases. Other aspects of management were also considered, but no attention was given to the occasional febrile reaction following such surgery. The present report concerns the febrile or pyrogenic reaction studied in 450 T & A cases.

Long of interest to us has been why 16 percent of postoperative T & A patients spike temperatures from 102 F. to 105 F. during the first 24 hours following surgery. Several theories were advanced. First questioned was the possibility that microorganisms freed into the blood stream by dissection of these diseased organs could account for the fever; for example, a bacteremia or a viremia. Second, dehydration factors were considered. Questioned third was the possibility that the difference in febrile reaction was related to the individual variations in the temperature regulating mechanism. And last asked was whether fever was attributable merely to foreign protein or "alarm" reaction to the surgery, or even to changes in body physiology due to the anesthetic. Anesthetic agents reduce metabolic activity, hence less heat is produced; temperature will then go down or up depending on room temperature. The last category was discarded as an unlikely answer since the response in each individual should be approximately the same to the stimulus of T & A surgery and anesthesia. In other words, patients should all run a high fever or all should have no fever.

In considering the first category, with reference to pyrogenic reaction, the possibilities of microorganisms showering into the blood stream, bacteremia, and viremia were considered. First, white blood count was determined for 45 patients before surgery and after surgery within the first 24 hours postoperatively. All patients showed an increase in white blood cell count including 38 with temperature elevation of less than 102 F. and 7 with temperature of 102 F. or more. This was not due to hemoconcentration because the differential count percentage of polymorphonuclear cells also increased while the lymphocyte differential decreased in percentage. Acting on the assumption that a bacteremia might exist, blood cultures for bacteria were drawn from five patients with temperatures of 102 F. or over when the fever was high. These cultures were all negative for bacterial growth. We thought, therefore, that the bacteremia immediately postoperatively did not account for the high fever. No obvious explanation was found, however, for the leukocytosis noted in all cases.

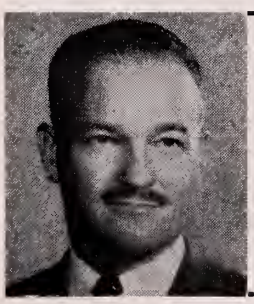
The subject of possible viremia or showering of viral bodies into the circulating blood is interesting. Apparently there is a high percentage of excised tonsils and adenoids which contain pathogenic virus. Rowe[5] has stated of the adenoidal, pharyngeal, or conjunctival (APC) virus or adenovirus that "Types 1, 2, and 5 have been found most frequently in association with febrile respiratory infections in young children and are unmasked in tissue cultures of adenoids and tonsils of most children undergoing tonsillectomy-adenoidectomy." He stated further[6] that "The persistence of Adenovirus is well known. When surgically extracted adenoids

To determine why 16 percent of their post tonsillo-adenoidectomy patients spiked temperatures of 102 F. to 105 F., the authors studied 450 patients.

are explanted in culture, as many as 90% may yield Adenovirus." This is, of course, considerably higher than our 16 percent who spiked higher fever. Melnick[4] has stated that there are at least 40 strains of adenovirus which already have been isolated.

Because of the expense entailed and other technical difficulties, only two cases were chosen for study of the viremia by tissue culture. One patient spiked temperature of 102 F. or over. The other did not. Postoperative blood samples (during the first 24 hours) were subjected to complement fixation tests for adenovirus antigen and these were reported as negative. Tissue explants were made from the tonsils of both patients and tissue cultures were obtained and studied for several months. No virus was isolated from the febrile patient. From the patient who did not spike temperature of 102 F. or more, a virus was finally isolated from the tissue cultures. However, it was the herpes virus and not adenovirus. It seems likely then that the viremia is not the cause of the pyrogenic reaction after T & A, particularly because most children's tonsils and adenoids contain adenovirus anyway, and only 16 percent have excessive febrile reaction.

To determine the possibility that dehydration played a part in spiking temperatures of 102 F. or more, 94 patients were studied. In this group, 39 patients were given intravenous infusion of 5 percent dextrose in 0.25 percent normal saline. The mean maximum temperature of the group given infusion was 100.28 F., whereas, the group not infused had an average mean temperature of 100.36 F., a difference of only 0.08 degree. This finding does not agree with Johnson[3] who reported a 1.5 degrees differential. The infused patients seemed to react with less morbidity and appeared brighter; however, it does not seem probable that running a fever of 102 F. or more results from dehydration in our T & A cases.



Dr. Withers and his co-author practice otolaryngology in Houston. Their paper was originally presented to the Texas Medical Association Section on Otolaryngology in April, 1964.

Thus, none of the first three categories postulated as a cause of the temperature spiking seems to explain this reaction. The final possibility[2] is that the children reacting with temperature of 102 F. or over are ones who do so routinely in response to the recurrent tonsillitis which led to tonsillectomy. Parents or pediatricians were questioned on this point, and it was found that 54 percent of those patients who spiked temperatures of 102 F. or more postoperatively usually ran high fever of at least 102 F. with tonsillitis bouts at home. Although there may be other considerations which we have not thought of investigating, it seems that an innate difference in temperature regulating mechanism is the most plausible explanation for the temperature elevation of 102 F. or more in 16 percent of postoperative tonsillectomy cases.

Summary and Conclusions

Four hundred fifty tonsillo-adenoidectomy patients were studied in an effort to determine why 16 percent have temperature elevation of 102 F. or more in the first 24 hours postoperatively.

The investigators considered and discarded the following possible causes: showering of blood stream with bacteria or viral bodies, dehydration factors, foreign protein or alarm reaction or physiologic changes due to general anesthesia.

The most likely explanation postulated was the individual variation in temperature control response: 54 percent of patients running temperature of 102 F. or more postoperatively had run this type fever with recurrent bouts of previous tonsillitis which led to the T & A surgery.

Acknowledgment

Dr. Joseph Stool and Dr. M. D. Burnett, Department of Pediatrics, and Dr. J. L. Melnick, Department of Virology and Epidemiology, Baylor University College of Medicine, Houston, assisted in this study.

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Acoustician Reports Hearing By Electromagnetic Waves

Findings which may prove useful in the diagnosis and treatment of hearing problems indicate that there is some truth in claims of "hearing" meteors and jet planes traveling toward an observer at speeds faster than sound.

However, people do not hear these meteors or jets with their ears in the usual sense, says Dr. Clyde E. Ingalls of Cornell University.

Speaking before the Acoustical Society of America meeting in Austin, Dr. Ingalls explained that this kind of hearing is a sound sensation produced by electromagnetic waves traveling at the fastest speed known to science—the speed of light—and registering directly on the listener's brain or nerves near the brain.

Investigations made with electromagnetic waves from a radar system showed that persons standing in the edge of the radar beam could hear a sound like that of a bee buzzing in a window. When a square of window screen was placed between the test subjects and the beam, the subjects could not hear it nor were they able to hear it if they put one ear through a hole in the screen.

Indication that the brain or surrounding nerves receive the electromagnetic waves directly was shown when subjects placed the hole in the screen over a point in the center of their foreheads. This way they could hear the radar.

Drug Therapy in Cerebral Palsy

Current Status

RESEARCH & INVESTIGATION

THE HOPE THAT the plight of the cerebral palsied child will be controlled or minimized has led to the trial use of several drugs. An ideal one which relaxes the muscle spasms, improves the skills, and eases the care of the child without producing too much lethargy has not been found. Drugs that have been used in the cerebral palsied child for muscle relaxation and relief of anxiety include curare, neostigmine, mephenesin, reserpine, zoxazolamine, carisoprodol, emylcamate, primidone, chlorpromazine, meprobamate,[3] chlordiazepoxide,[2, 4, 5, 9] and diazepam.[6, 8]

Perhaps too much has been expected from one drug. There have been no drugs that show good results in a majority of patients by any method of measurement or clinical evaluation. However, usually a scattered few children seem to do extremely well on each medicine. Therefore, it is imperative to keep on trying newer ones which may prove valuable.

Some drugs have proven helpful in controlling certain associated symptoms in the cerebral palsied child. Examples of these are the anticonvulsants and antihyperkinetics. The anticonvulsants are too numerous and well known to mention here. The amphetamines are used frequently in the mildly brain injured children and are sometimes helpful in the motor impaired child with the superimposed "Strauss syndrome." Chlorpromazine, thioridazine, chlordiazepoxide, and diazepam have also been used as tranquilizers.

The difficulty in finding a method of assessment of drugs in cerebral palsied children is well known. Phelps[7] has advocated the use of graphs of various skills in the different areas of therapy. Baird[1] and Denhoff[3] have both indicated that specific measurements used in their clinics are not ideal for use in children and conclude that the clinical evaluation is more meaningful. The effect of a drug on everyday function as advocated by Woods[10] is difficult to compare from one study to another.

Sometimes the drugs have been evaluated in mentally retarded cerebral palsied children whose skills are poor and whose learning capacity is not of primary importance. In these an agent which will relax the child and make custodial care easier is of value. In a residential training school the situation is quite different. Two types of measurements are indicated, one a measurement of performance of skills and the other, an assessment of behavior. Graphs similar to those of Phelps[7, 8] seem most efficient for skill evaluations, and behavior rating scales based on Denhoff's[3] suggestions are being developed for the latter.

A study with the primary purpose of assessing the value of diazepam in the treatment of a group of cerebral palsied children participating in an intensive training program and with a second purpose of developing a method of evaluating the performance and behavior of cerebral palsied children on various drugs and treatment procedures was undertaken in a residential setting for educable cerebral palsied children. This report covers a pilot study.

Subjects

The subjects were 20 residential students at the Moody State School for Cerebral Palsied Children varying in age from 5 to 14. Various types and degrees of severity of cerebral palsy were included in the group, but in general the children represent the middle category, inasmuch as both the severely mentally retarded and the mildly

Reported is a pilot study of diazepam in treatment of cerebral palsied children in a residential training school.

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physically handicapped are excluded. Initially it was planned that all of the 45 students in the school would participate in the study and therefore the only criterion for selection of a child for the study was a period of adjustment (three months) to the school program before being placed on the study.

On admission to the school each child is examined by a pediatrician and an orthopedic surgeon who establish the diagnosis and possible etiology. Each student is then placed on an intensive training program including physical, occupational, educational, and speech therapy. The therapists are all experienced workers in the field of cerebral palsy. There were no changes in the program or personnel during the study.

Methods

The study was a crossover double blind design, each child receiving active medication for six weeks and an identical placebo for six weeks, thus acting as his own control. Random assignment determined the order in which the active medications and placebo were given. Personnel evaluating the children did not know whether the drug or placebo was being used and did not know at which point the switch was made. The dose of diazepam was either 2 mg. three times a day or 5 mg. three times a day and was not calculated according to body weight.

During the 12 to 18 consecutive weeks of the study the child's skills and behavior were evaluated and reported by each therapist. Two types of rating scales were used. An attempt to graph the child's performance in certain tasks in each department was made. Examples of this are length of standing balance, steps taken in braces, or with sawhorses in physical therapy; length of time for dressing or carrying through a feeding procedure in occupational therapy; number of correct sounds or length of time a sound could be sustained in speech; and number of correct responses in education.

The other rating scale was used to evaluate broader aspects of behavior such as alertness, cooperativeness, distractibility, and coordination. This is shown in Table 1. A scale of one to nine was used with five being the optimum level.

Results

The graphs of the child's skills were inspected. If the performance improved while the child was on the drug a "positive" score was given even though he may have been improving before medication was started. If there was a plateauing while on the drug, it was noted as "no change." If there was improvement before drug and a drop in performance afterwards, a "negative" score was given. Each child was not seen in each therapy. There were 49 graphic records on the 20 children. Fourteen were positive, 18 negative, and 17 showed no change. Table 2 shows the breakdown of these data according to therapy.

Behavioral ratings were given by each therapist on each child attending the specific class. Seventy-six ratings were made. The response to the drug was favorable in 24 of these and unfavorable in 35. No change was noted in 17 instances. Table 3 shows the relationship to the various therapies.

In the graphs of skills performed a child often showed improvement in one therapy and decreased performance in another. None showed improvement in all areas and eight had no positive ratings in any therapy.

Behavior ratings were also inconsistent in each child when measured by personnel in the different therapies. Four showed over-

TABLE 1.—*Moody State School for Cerebral Palsy Rating Scale for General Behavior.*

Name:_____ Dates:_____

1	2	3	4	5	6	7	8	9
				ACTIVITY				
Acutely hyperactive, restless, not still a minute.		Restless, frequently moving about, but able to settle down with encouragement.		Energetic and busy without excess of unnecessary activity.		Slow, not spontaneous, requires extra stimulation to act.		Inert, quite lethargic, stares blankly.
				SOCIALIZATION				
Unrestrained, extremely intrusive.		Rather forward, and sometimes meddlesome.		Usually plays and talks with others with active interest. Cooperation with group.		Occasionally joins with or speaks to others in group play, introverted.		Shy, never voluntarily plays with others.
				DISTRACTIBILITY				
Constantly shifts attention from one activity to another inappropriately.		Moderately distractible, quickly changes focus of attention.		Usually remains at activity till completion, and fairly persistent.		Overly persistent, difficulty in shifting attention appropriately to new events.		Extremely difficult to interrupt.
				AGGRESSIVENESS				
Very cruel to other children, combative, often requires reprimands.		Belligerent and dominating, aggressive, demanding, bossy.		Not outstandingly aggressive or passive, very seldom strikes except in self defense, makes reasonable demands.		Tends to be picked on by others, puts up minimal resistance, reluctance to make appropriate demands.		Often hurt by others, does not defend self, extremely passive.
				MOOD				
Euphoric, always joking, never serious.		Enthusiastic, happy, likes jokes, usually smiling.		Stable balance between happy, optimistic and unhappy, pessimistic.		Rather sober, quiet and unhappy.		Irritable, unfriendly, extremely unhappy, cries frequently.
				DEPENDENCY				
Refuses assistance, extremely resistant to outside help, efforts to perform tasks beyond his ability.		Expects to complete task without help, even when the situation requires help.		Asks for necessary help when needed, but usually prefers to do it on his own.		Rather reliant on help from others.		Always asks for help, even when able to perform alone. ("I can't do it.")
				COORDINATION (Gross) WITHIN PHYSICAL COMPETENCE				
Seems weak, no muscle tone.		Tires easily, lacks energy.		Can do nearly all motor acts with minimal extraneous movements.		Tenses up under stress, awkward, clumsy.		Tense all the time, falls frequently.
				COORDINATION (Fine) WITHIN PHYSICAL COMPETENCE				
Muscles appear weak, makes only few movements.		Tires easily.		Smooth movements and accuracy good.		Drops things frequently.		Unable to use hands at all.
				APPETITE				
Always voracious appetite, stuffs himself, needs to be restrained.		Often tends to overeat moderately.		Eats ample servings regularly.		Eats very little but will eat more when helped or encouraged.		Eats very little and refuses to eat more when encouraged.
				SLEEP				
Wakes frequently, cries, demands attention before going back to sleep.		Wakes frequently during night, restless, but does not require attention from personnel.		Sleeps soundly all night unless there is considerable commotion.		A heavy sleeper, never disturbed by noises.		Falls to sleep in class frequently.

TABLE 2.—*Response to Diazepam—Skill Ratings.*

Therapy	No.	Pos.	Neg.	No Change
Speech	9	3	3	3
Occupational	13	4	4	5
Physical	14	5	6	3
Special education	13	2	5	6
Total	49	14	18	17

These results were obtained on the graphs of skills in the various therapies while child was being given diazepam.

TABLE 3.—*Response to Diazepam—Behavior Ratings.*

Therapy	No.	Pos.	Neg.	No Change
Speech	16	5	7	4
Occupational	20	8	9	3
Physical	20	5	10	5
Special education	20	6	9	5
Total	76	24	35	17

The results of ratings of behavior of child on diazepam.

all improvement and six were rated negatively in all areas.

Three children had no negative responses in either the skills or behavior and two children had no positive responses in any of the rating scales. These two were incapacited and could not participate in the training program while on the medication.

Side Effects

Drowsiness was the only side effect noted. However, in some children the lethargy was to the degree that the student could not participate in the training program. One child on 5 mg. three times a day fell asleep while in a swimming pool. Another child was withdrawn from the school by her parents because she became so lethargic she could no longer walk. The dosage had to be decreased to either 2 or 5 mg. daily in four children.

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Comments

This double blind trial of diazepam in cerebral palsied children capable of participating in an intensive training program showed that the patients had no dramatic improvement. The two types of rating scales are still crude assessments of evaluation and when several areas of therapy are involved the child does not respond in similar manner to all at one time. Perhaps this is expecting too much since normal children also have good and bad grades during training procedures.

Behavior rating scales such as the one used need to be studied to determine how much of the variability is due to lack of agreement among raters and how much is due to variability of the patient's behavior from one situation to the next.

Two children showed an overall improvement of moderate degree. This was enough so that the therapists and parents requested continuation of the medication at the conclusion of the study.

A rejection of this drug for cerebral palsied children based on the results of this study would be premature in view of some of the design limitations, notably the small number of the subjects and the fixed dosages. It is possible that a careful adjustment of dosage on an individual basis would result in positive changes. However, the results of this preliminary study do not suggest that this drug will be particularly promising.

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Retropharyngeal Hemorrhage

A Fatal Case

HARVEY HAYS, M.D.

CASE REPORTS

IN 12 YEARS of general practice, I had never seen a case of acute hemorrhage caused by retropharyngeal abscess. There is a surprising scarcity of information on the subject, current or old, and little emphasis is accorded it in textbooks. Most articles are in specialty journals, however, the general practitioner is likely to see this condition at its onset, and should be alert to its dangers.

Review

Retropharyngeal abscess is characterized by high fever and bulging of the pharyngeal wall. There is great danger of hemorrhage. Rhodes[2] describes the pathogenesis and recommended procedure for treatment. Most cases reported have been in children; this probably is because of the rich lymphatic vessels of the retropharyngeal space. Of the cases autopsied, 75 percent had erosion of the internal carotid artery, and in the others, some branch of the external carotid artery was eroded.

Todman[3] reports an unusual case in a 22-year-old white man who had pharyngeal and cervical swelling with a Horner's syndrome, which was undiagnosed until the fatal hemorrhage. Havens[1] reports a case in which a hematoma was diagnosed preoperatively. The common carotid artery was ligated without draining the hematoma, with good results. Early recognition of the condition and its potential danger is important. Treatment is by ligation of the bleeding artery, usually the common carotid artery. The procedure is performed with the patient under general anesthesia, and endotracheal intubation is utilized. If a bleeding point can be seen, a ligature is placed loosely around the artery before making an incision. Children apparently tolerate ligation of the common carotid artery without serious consequences. If no bleeding point is evident, the mass should be aspirated with a small gauge needle to be sure the mass is an abscess and not a hematoma. Packs should be placed around the endotracheal tube before incising the abscess, and continuous suction should be used.

Case Report

This two and a half-year-old white girl was first seen Jan. 14, 1964. Her parents thought she had mumps. Examination revealed tonsillitis with temperature of 99 F., with bilateral cervical adenopathy larger on the left. Antibiotic therapy was prescribed. On Jan. 20, the child was seen again; the mother stated that the child had had a profuse nose bleed, and thought that she had "vomited blood." Examination revealed clotted blood in both nostrils with some bloody drainage on the pharyngeal wall. The tonsils were still slightly enlarged and inflamed, and the adenopathy was still present. Temperature was 98.8 F. Complete blood count numbered 3,700,000 RBC's; 10.4 gm. hemoglobin; 12,400 WBC's with normal differential. The patient was placed on a hematinic coagulant therapy, and her antibiotic was changed.

She continued to have a low grade fever, and had intermittent episodes of epistaxis. She was admitted to the hospital on Jan. 23 for work-up and observation following another bleeding episode. Her hemoglobin had dropped to 8.8 gm., and a chest roentgenogram showed mild bilateral pneumonitis. Hematinic and intensive antibiotic therapy was continued. The patient was dismissed on Jan. 27, afebrile and free of bleeding.

She was next seen on Feb. 6 for routine recheck. She still seemed chronically ill. Her mother had noted one "black stool" and said the child held her head to the left. The left tonsil was bulging medially and the adenopathy on the left was enlarged. Hemoglobin was 9.4 gm., RBC 3,700,000, WBC 5,100, and differential normal.

At this time I suspected a peritonsillar abscess. The patient was given an injection of gamma globulin, antibiotics were resumed, and she was scheduled for tonsillectomy and adenoidectomy, or incision and drainage of abscess and bone marrow studies.

She was admitted Feb. 10. Her hemoglobin had risen to 11.8 gm.; chest roentgenogram and upper gastrointestinal study were normal. Throat culture showed a nonhemolytic streptococcus sensitive to

The patient had tonsillitis, slight fever, and bilateral cervical adenopathy. She had profuse epistaxis, after which some bloody drainage was seen on the pharyngeal wall. Three weeks later, she was hospitalized for possible surgery and further studies, but before any surgical procedure was attempted, the child died of a sudden profuse hemorrhage.

every antibiotic tested except Kantrex. Following her studies, the child seemed to feel well. She ate her evening meal, and suddenly began hemorrhaging from her nose and mouth; she died in extreme shock.

At autopsy, there was a perforation (1.5 to 2.0 cm. in diameter) of the left lateral pharyngeal wall at the level of the soft palate. Evacuation of this region revealed a pocket which extended upward, downward, and laterally, and measured approximately 3 by 5 cm. Biopsies were taken from all four borders of the lesion. The pathology report described the specimens as pharyngeal tissue with acute hemorrhagic ulceration and chronic reactive inflammatory changes. The remainder of the autopsy was negative.

Discussion

A case of fatal hemorrhage climaxing chronic retropharyngeal inflammation has been presented. I omit the word "abscess" because this patient at no time had any clinical evidence of abscess formation. What was thought to be a peritonsillar abscess was in fact a hematoma posterior and lateral to the tonsil fossa, pushing the tonsil medially.

In retrospect, the first episode of bleeding was no ordinary "nose bleed." This child had a chronic, smoldering, granulating inflammatory process unchecked by several broad-spectrum antibiotics. The throat culture obtained the morning of her fatal hemorrhage may not have been the true pathogen that produced the eroded artery.

Persistent epistaxis and anything that bulges in the throat are conditions for which I have renewed respect. I hope that others will, also.

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COMMENT

The differential diagnosis in this case is either peritonsillar abscess, intratonsillar abscess, or retropharyngeal abscess. Two and one-half years is within the average for retropharyngeal abscess to occur; as such usually occur between three months to five years of age. These patients are always septic, and on inspection a protrusion of the posterior pharyngeal wall can be elicited either in the region of the nasopharynx, oropharynx, or occasionally in the lower hypopharynx. Retropharyngeal abscess is nearly always associated with dysphagia, altered speech, stiffness of the neck, and almost always cervical adenitis. The onset is acute with a septic type of fever and the history of an upper respiratory infection. Please note, however, that these abscesses are a little slow in developing. Both inspection and palpation should be carefully employed.

Blood from the nose is a warning sign of possible imminent danger. While it is true that young children sometimes will have epistaxis from the anterior septal vein, profuse bleeding, especially when seen on the posterior pharyngeal wall, should direct one's attention to larger blood vessels in the region of the neck. Occasionally a cervical adenitis with suppuration lying along the carotid sheath may result in erosion of the carotid vessels. In the case under discussion, an early carotid ligation might have been carried out with no untoward sequelae.

Other differential diagnoses include neoplasm and aneurysm, which should be kept in the back of one's mind. To help locate the aneurysm and plan the attack, an angiogram is useful to visualize such pathology and will show any leak or erosion that may be present. Angiography would also help to outline any neoplastic mass. This is a most instructive case and one that will not soon be forgotten.

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New Laws Promote Child Health

FRED P. HELM, M.D.

REVIEW ARTICLES

RECENT LEGISLATION, both federal and state, has the so-called new forward look; the purpose of this article is to review these laws, with attention to their effect on the Texas State Department of Health.

Federal Legislation

Congress raised the ceiling related to Children's Bureau* funds from $25,000,000 to $50,000,000. These monies are to increase by $5,000,000 per year for the next three years, and by the same amount every other year thereafter until the new ceiling is reached. Of these new monies, one-third must be spent in programs involving mentally retarded children, or for programs directly associated with mental retardation. Both maternal and child health and crippled children's funds will be increased for these services. It has been suggested that all maternal services be increased for (1) Pre- and postnatal care, (2) Extended nursing services for home care follow-up, (3) Establishing nurse midwife programs in some areas, (4) Day care facilities for children, to be associated with a hospital where overall clinical care can be extended, (5) Supervision of children in and out of school, (6) Training for nonprofessional personnel to care for children in day centers and provide other services, and for specialized nursing and other professional personnel needed for work with the mentally retarded, (7) Related research studies (these grants may be made directly to an individual through the Children's Bureau), and (8) Expansion of crippled children's services to include the mentally retarded.

*The Children's Bureau is a component of the Welfare Administration of the Department of Health, Education, and Welfare.

Knowledge of what is available is basic to meeting the patient's needs. This review will help acquaint physicians with new laws which have provided for funds to care for those who are unable to care for themselves.

State Legislation

Two far-reaching state programs were enacted; they concern phenylketonuria (PKU) and cystic fibrosis. In the Appropriation Bill, the 58th Legislature provided funds for case finding, diagnosis, medical care, purchase of dietary supplement as prescribed by the physician, and follow-up treatment or home care for the patient.

Phenylketonuria is a hereditary metabolic disease which, if untreated, leads to severe mental retardation. A screening test, done at about four weeks of age, detects phenylpyruvic acid in the urine of affected children. Positive diagnosis is confirmed by blood studies, and treatment is then begun. In Texas six PKU patients are being furnished dietary supplements for this condition at the request of their attending physicians. This is on prescription only.

Throughout the state there are 19 local health departments and 42 hospitals participating in a screening program for PKU. It is hoped that this program will be more widely accepted, and efforts are being made to acquaint both hospitals and physicians in the state with these services.

Cystic fibrosis is one of the most serious common chronic diseases of childhood. It is a generalized hereditary disease in which both parents carry and transmit the recessive gene. It has been estimated that 5 to 20 percent of the population carry the recessive gene, and that one case per 1,000 live births can be expected. At the present time, 96 cases are being treated at one medical center. It has been estimated that 25 percent of the total patients seen are medically indigent.

The major pathological lesions of cystic fibrosis are found in the sweat glands, tracheobronchial tree, lungs, and digestive glands. Before present advances in medical care, these patients died of bronchial pneumonia during infancy.

The eligibility requirements set forth in the statute governing the Division of Crippled Children's Services will, in general, apply to this service; included is a provision that the applicant be under 21 years of age and amenable to treatment.

The judge of the county of the child's residence must certify that the parents cannot finance the patient's care and treatment, and that a physician practicing under the laws of Texas has examined the patient and recommended his acceptance under the state program.

This service will be restricted to children believed to have cystic fibrosis, or who need treatment for this condition. This is essential, because the funds were made available "for salaries and wages, medical care, drugs, and related expenses for medical treatment of cystic fibrosis patients." Those funds are limited, and all monies appropriated were budgeted for case services only.

This service will include diagnosis for patients believed to have cystic fibrosis, including laboratory and all surgical procedures which may be necessary. It will also include hospitalization, special nurses, treatment in outpatient departments of a hospital or hospitals, special inhalation therapy including special equipment, and drug therapy which will be provided for hospital and home care.

The program also provides financing for transportation to and from treatment centers, and for any emergency treatment or transportation which may be necessary.

Authorizations will be issued for the examination or treatment of individual patients, just as they are issued in the crippled children's program.

At present, two treatment and diagnostic centers are used—the Texas Institute for Rehabilitation and Research in Houston, and the Cystic Fibrosis Center at Southwestern Medical School in Dallas. An additional center is to be established in the Department of Pediatrics, University of Texas Medical Branch, Galveston.

Patients requiring emergency treatment may be cared for in their home communities when necessary, preferably in hospitals which are used in the crippled children's program. Such cases, however, will be referred as soon as possible to one of the established centers for follow-up and further guidance.

Even though the program has been of short duration the case load is increasing monthly. To the present time, 56 cases have been authorized with an average cost of $427 per case. Of these there have been two deaths—one child age 4 years and 7 months, and one adolescent, age 14.

In spite of early diagnosis and comprehensive care, 50 percent of these children die before age 12, and 20 to 30 percent die during adolescence. Only a few live to adult age.

Dr. Helm, who is with the Texas State Department of Health, presented this paper before the Conference of City and County Health Officers held April 25, 1964, in Houston.

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Psychiatric Treatment Facilities Of a Metropolitan Community

RESEARCH & INVESTIGATION

A MOST IMPORTANT, influential, and controversial document in the psychiatric field is "Action for Mental Health,"[2] the final report of the Joint Commission on Mental Illness and Health. It summarizes the findings and recommendations of the Joint Commission, based on its studies between 1955 and 1961. The report points out that care of the mentally ill has lagged, that there is serious prejudice against mental illness, and that there is much need for further research and training; it also makes specific recommendations concerning application of present knowledge.

"Action for Mental Health" has been criticized on the grounds that it appears to ignore or minimize the private practice of psychiatry, both outpatient and inpatient. Another common criticism is that it ignores the unequal distribution of psychiatrists and psychiatric facilities throughout the country, so that some areas are adequately supplied, perhaps oversupplied, especially the large urban areas.

Since the publication of "Action for Mental Health" there have been numerous conferences at national, regional, state, and local levels to discuss the recommendations and implications. Legislation has been passed to begin implementation of some of the recommendations, and more is being considered. In Texas, the State Department of Health's Office of Mental Health Planning is developing a mental health plan for Texas emphasizing psychiatric treatment in the home community. In view of these developments it is timely to consider some of the alleged shortcomings and omissions of the Joint Commission's report. In this context it may be of interest to review some relevant findings from a recent survey done in one metropolitan Texas county.

Procedure

The data for this report were obtained from surveys of Houston and Harris County which were done in the course of a Community Council-sponsored study.[1] In 1960, the Health Section of the Community Council established a Mental Health Services Study Committee to study the entire range of mental health problems in the community, the range of services available to meet these problems, and to recommend appropriate community actions to fill gaps between needs and resources. The 36-member committee was made up of representatives of medical groups and institutions, social agencies, public health agencies, schools, and government, as well as lay community leaders. Six subcommittees were formed, one of which was to study diagnostic and therapeutic services for adults. This subcommittee conducted the several surveys which are the basis of this report.*

Information on Harris County hospitals was obtained from each member of the Houston Area Hospital Council, and from officials of the state hospital system and the county psychopathic ward. Data from psychiatrists in private practice were obtained through the Houston Psychiatric Society. Data concerning private practice of psychologists were obtained via the Houston Psychological Association. Social workers were approached by the National Association of Social Work-

*Dr. Spencer Bayles provided assistance, and reviewed this material for the author. The Community Council of Houston and Harris County granted permission to use the data.

In Houston and Harris County, 10 of 34 hospitals designate sections for psychiatric patients. Slightly more than half the patients are admitted to private rather than public hospitals, and psychiatrists in private practice see 72 percent of the known mental patients. Data on unmet needs in this field are inadequate for evaluation.

ALEX D. POKORNY, M.D.

ers, San Jacinto Chapter. The Community Council approached 25 appropriate community social agencies. Finally, caseload data were obtained from all of the adult psychiatric clinics in the community.

Replies were received from 100 percent of the hospitals and clinics, and from 40 of 56 psychiatrists in private practice. For most calculations, it was assumed that the practice of those who did not respond was similar to that of the ones who did; to get an estimate for the entire community, then, the totals for the 40 are multiplied by a factor of 1.4 (hereafter called extrapolation). Only 29 replies were received from 88 psychologists polled, and only 12 of these did any private practice. The social work chapter polled only those five workers known to do private practice, with only one valid return. Because the information about psychologists and social workers was incomplete, and because it appeared that they saw only a small percentage of the total number of patients, their contribution is not considered in the rest of this report.

It should be pointed out that these community survey data have certain limitations. The most important shortcoming is that patients were not identified individually, so that there was no way of picking up duplications (such as when a patient is readmitted, or is discharged from one hospital and readmitted to another). Much of the hospital admission data is exact, whereas the data from psychiatrists in private practice is often based on estimates. There is no assurance that the individual cases treated in different settings are of equivalent type or severity; on the contrary, it is likely that the office patients were less sick than the hospital patients, and that the public hospital patients were more chronically ill than the private hospital patients, but the surveys provided no direct evidence on these questions. These limitations should be kept in mind in interpreting this report.

Results

Hospital Survey.—Of the 34 hospitals in the county, 10 have formal psychiatric bed sections. In addition, the state mental hospital serving the area is located outside the county. One of the 10 sections was in the county psychopathic ward, 1 was in a V. A. general hospital, 1 was in a state research and training institute, 2 were in private psychiatric hospitals, and 1 was in the city-county general hospital. The remaining 4 sections were in large voluntary general hospitals. The hospitals without psychiatric bed sections consisted of 3 specialized hospitals and 21 general hospitals, all of them smaller than the 4 with psychiatric beds.

The total number of psychiatric beds in the county at the time of the survey was 662. If the 367 V. A. beds were subtracted, this left 295. The state hospital did not have any particular number of beds assigned to the county. The psychiatric beds in the county can therefore be grouped as follows: V. A., 367; other public hospitals, 126; private psychiatric hospitals, 72; private general hospitals, 97.

Each of the hospitals with psychiatric beds, including the state hospital, provided us with either the number of admissions per year, or data on average patient census and average length of stay, from which the total number of admissions for the year could be calculated. For each hospital, this total of admissions was then decreased by the estimated percentage of patients who came from out-

side the county (none for city-county hospital, 10 percent for the private hospitals, 50 percent for the V. A. hospital). The county psychopathic ward, which is a receiving facility for patients undergoing commitment, was credited with only the estimated 10 percent of its admissions who, in the survey year, were not transferred directly to one of the other hospitals. The figures for the state hospital were already limited to residents of this county.

Using the total admissions for the year and applying the stated corrections, we arrived at a total number of psychiatric hospital admissions per year of 5,276. This is in a population of about 1,250,000, or about one admission per 250 population. Some of these were undoubtedly duplications and readmissions, so that the proportion of *persons* hospitalized in a year is smaller than 1 in 250.

The admissions from the county to the five public hospitals (city-county hospital, county psychopathic ward, state psychiatric institute, V. A. hospital, state hospital) totaled 2,508. Yearly admissions to the private hospitals were 2,768. Thus 52.5 percent of the admissions were to private hospitals in the community.

The average lengths of stay in the public hospitals were city-county, 23 days; county psychopathic ward, 10 days; state psychiatric institute, 30 days; and V. A., 60 days. No figure is available for the state hospital. The average lengths of stay in the private hospitals were shorter (7, 15, 18, 19, and 20 days; one not available). The longer stay in the public hospitals may reflect a slower pace of treatment, but may also mean that the more refractory cases gravitate to the public hospitals.

Clinic Survey.—Data on caseloads were obtained from the four adult outpatient clinics in the city (one voluntary general hospital, a V. A. clinic, the city-county hospital clinic, and the state psychiatric institute). Figures were obtained on the number of cases active at the beginning of one year and the number of new cases picked up that year. These were added to yield the number of different persons seen in one year, a total of 2,798.

Survey of Psychiatrists in Private Practice.—As has been stated, replies were received from 40 psychiatrists in private practice. To arrive at county totals, it was assumed that the other 16 had the same caseloads as the average of the 40.

The mean number of different individuals seen during a year by the average psychiatrist was 155. If this is multiplied by 56, it yields a total of 8,680 individuals seen during a year. The average psychiatrist's caseload contained 16 percent children under 18, and if this is subtracted, it leaves a remainder of 7,291 adults seen during a year. This should be compared with the 2,798 adults seen in clinics. Thus the private practicing psychiatrists saw 72 percent of the total number of individuals seen, disregarding all possible duplications.

One of the points made in "Action for Mental Health"[2] is that psychiatrists, who are the most broadly trained practitioners in the mental health area, should concentrate their efforts on "major mental illness," also called the "core problem," leaving the more minor or transient maladjustments to nonmedical therapists, counselors, and others. An attempt was made to see how the practitioners of Harris County spend their time in relation to the question of treating the most seriously ill versus the less seriously ill. There was no direct way to evaluate the seriousness of the illness of patients. It is no longer necessarily true that the seriously mentally ill are hospitalized, and that only neurotics and less seriously ill persons are treated as outpatients. This is especially so since the introduction of psychoactive drugs and various social changes, such as the open hospital policy and the greater tolerance of mentally ill persons in the community. Subject to these limitations, we do have some figures on proportions of time the psychiatrists spent with hospital patients and outpatients. The percentages of time spent at various kinds of direct patient services by the 40 responding psychiatrists were converted into "full-time equivalents"; that is, the fractions were added, yielding a number equivalent to a certain number of persons working at such activity full time. These are shown in Table 1.

TABLE 1.—*Time Spent in Patient Services.*

Type	Full-time Equivalent
Psychoanalysis	3.6
Other individual psychotherapy	19.5
Group psychotherapy	1.8
Management of hospital patients	4.3
Diagnostic consultations	6.7

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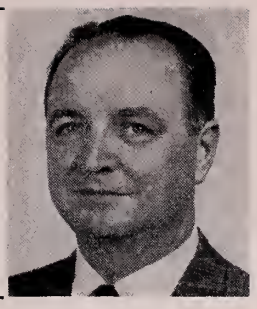

From this, it appears that if management of hospital patients is taken to be the equivalent of dealing with "major mental illness" and the other types of service are assumed to be for "minor mental illness," then certainly the private practitioners are spending a majority (88 percent) of their professional time with the less seriously ill.

Unmet Needs

One of the main goals of the survey was to assess the unmet needs in the area of psychiatric care, the shortages of service existing in the community. As it turned out, no very good information on this question was obtained. The 662 psychiatric beds in the county had an average occupancy of 554 patients (84 percent). Except for the V. A. hospital, the hospitals reported small or no waiting lists. Of 40 psychiatrists, 3 said that they had a waiting list of a few days, 12 said patients would have to wait one to two weeks, and 5 had waiting lists of two to six weeks.

Of 40 psychiatrists, 36 said that they saw patients who were unable to pay for recommended treatment. If the number of such individuals mentioned is extrapolated to all 56 psychiatrists, it would include 1,625 individuals per year.

The survey as conducted came up with fairly good data on what facilities *are* available, how many and what kind of patients *are* being treated, and other positive, existing situations. However, little light was cast on the negative or missing aspects, such as the additional beds needed and the psychiatrically sick who were *not* being seen. It is likely that there is a large number of potential psychiatric patients who never apply because they do not think of their symptoms as being psychiatric, do not know of treatment resources, refuse to come because of the nature of their symptoms, feel they cannot afford it, cannot arrange time, or because their relatives object, and so forth. There are probably others who make some contact, but drop out when they encounter realistic difficulties.

It has been noted that most of the hospitals reported no waiting lists, or short ones. This probably is misleading in regard to private psychiatric bed sections; the nature of private hospitalization is such that most admissions are "emergencies," and if a bed is not immediately available the patient will be referred elsewhere, even out of town, rather than placed on a waiting list.

The agencies, psychiatrists, psychologists, and hospitals surveyed did make numerous individual comments about unmet needs in the community. A large proportion of these had to do with needs and functions outside the adult diagnostic and treatment field, including such needs as residential facilities for disturbed children, hospital facilities for psychotic children, more services for delinquent youth, more facilities for training and research, and more effort in public education. Needs and shortages which did fall in the adult area included a state hospital in or adjacent to the county, precommitment and follow-up services for public mental hospital patients, late afternoon and evening clinics, halfway houses, sheltered workshops, and other rehabilitation services. Even these comments and opinions were based on the visible group—persons who already had made some application or overture for service. Most of the persons polled would not have any knowledge of those potential patients who stay away completely.

Discussion

One of the questions raised early in this report concerned the relative capacities of public and private facilities. Within the limitations of this study, it appears that in Harris County private hospitals account for more than half the admissions, and private practitioners account for almost three-fourths of the adult cases seen as outpatients. This would be more impressive if duplications and readmissions could have been eliminated, and if it were known that cases were of comparable severity. It is hoped that the data being gathered (1964) by the State Office of Mental Health Planning will provide such additional information.

The question of whether shortages exist in this county was more difficult to answer. There were no impressive waiting lists and hospital sections were not overcrowded. However, the nature of the conditions dealt with is such that patients and families seem to make other arrangements rather than wait. No satisfactory picture of the really hidden needs was obtained.

Summary

A portion of the findings of a survey of the psychiatric treatment facilities of Houston and Harris County is reported. Ten of 34 hospitals were found to have formal psychiatric bed sections. It was calculated that there was one psychiatric hospital admission per year for each 250 population. Of the total admissions, 52.5 percent were to private hospitals, and 47.5 percent to public hospitals. When adult caseloads of public clinics and private psychiatrists were compared, it was found that 72 percent of the individuals were seen by private practicing psychiatrists. Management of hospital patients accounted for only 12 percent of the patient-contact time of the psychiatrists. An attempt was made to assess the unmet needs in the field of psychiatric care, but the survey as conducted did not yield adequate data on this point. These findings are discussed in relation to recommendations made in the volume, "Action for Mental Health."

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Dr. Pokorny, 2002 Holcombe Blvd., Houston.

Tests Now Able To Confirm Contraction of German Measles

Whether a pregnant woman has contracted German measles can now be determined by laboratory tests.

Dr. Stanley A. Plotkin of Philadelphia reports his laboratory has been "deluged" with requests for help in cases where women have been exposed to German measles during the early months of pregnancy and where there is doubt about whether she has actually contracted the infection.

Diagnosis of the disease can be difficult because it has mild and atypical forms. Confirmation or denial of the disease in the expectant mother can determine whether therapeutic abortion is to be considered. The disease early in pregnancy can cause mental retardation or other defects in the offspring.

Dr. Plotkin has reported his findings in the Oct. 26 issue of *Journal of the American Medical Association.*

Chromoblastomycosis in Texas

Report of Four Cases

John T. Gardner, M.D.; Bedford F. Pace, M.D.;
Robert G. Freeman, M.D.; John M. Knox, M.D.; and William Smith, M.D.

CASE REPORTS

CHROMOBLASTOMYCOSIS was first reported by Lane[7] from the United States in 1915. Although most cases have occurred in tropical and subtropical countries, its worldwide distribution is now recognized. A review of the literature revealed that only 28 cases have originated in North America, most from the southern states. Howles and associates found nine cases in Louisiana during a two-year period,[6] whereas only 12 cases were reported from the United States in the preceding 38 years.

The only report of the disease from this state was Wilson's[12] in 1933. Recently four cases of cutaneous chromoblastomycosis from southeast Texas have come to attention.

Case Reports

Case 1.—A retired refinery worker who lived near Beaumont was first seen by his physician in June, 1961, for a lesion on the dorsum of the proximal phalanx of the left middle finger. It was painless and had grown slowly since onset one year previously.

The lesion was pale, blue, verrucous, raised, and flat-topped. A specimen obtained by punch biopsy revealed a granuloma containing clusters of brown yeast-like organisms characteristic of chromoblastomycosis. Culture on Sabouraud's agar at room temperature grew a black colony identified as *Hormodendrum pedrosoi.* Intralesional injections of amphotericin B (4 mg. in 4 cc. 2 percent procaine) were given and repeated in July and August, 1961. After the last injection a second specimen contained only a few organisms. By late August the lesion regressed to a flat, erythematous, atrophic patch. The patient refused further injections and was not seen again.

Case 2.—A Beaumont housewife consulted her physician for a slowly growing, raised, verrucous, plaque-like eruption involving the dorsum of the great, second, third, and fourth toes of the right foot. The lesions were asymptomatic and had been present five years. A skin biopsy was interpreted as chromoblastomycosis. *H. pedrosoi* was identified from a culture on Sabouraud's agar. Intralesional injections of amphotericin B were so painful that the patient refused further injections. She then received a short course of x-ray therapy at weekly intervals for four weeks. On several occasions afterward the skin was abraded and 20 percent podophyllin in alcohol was applied. At first the podophyllin was applied for one hour, but later overnight. The lesions healed rapidly and only a macular hyperpigmented plaque remained. The disease has not recurred.

Case 3.—After scrubbing floors on her hands and knees for many years, a 59-year-old colored housewife from Baytown developed a small, painfully tender lesion over the left patella. When she consulted her physician the nonspecific-appearing granulomatous nodule had been present for six weeks.

This lesion was surgically excised and the specimen was an ellipse of skin 1.8 by 1.2 cm. with a 1 cm. slightly yellow nodule in the center and extending into the superficial dermis.

Microscopically the lesion was granulomatous with pseudoepitheliomatous hyperplasia. Focal small abscesses in the epidermis and dermis contained groups of giant cells, epithelioid cells, and brown thick-walled yeast organisms singly and in clusters. In the keratin of one plugged follicle and in adjacent keratin, organisms were present as short chains or mycelial forms. The diagnosis was chromoblastomycosis. No culture was obtained since the lesion was completely excised and fixed in formalin. Excision apparently was curative because there has been no evidence of recurrence.

Case 4.—This patient, a white farmer from Corrigan, Tex., noted the onset of an asymptomatic verrucous lesion on the medial plantar surface of his left foot 12 to 14 years ago, which slowly enlarged. In the past two years he developed mild to moderate edema of his left foot and leg. Within the past year a crusted nodule appeared at the vermillion border of his lower lip.

The patient had lived in Corrigan since he was

Recently four cases of cutaneous chromoblastomycosis from southeast Texas have come to attention. Prior to this only one case has been reported in the state and that was 31 years ago.

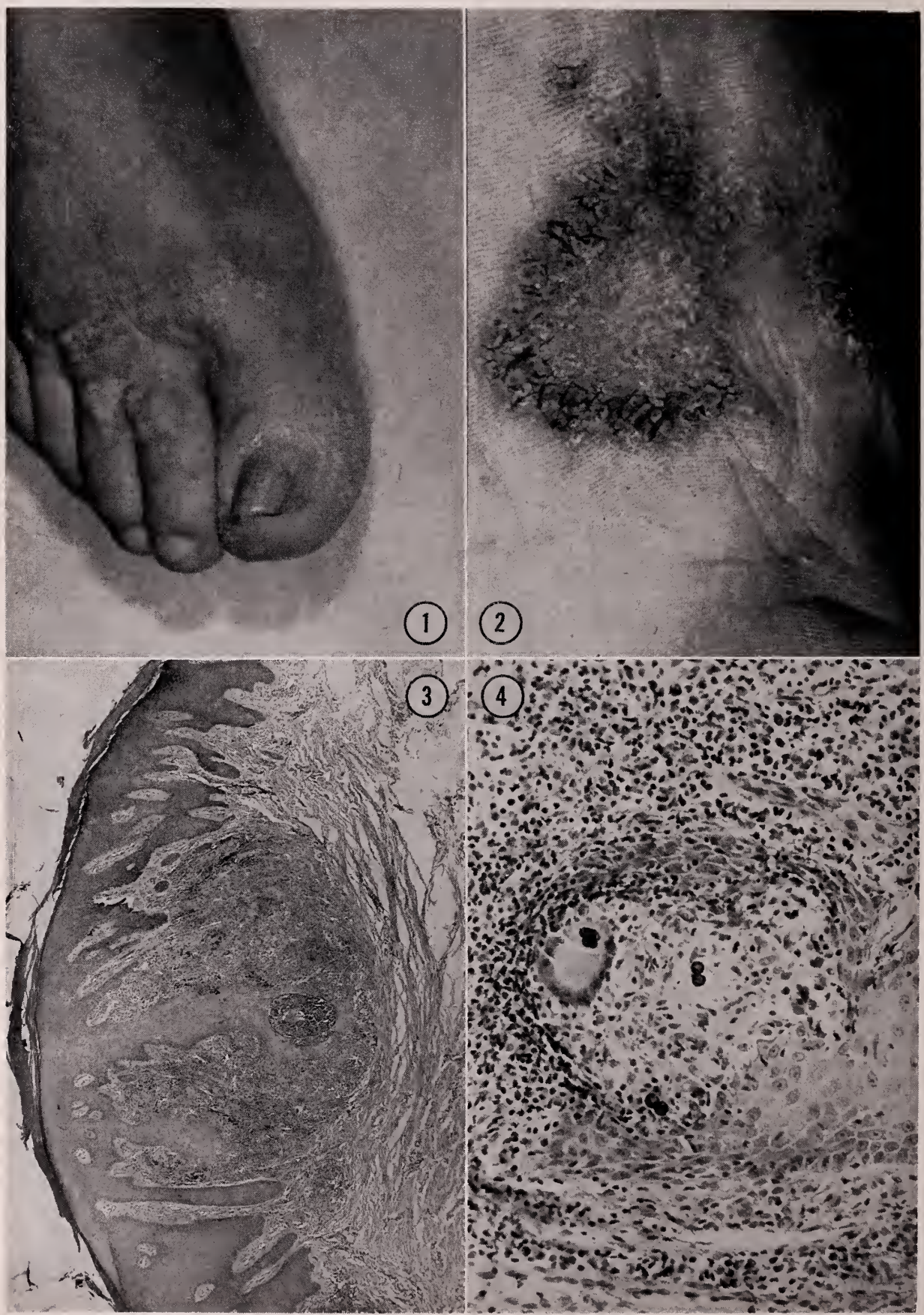
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Fig. 1. (Case 2) These raised verrucous plaques of the dorsum of the foot and toes had been present five years.
Fig. 2. (Case 4) These greenish-brown hyperkeratotic plaques had slowly enlarged since their appearance 14 years ago.
Fig. 3. (Case 2) Hyperkeratosis and extreme acanthosis were associated with a granulomatous reaction and intra-epidermal abscesses. (Hematoxylin-eosin stain. 56 X.)
Fig. 4. (Case 2) The granuloma contained clusters of brown yeast organisms. Some of these were in Langhans' cells. (Hematoxylin-eosin stain. 395 X.)

five. He had done farming, sawmill work, and school maintenance. His general health had been excellent, and he had no other complaints.

Physical examination revealed an alert elderly white man whose pertinent physical findings were confined to the left foot and leg. A large raised green-brown verrucous plaque on a slightly erythematous base was located on the instep of his left foot. Several smaller lesions on the medial surface of his foot were similar. Central clearing and superficial scarring were observed in the large plaque. There was mild edema of his left foot and leg. X-ray examination of the left foot revealed no bony abnormality. A 4 mm. crusted nodule with telangiectasia on his lower lip was a basal cell tumor clinically and histologically.

A biopsy of the verrucous lesion demonstrated epithelial hyperplasia and a granulomatous infiltration of cell with brown septate yeast organisms of chromoblastomycosis. No treatment has been attempted.

Etiology

Five closely related species of fungi are recognized as etiologic agents for cutaneous chromoblastomycosis. These include *Phialophora verrucosa; H. pedrosoi; H. compactum; H. dermatitides,* and *Cladosporium carrionii. C. trichoides* has been recovered from brain abscesses in rare instances. Nomenclature for these organisms is confused and some authors prefer the generic name *Cladosporium* while others prefer *Fonsecaea* rather than *Hormodendrum. Phialophora* also has been used as the generic name for all the pathologic species. *P. verrucosum* and *H. (Cladosporium) pedrosoi* are the etiologic agents common in this country.

Various saprophytic species of *Cladosporium* appear morphologically and serologically identical to some of the pathogens. The five pathologic species are known to sporulate by one or more of three distinct methods of conidial production: *Cladosporium (Hormodendrum),* pseudo-*Acrotheca,* and *Phialophora.* Since all three types of sporulation can occur simultaneously or in various combinations and proportions, depending on the individual isolate and conditions of culture, identification may require the efforts of a skilled mycologist.

The discovery of an undisputed *Cladosporium (C. carrionii)* as a frequent and widespread agent of chromoblastomycosis requires thorough mycological identification. Suspected contaminants can no longer be discarded if they lack *Phialophora* or pseudo-*Acrotheca* types of sporulation. Silva[10] points out that various species of *Cladosporium* are frequently introduced as air contaminants into cultures of clinical specimens. Also saprophytic *Phialophoras* that attack wood may possibly be confused with *P. verrucosum.*

Mode and Source of Infection

Chromoblastomycosis is probably contracted by direct inoculation of the fungus into the skin from rotting vegetation, wood, and soil. An injury by wood or vegetation often precedes onset. Emmons and colleagues[5] emphasize the disease is not contagious. In 1952, Azulay[1] reported the first case of experimental chromoblastomycosis in man. Typical lesions followed inoculation of a suspension of *Fonsecaea pedrosoi.* In 1957 Ridley[9] reported the development of chromoblastomycosis in volunteers inoculated with *C. carrionii* obtained in cultures from fence posts.

Clinical Features

Chromoblastomycosis is primarily a skin disease but internal organs have been involved with and without skin lesions. Brain abscesses due to chromoblastomycosis have occurred in 18 patients but only one had an associated skin lesion.

All ages and all races may be affected although the disease has more often afflicted men. Chromoblastomycosis occurs chiefly among farmers or those associated with farm produce. Lesions are usually on extremities but have been seen on most other areas of the body, especially the face, neck, chest, shoulders, and buttocks. A case involving the nose resembling rhinosporidiosis has also been reported.[11]

Pardo-Costello and co-workers[8] classified chromoblastomycosis into five clinical types.

1. The *verrucous* or *papillomatous* type begins as a group of small nodules or papules

with a scaly surface. These nodules grow and usually form large circular masses which tend to heal in the center and leave scars. Abscesses may be found in the area of granulation tissue but suppuration does not result. They may resemble blastomycosis or tuberculosis verrucosa cutis.

2. The *tuberculoid* type is probably an early form of verrucous chromoblastomycosis. The lesions begin as small patches or nodules with erythematous areolas and little scaling.

3. The *syphiloid* type is nodular and scaly with some erythema. The nodules are small, flattened, and serpiginous, annular or arcuate in their arrangement. The lesions may resemble a superficial late syphiloderm. Ulceration with a hypertrophic base may be present due to granulation tissue. The surface may be covered with crusts.

4. The *psoriasiform* type consists of superficial inflammation and infiltration with lesions covered with thick white adherent scales.

5. The fifth group consists of those lesions which become *cicatrized,* and those which, by blocking the lymphatics, produce elephantiasic enlargement of the extremities. This group probably represents an end result or an advanced stage of the disease, since scarring is found in lesions of long duration, and swelling of affected areas occurs when there are many lesions on an extremity.

Thus it may be concluded that the differential diagnosis of chromoblastomycosis must include tuberculosis verrucosa cutis, late superficial syphiloderm, epithelioma, other mycotic granulomas such as blastomycosis and coccidiodomycosis, psoriasis, sarcoid, granuloma annulare, pyoderma vegetans, Madura foot, porokeratosis, and possibly Hansen's disease.

Diagnosis

A high index of suspicion must be maintained to make the clinical diagnosis of chromoblastomycosis. When a biopsy is performed the diagnosis can usually readily be made on routine hematoxylin and eosin stained tissue because the characteristic brown yeast organisms are usually evident. Frequently, scrapings of scales and debris from the lesions cleared in 20 percent potassium hydroxide will, on direct microscopic examination, reveal the typical dark brown septate fungal bodies. Culture on Sabouraud's media at room temperature is not difficult. In very few cases will the diagnosis depend solely on culture.

Dr. Gardner presented this paper at the meeting of the Section on Dermatology in 1963 at the TMA Annual Session in Dallas.

Prognosis and Treatment

Cutaneous chromoblastomycosis is a benign disease with good prognosis if treated before the infection becomes widespread. Slow progression aids the physician in this regard. In very late cases scarring and elephantiasis may be troublesome.

Small lesions can easily be destroyed by cauterization, electrocoagulation, or surgical excision. For more extensive lesions, oral iodide preparations given to the limit of tolerance are often recommended. X-ray therapy also has its advocates. Bonilla[2] treated three cases successfully with large weekly oral doses of calciferol.

Intralesional amphotericin B was effective in two patients of DeFeo and associates.[3] The 40 mg. per ml. blood level necessary to inhibit growth of organisms in vitro far exceeds the maximum blood level tolerated by human beings, therefore, local infiltration is the only feasible method for obtaining an effective concentration. Derbes[4] has recently described the intralesional injection of amphotericin B by vibropuncture. Pain is the chief objection to intralesional injections of amphotericin B. No serious systemic effects have been noted.

Summary

Cutaneous chromoblastomycosis has been recently diagnosed by histopathologic examination in four residents of southeast Texas. Two cases were caused by *H. pedrosoi;* however, cultures were not obtained in the third and fourth cases. One patient was treated successfully with intralesional amphotericin B, another with topical podophyllin, and the third by surgical excision. The fourth patient has not yet been treated.

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Pharmacist's Plan Helps Prevent Poisoning Accidents

Because many children are poisoned from taking old medicine left lying around, a New Jersey pharmacist conducts an annual clean-up campaign and offers to pay for every leftover prescription that is returned to him.

During a well-advertised three-day campaign this druggist offers a 25-cent credit towards the purchase of anything in his pharmacy for every old Rx item returned—regardless of when or where the prescription was originally bought.

Since most patrons use this credit to buy substantial amounts of needed drug items, the store has always profited by the campaign, reports the Sept. 14 *American Druggist.*

Historical and Philosophical Aspects Of Iatrochemistry and Iatromechanics

Part I. The Alchemists

HISTORICAL ARTICLES

MEDICAL SCIENCE progresses slowly and much is learned from failure. Problems remain unsolved, and physicians in every age have relied on philosophy to supplement their scientific knowledge.

Every system of philosophy has repercussions on medicine, just as the experience of science and medicine has repercussions on philosophy. Speaking in a general way, metaphysics as applied to medicine is the speculative search for the "essence" of the disease instead of its observable causative factors and nature. Hence, *iatrochemistry* and *iatromechanics* are metaphysical creations in the sense that they are not founded on natural observed facts, but upon the intellectual faculties of their inventors who developed their doctrinal systems according to their own viewpoints. Metaphysical medicine does not consist merely of forces, an archeus, vital principle, or soul. It gives to "phenomena" an importance that they do not have exclusively, and derives other principles and processes from other purely rational metaphysical doctrines.

While there have always been physicians who sought to give medicine a philosophy, at certain periods in history, their attempts have been more pronounced. One such period was in the 16th and 17th centuries, the time of iatrochemists and iatromechanists.

The medical philosophy of the 16th and 17th centuries reflected the tempestuous tendencies of this period. The minds of the scientists and philosophers mirrored the nature of change as it was exhibited in the political and social events as well as in the spiritual revolution of the time. New ideas flourished and political revolutions against foreign domination were accompanied by a similar revolt against the restraint on research. Italy was in a grave economic crisis, for the discoveries of Columbus and others had diminished the maritime importance of the Italian ports of Venice and Genoa by opening new commercial routes. The Thirty Years War in Germany destroyed flourishing cities, ruined industries, and interrupted commerce. On the other hand, Holland and England reached the height of their maritime power. In art, motion and emotion found expression in the works of El Greco, Bernini, Velázquez, Rubens, and Rembrandt. The literature of Shakespeare, Milton, and John Donne along with the satirical darts of Molière reflected the glory of the age.

The 17th century was a period of great philosophical individualism. Giordano Bruno, Campanella, and Galileo bear witness to the strivings toward freedom of thought and to the dawn of scientific liberty. Voices were being raised against the ancient authorities, especially against their epistemology and metaphysics.

Francis Bacon and John Locke emphasized the value of observation and the experimental method, while the static universe of Copernicus was replaced by the dynamic one of Kepler, Galileo, and Newton. René Descartes was of great importance because of the effect of Cartesian philosophy on medicine. He took the idea of Plato, as developed by St. Augustine, that our intelligence knows nothing as certainty except that which confronts it at the moment, and that nothing is more present in our thought than the thought istelf. He made this the basis of his philosophy. Descartes' book *De Homine* (1662) is accepted as the first physiological text. The human body is a machine created by God, and in it dwells a soul, which

History shows that medicine does not remain apart from the philosophical trend of the times. This is well demonstrated during the 16th and 17th centuries when the lore of the alchemists was blended with the mysticism of Paracelsus and the dualism of Descartes to steer medicine in the direction of experimental research.

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synthesizes the thinking substance. From this and his belief that all the activities of the organism are forms of motion, and that this motion can be submitted to precise physical and mathematical examination, was born the school of *iatrophysical* medicine.

Sanctorius made metabolic studies on patients by the use of his scale, and Borelli used mechanical laws to explain vital phenomena. Along with this mechanistic image of man, there developed another movement, one toward a chemical explanation of vital phenomena. Van Helmont (1577-1644), a Capuchin friar of Brussels and an able chemist, was the founder of the *iatrochemical* school. He swayed between science and mysticism. Much of his image of man was based on ideas of Paracelsus. Paracelsus had tried to set new tasks for chemistry and make it a scientific discipline, but his own mysticism misled his followers.

Francios Sylvius (1614-1672) at the University of Leiden constructed a chemical picture in a purely Galenical frame: All diseases were explained and treated chemically. Willis (1622-1675) of Oxford, combined the purely chemical approach with keen observation.

The principal characteristic of the 17th century was an orientation toward experimental research, which also was influenced by the still prevalent authoritative words of the Ancients, the mysticism of Paracelsus, and the dualism of Descartes.

Origins of Alchemy

In the Middle Ages it was commonly thought that God himself had given Adam knowledge of alchemy. It was revealed through Raziel, the angel of mysteries, and it was passed on through Enoch, Abraham, Moses, and Job. It passed from Job to his descendants and on to the alchemists of the Middle Ages. Enoch became identified with both Hermes and the Moslem prophet Idris. Western alchemy was not born on the soil of ancient Greece, but came from Egypt. There it was a product of a late era; in support of its Egyptian origin, it is known that the Arabic word "el-kimyâ" (alchemy) came from the Egyptian "kême" (black earth). "Black earth" refers to the life-giving soil of the Nile,[1] and according to the Alexandrian alchemists, it was the original matter to which all metals must be reduced before being turned into gold. When the Greeks took possession of Egypt under Ptolemy they found in the temples a collection of works on metallurgy. These were attributed to the god Thoth, and the priests claimed that by using these sacred writings, they were able to convert base metals to gold. The Greeks saw in Thoth their own god, Hermes, who was among other things, the patron of thieves.[2] Little wonder that they accepted these tales of the transmutation of base metals into gold without question. Plato had stated that there were many kinds of fire and water, and also that it was not impossible for one element to pass over into another.[3] Aristotle's basic view of the elements and his doctrine of *exhalation*, as expressed in his *Meteorology*, was taken over by the Islamic alchemists and became the foundation of their theory of metals, the sulfur-mercury theory.[4]

The metallurgy of the Egyptians and the philosophy of Plato and Aristotle provided the experience and the theory; but the mystical element, which bound alchemy into a whole, was the Hermetic, Gnostic, New-Py-

thagorean, and Neo-Platonic literature of the first three centuries after Christ. Success in alchemy could never be achieved by base persons. Aspiration, self-discipline, and self-consecration were necessary, along with diligence and a knowledge of the Hermetic writings. The steps taken by the alchemist could not be displayed to the gaze of the vulgar, so a form of cryptic writing grew up whereby the true sense was concealed from all but the initiated.

The Arabian alchemists obtained their initial knowledge from two sources: the works of the Persian School and the writings of the Greeks of Alexandria. In their hands it developed into chemistry, and from them, via the Spanish Moors, the European chemistry of the late Middle Ages was derived. The most famous of these Arabian alchemists was Abu-Musa-Jabir-ibn-Haiyan (Jabir or Geber) who lived about 776 A.D. He divided alchemy into (a) spirits and volatile bodies, such as camphor, mercury, arsenic, and sulfur; (b) metallic bodies; and (c) pulverized solids.[5] Alchemy was combined with astrology, and it was believed that there was an occult connection between the planets and the metals, as well as between the various parts of the human body and the signs of the Zodiac. Among the disciples of Geber were Avicenna, Avenzoar, and Averroes. The returning Crusaders aided the spread of alchemy, and in Italy a warm welcome was given to it, especially among the educated people. Roger Bacon was among the few scholars who denied the possibility of transmutation of metals, yet he made many chemical experiments and described gunpowder.[6] Some of the learned men of the 13th and 14th centuries who studied alchemy were Albertus Magnus, Thomas Aquinas, Arnoldus Villanovanus, Raymondus Lullius (Ramon Lull), and Vincent of Beauvais. The alchemist has always been a favorite figure in art and literature, and was criticized by Chaucer and Ben Johnson in their writings.[6]

[Next month: Part II. Paracelsus: Alchemical Mystic.]

Dr. Keezer, the medical director of the Oklahoma State War Veterans Home Facility at the Central State Griffin Memorial Hospital in Norman, has acknowledged indebtedness to Drs. Carlton W. Berenda and William Horosz of the Department of Philosophy, University of Oklahoma, for their constructive criticism of this paper.

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Ceramics May Aid Orthopedic Patients

The ancient craft of the potter has given researchers an idea for a bone prosthesis. A mixture of clay and new epoxy plastic has been used in experimental studies with animals to repair difficult fractures. It is reported to stand up well.

Dr. Lyman Smith of Northwestern University Medical School, in *Archives of Surgery,* states that the new material probably will be used first for hip prostheses since it has "the same modulus of elasticity as bone and will bend symmetrically with it," thus avoiding localized stresses.

Clinicopathologic Conference

Michael Blaese, M.D.; George B. Carter, M.D.; Burton Combes, M.D.; John Fordtran, M.D.; Leonard Madison, M.D.; Jack Barnett, M.D.; Donald Seldin, M.D.; and Sidney Stewart, M.D.

University of Texas Southwestern Medical School

Clinical Summary

DR. BLAESE: This 47-year-old white saleswoman was first seen at Parkland Memorial Hospital on June 18, 1964. She had been having episodes of coughing with epistaxis and hemoptysis for three months. Prior to admission, a chest x-ray obtained as a tuberculosis screening measure was said to have demonstrated "walking pneumonia." The tuberculin skin test was negative. Following treatment of unknown type, her cough subsided, but she continued to have malaise, anorexia, and weakness. She lost about 20 pounds during this period, but continued to work five days a week until the day of admission. A history of excessive intake of alcohol was obtained, but reportedly she had been eating well in the time prior to this first admission. She complained that two days prior to admission she developed swelling of her hands and feet, and that on the morning of admission her hands would "curl up" in association with painful tingling of her fingers.

Following physical examination, she was described as a chronically ill white female. Blood pressure was 100/60 mm. of mercury, pulse 80, and temperature 99.4 F. A positive Trousseau's sign was elicited, and there was three plus edema of feet to ankles and hands to wrists. The liver was not tender. It was palpable 6 cm. below the right costal margin, and the spleen was palpable 2 cm. below the left costal margin.

Chest x-ray showed a mass in the right hilar region. [See radiologist's discussion.] The serum calcium was 7.5 mg. per 100 cc. and the potassium was 2.9 mEq. per liter. These electrolyte values returned to normal on hospital diet alone, although the Trousseau's sign was again positive on July 4.

Liver biopsy showed Laennec's cirrhosis, fatty state. Laboratory findings on this admission are shown in Table 1.

On June 29 an exploratory thoracotomy was done because of the persistent right hilar mass. A 5 by 5 cm. hard nodular mass in the subsuperior segmental area of the lower lobe of the right lung was found, and this lobe was resected. The remainder of the lung was normal, although it was adherent to the chest wall by fibrous adhesions. The pathology report indicated that the mass was an area of organizing pulmonary infarction with "central degeneration." The postoperative course was unremarkable, and she was discharged on July 6.

The next and last hospital admission was on Aug. 18. At that time she complained of profound weakness, which necessitated spending most of the time in bed. She had not had the strength to eat much for several days prior to admission. She had lost 15 pounds since discharge, and stated that she had been having fever with soaking sweats for about three weeks. A cough had persisted after surgery, and she had had several

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This is the eighth in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School. Dr. C. T. Ashworth, professor and chairman of the Department of Pathology, serves as guest editor for the series.

Participating in this conference are Dr. Michael Blaese, Straight intern in medicine, Parkland Memorial Hospital; Dr. George B. Carter, Department of Radiology; Drs. Burton Combes, John Fordtran, Leonard Madison, Jack Barnett, and Donald Seldin, Department of Medicine; and Dr. Sidney Stewart, Department of Pathology.

A 47-year-old woman was hospitalized with coughing and hemoptysis. She had hypocalcemia, mild tetany, and developed terminally a septic temperature and a mass in the left upper abdominal quadrant.

TABLE 1.—*Major Laboratory Findings on First Admission.*

	6/18	6/22	6/26	6/30
Hemoglobin, gm./100 cc.	13.5	12.2	12.3	9.8
Sed. rate (Westergren), mm./hr.			93	
WBC, per cu. mm.	7100	4950	5200	
Segs; bands; juveniles, %	33; 9; 0		54; 2; 0	
Lymphocytes; monocytes; eosinophils; %	44; 13; 1		39; 4; 1	
Platelets, per cu. mm; reticulocytes, %	165,000; 1.7			
BLOOD CHEMISTRY:				
Glucose; urea N.; uric acid, mg./100 cc.	75; 4; 3.3	69; 5; —		
Na; K; Cl; CO_2, mEq./liter	142; 2.9; 98; 21	140; 3.1;100; 27	140; 3.2; 96; 28	
Ca; P, mg./100 cc.	7.5	8.8; 3.9	9.2; 4.5	
Albumin; globulin, gm./100 cc.	3.0; 3.6	3.2; 4.0	3.7; 3.2	
Bilirubin, total; indirect, mg./100 cc.	1.2; 0.3		0.7; —	
Thymol turbidity, units	10.8	11.6	12.5	
Cephalin flocculation, 24; 48 hrs.	1+; 3+	3+; 3+		
Cholesterol, mg./100 cc.	152			
Alkaline phosphatase, Bodansky units		13.9	12.8	
SGOT units	97	69	27	
Leucine aminopeptidase units		311		
Prothrombin time, control; patient secs.	13.5; 12.5			
Arterial pH		7.50		
pCO_2; O_2 sat.		37.5; 93.8		
URINE:				
pH; sp. gr.	7.5; 1.006		7.5; —	
Albumin; sugar	Neg.; Neg.		Neg.; Neg.	
RBC; WBC per HPF	Rare; 0-6		4-6; 0-2	
Na; K, mEq.	93; 12 per liter	29; 7 per 24 hrs.		
Sulkowitch test		Cloudy		
Stool guaiac	Neg.		Neg.	

episodes of shaking chills during this period.

Physical examination revealed a cachectic white woman with temperature of 103 F. pulse 108, and blood pressure of 96/50 mm. of mercury. The liver and spleen were enlarged. There were pallor of the mucous membranes and faintly icteric sclerae. Neurologic examination was normal except for hyperactive reflexes, generalized weakness, and a shuffling gait.

Laboratory findings for this second admission are reported in Table 2.

The hospital course was characterized by septic temperature with daily spikes to 103-105 F. and rapid return to almost normal levels. A liver scan was reported as "probably normal." An intravenous pyelogram demonstrated displacement of the left kidney to an inferior-lateral position by a 5 cm. extrinsic mass. Pararenal CO_2 insufflation confirmed that the mass was present and probably was retroperitoneal. Serum potassium was 2.0 mEq. per liter and calcium was 7.8 mg. per 100 cc. Trousseau's sign could not be elicited. The patient was started on oral potassium chloride. On the third hospital day, reports of two urine cultures showed *Aerobacter*, greater than 100,000 colonies, and the patient was started on kanamycin and colistin. Five blood cultures taken previously, and four obtained after this time were all sterile. On the fourth hospital day, the patient was noted to be confused transiently during a fever spike to 104 F. A lumbar puncture was performed, and there were no abnormalities in the cerebrospinal fluid. A cholecystogram failed to show the gallbladder. On the morning of the fifth hospital day, shock developed. Her hemoglobin had fallen to 6.4 gm. per 100 cc. without evidence of external blood loss. Treatment consisting of vasopressors, intravenous fluids, 20,000,000 units of penicillin per day, and two units of blood was instituted. Over the next 36 hours her condition continued to deteriorate. Blood pressure could not be maintained with levophed and steroids. She became acidotic, hyponatremic, comatose, and had two seizures characterized by general body rigidity and apnea. She died on the evening of the sixth hospital day.

TABLE 2.—*Major Laboratory Findings on Second Hospital Admission.*

	8/19	8/21	8/23	8/24
Hemoglobin, gm./100 cc.	8.2	6.9	6.4	11.4
Sed. rate (Westergren), mm./hr.	137	143		
WBC, per cu. mm.	15,000	10,000	17,000	12,900
Segs; bands; juveniles, %	70; 4; 0	48; 32; 0	62; 26; 0	27; 63; 2
Lymphocytes; monocytes; eosinophils, %	26; 0; 0	16; 4; 0	5; 6; 0	6; 2; 0
Platelets, per cu. mm; reticulocytes, %	275,000; 0.5	—; 0.7		
Mean Cell Volume	89			
BLOOD CHEMISTRY:				
Glucose; urea N.; Creatinine, mg./100 cc.	85; 6; 0.5	87; 11		
Na; K; Cl; CO_2, mEq./liter	135; 2; 85; 37	134; 3.5; 90; 32	127; 3.8; 91; 23	117; 43; 82; 13
Ca; P, mg./100 cc.	7.8; 2.8	7.8; —		
Albumin; globulin, gm./100 cc.	2.2; 3.3			3.0; 3.1
Bilirubin, total; direct, mg./100 cc.	3.4; 1.4			6.8; 4.4
Cephalin flocculation, 24; 48 hrs.	3+; 3+			
Alkaline phosphatase, Bodansky units	16.8			14.7
SGOT units	31			1115
Prothrombin, patient; control, secs.				25; 12.5
Arterial pH		7.58		7.58
pCO_2; O_2 sat.; CO_2 content		34; 95; 32		18; 96; 11
Amylase, Somogyi units				Less than 320
Iron, total; binding capacity, μg./100 cc.	19; 150			
URINE:				
pH; sp. gr.	7.5; 1.002			
Albumin; sugar	Neg.; Neg.			
RBC; WBC per HPF	0; 5			
Bile	Neg.			
Urobilinogen	4 plus			
VDRL	Neg.			
Febrile agglutinins	Neg.			
Cultures on stool, blood, spinal fluid	Neg. (repeated)			
Urine culture	*Aerobacter*, greater than 100,000 colonies (3 occasions).			
SPINAL FLUID:				
Chemistry and cell counts				Normal

Radiologic Findings

DR. CARTER: The initial examination of the chest on June 10, 1964, reveals a hazy, ill-defined infiltration at least partially superimposed upon the right hilar area. In the lateral view the composite hilar shadow does not appear prominent, and the infiltration is difficult to visualize. Films on June 25 show a decrease in the amount of infiltration, and now the margins of the lesion are sharply defined. In the lateral view the lesion appears to be located against the pleural surface in the superior segment of the right lower lobe. This change would be compatible with what one might expect to see with an organizing pulmonary infarct. There is no convincing evidence of a mass in the hilus (Fig. 1).

Films on July 2, after surgery, reveal pleural thickening and/or fluid in the right base. There is also evidence of atelectasis in the right middle lobe. On the films of the chest one cannot see elevation of the left hemi-diaphragm at any time.

The initial films of the abdomen on June 10 show both kidneys in normal position, the spleen normal in size, and certainly no mass in the left upper quadrant of the abdomen. This was also the case on Aug. 1. On Aug. 20, however, a large mass in the left upper quadrant is obvious and displaces the left kidney inferiorly. An excretory urogram done at this time shows the upper collecting systems to be normal. A retroperitoneal CO_2 study was performed and the mass was not outlined by the CO_2. I am not sure exactly what the mass is, but I cannot separate

a splenic shadow from the mass and therefore my first choice is that this is an enlarged spleen. In view of the clinical findings, I wonder about a splenic abscess. I cannot exclude a pancreatic pseudocyst (Fig. 2).

Clinical Discussion

DR. COMBES: In dealing with this complicated problem I should like to start with a brief consideration of disorders that cause weight loss in people who apparently eat well, as she claims she did. We would have to consider hyperthyroidism, a malabsorption state, or some neoplastic process. On the basis of physical findings, with absence of an enlarged thyroid gland, we can probably exclude hyperthyroidism. In a moment, we shall consider whether or not the patient had evidence of a malabsorption state, a neoplasm, and indeed whether malabsorption, if present, may have been secondary to a neoplasm.

I will not discuss the episode of hemoptysis. I do not know how to tie this in with the rest of the story, other than to say that the patient had a pulmonary embolism, and that pulmonary emboli occur frequently in patients with neoplastic disease.

The patient at the beginning had a 20-pound weight loss, with manifestations of tetany, and had hypokalemia and hypocalcemia, both of which improved significantly on diet alone. Apparently vomiting was not a cause of the hypokalemia. Laboratory data indicate that her kidneys were able to conserve potassium moderately well; thus, urinary loss of potassium seems unlikely. It would seem reasonable to consider that she lost potassium in her stools, perhaps as a consequence of a malabsorption state. The adequacy of food intake and how this may have contributed to hypokalemia cannot be assessed from the history. The findings with which she presented are consistent with a malnutrition state. She had hepatosplenomegaly. A liver biopsy, which I have not seen, was described as demonstrating cirrhosis and fatty changes. There is a history of chronic alcoholism. We have no idea how extensive this was. However, at least one sequence of events that could have occurred in a chronic alcoholic is recurrent pancreatitis, resulting in poor absorption, with significant losses of nitrogen, fat, potassium,

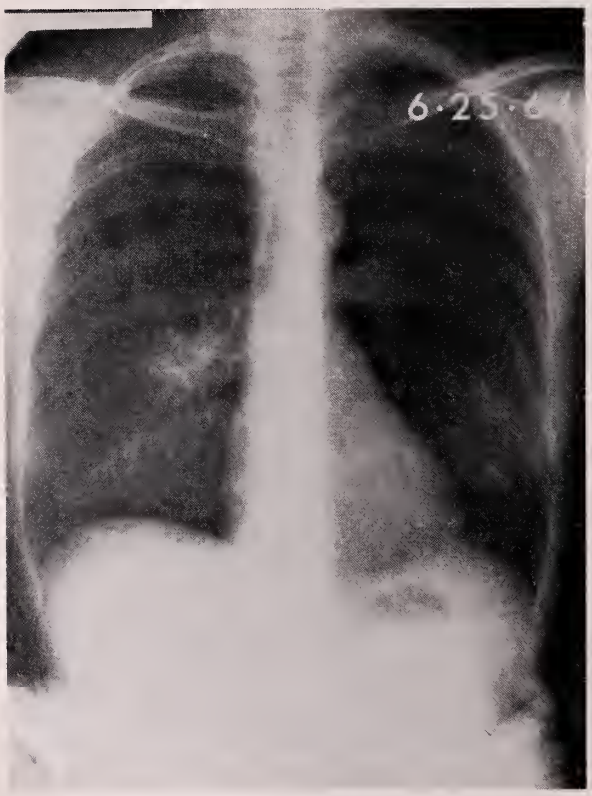


Fig. 1. Anteroposterior view of chest x-ray obtained during first hospital admission.

Fig. 2. Excretory urogram obtained Aug. 20 during second hospital admission.

and calcium in the stools. It is conceivable that some of her abnormalities would respond in the hospital to a good diet, particularly if diet had been somewhat inadequate before admission. Unfortunately, stools were not examined for fat. There are several things which do not fit in with this analysis of the problem, but at least let us work with it for now.

The patient felt better when she left the hospital at the end of her first admission. In the interim, she was seen in the liver clinic. There was nothing particularly new in her history then, and liver tests were consistent with cirrhosis. Finally the patient became anorectic and very weak, and developed a febrile illness accompanied by soaking sweats, shaking chills, leukocytosis, and anemia. One of our major problems is to determine the cause of fever and the progressive downhill course. The patient had cirrhosis. Cirrhotics are prone to develop certain serious infections, including miliary tuberculosis, histoplasmosis, and cryptococcosis. These could account for many of her findings. Such infections may involve the adrenals, and one might raise the question of whether adrenal necrosis could have accounted for shock and death. Against adrenal insufficiency is the absence of skin pigmentation, absence of hyponatremia until the day before death, lack of hyperkalemia or elevation of the blood urea nitrogen. I think we can exclude adrenal necrosis and Addison's disease therefore. The presence of disseminated infection with one of the organisms mentioned has to be considered, nevertheless. Another possibility, of course, is the presence of undrained pus. We have a mass in the abdomen, chills, and fever. In a patient with chronic pancreatic disease, pseudocysts may develop. These may become secondarily infected, and certainly one possibility is that she had a retroperitoneal abscess in the region of the pancreas. Other considerations include neoplasms. Lymphomas can produce this type of fever, commonly produce retroperitoneal masses and cause malabsorption with involvement of the intestines and mesentery. Early in her course, the patient had a high sedimentation rate and developed anemia findings commonly associated with lymphomas. Failure to find histologic evidence of lymphoma on her liver biopsy by no means excludes the diagnosis, since the liver is frequently uninvolved by the tumor. Finally, we should consider other neoplastic diseases, particularly pancreatic neoplasms which could account for most of her findings. This is as far as I can go with a differential diagnosis. I find it difficult to single out one of the possibilities. I lean to the presence of malabsorptive state due primarily to pancreatic disease. A chronic pancreatitis with pseudocysts that became secondarily infected, or a pancreatic neoplasm are my leading choices; lymphoma is a second choice.

DR. FORDTRAN: It would seem important to attempt to tie the early pulmonary embolus together with the more recent development of fever and cachexia, and a left upper quadrant mass and evidence of liver disease. Although I cannot reach a definite diagnosis, I think the best way to approach the problem would be from the standpoint of the mass. From the protocol, it seems that the mass more or less appeared suddenly. Dr. Carter has told us from his x-ray study that the examination of Aug. 20 is the first one that shows the mass to be present in the left upper quadrant. Therefore, between the two admissions she developed a large mass which pushed the kidney down. I think probably this is an enlarged spleen, and that we should try to think of some of the diagnoses which might account for sudden enlargement of the spleen. Pancreatic carcinoma comes immediately to mind because this could cause thrombosis of the splenic or portal vein and account for rapid enlargement of the spleen, and at the same time would account for her cachexia and for the preceding pulmonary embolus. Another possibility is acute pancreatitis. Although we do not have any direct evidence for this, the negative serum amylase levels certainly do not rule it out. Only about 80 percent of patients with acute pancreatitis have a high amylase, and occasionally this disease causes thrombosis of the portal vein with rapid enlargement of the spleen. Another possibility for rapid enlargement of the spleen might be, as Dr. Carter has suggested, a splenic abscess. So in summary, I would say that the mass is probably a large spleen, and my best bet would be that it was caused by thrombosis of the splenic or portal vein, most likely secondary to pancreatic carcinoma.

DR. MADISON: In a sense we are victims of the protocol. I think that the available information can be tied together in much the same manner as the other discussants. A pancreatic neoplasm could explain

the excessive weight loss, which is a common manifestation and is not always accompanied by jaundice. Certainly weight loss of 20 or 30 lbs. in a short period of time is not uncommon with pancreatic carcinoma. Pancreatic carcinoma could also have been responsible for pulmonary embolism, as was mentioned. Not uncommonly, in about 25 percent of the patients, pancreatic carcinoma leads to splenomegaly due to thrombosis of the splenic vein. The mass could have been part of the pancreas or the spleen. Pancreatitis associated with pancreatic carcinoma is not uncommon, and may have been responsible for the lowered serum calcium; or this may have been lowered because of both pancreatitis and malabsorption. Pancreatitis could account for potassium loss and alkalosis, which were noted at one point. So I think perhaps one could, if he wished, tie all of these loose ends together with a pancreatic neoplasm, perhaps with a cyst that formed distally in the pancreas and associated pancreatitis.

When I saw the patient, she was being treated at that point with vasoconstrictors because she was in collapse, but she also appeared to me to be severely volume-depleted. As a matter of fact, after receiving some 4 or 5 liters of intravenous fluids there was not a trace of edema in the patient. I think that this eventually led to a worsening, perhaps, of her liver condition; the jaundice increased; the serum glutamic oxaloacetic transaminase became greatly elevated; and I think the acidosis at the end was a severe lactic acid-acidosis associated with the administration of vasoconstrictor agents in a patient who had a volume deficit.

DR. BARNETT: At the time of final admission, the diagnostic possibilities in this patient were numerous. As has been pointed out, the progressive weight loss and weakness culminating in a hectic and febrile course could well be explained by neoplasm with recent metastases or by disseminated granulomatous disease, either tuberculosis or histoplasmosis. An alternate possibility would be that her course of gradual debility was related to alcoholism, malnutrition, and cirrhosis, and that the terminal four weeks represented intra-abdominal suppuration. The point to be emphasized is that the course and physical findings would be consistent with either of these diagnostic possibilities, and unfortunately, there is no *in vitro* test which could quickly and dependably make the distinction. In clinical circumstances such as these, the application of Sutton's rule would seem potentially rewarding. The presence of an intra-abdominal mass was detected fairly early in her last hospitalization, presumably at a time when a limited exploratory laparotomy might have been tolerated. Knowledge of the nature of the intra-abdominal mass would have enabled one to select more specifically a course of therapy.

DR. SELDIN: There are three causes for hypercalcemia in alcoholics. Steatorrhea has already been mentioned as a possible cause of hypocalcemia in this patient. One would have to postulate that the patient had developed sufficient pancreatitis secondary to alcoholism to produce a malabsorption syndrome. The likelihood of steatorrhea being the factor responsible for hypocalcemia in this particular patient is very small. No history of steatorrhea was obtained. The x-ray disclosed no calcification of the pancreatic bed. Finally, severe hypocalcemia is much more common in idiopathic sprue than in pancreatogenous steatorrhea. It seems reasonable to assume that if the patient had sufficient pancreatitis to produce steatorrhea severe enough to cause hypocalcemia, the other clinical features of malabsorption would have been apparent.

A second cause for hypocalcemia in alcoholics is acute pancreatitis. In this instance, there is a rapid movement of calcium into the pancreatic bed and into the peritoneal fat, which has been digested as a result of the liberation of pancreatic enzymes. The absence of symptoms of pancreatitis and the normal amylase militate strongly against this diagnosis.

The third cause of hypocalcemia in alcoholics is alcoholism *per se*. It has now been demonstrated that alcohol accelerates the renal excretion of both calcium and magnesium. It is highly probable that the frequent observation of hypocalcemia in alcoholics immediately on admission and the rapid return of the serum calcium to normal during the first week of hospitalization is attributable to the effects of alcohol. I suspect that the cause of this patient's hypocalcemia is accelerated renal excretion of calcium secondary to alcoholism.

It should be pointed out that hypocalcemia and hypokalemia were simultaneously pres-

ent. Hypokalemia tends to counteract the effects of hypocalcemia so that even when the serum calcium is previously depressed, tetany may not develop. If the serum potassium is restored to normal while the serum calcium remains depressed, tetany may then supervene. The hypocalcemia was not particularly severe in this patient, so that I would suspect that the positive Trousseau sign was principally an expression of respiratory alkalosis.

Pathologic Findings

DR. STEWART: Review of the lung segment removed surgically at the first admission reveals that the lesion was actually an acute and chronic abscess, apparently originating in an area of saccular bronchiectasis. The abscess cavity was partially lined by respiratory epithelium and partly by squamous epithelium (Fig. 3). The presence of this epithelial lining suggests that saccular bronchiestasis possibly preceded the abscess. Granulation tissue and fibrosis were observed in the surrounding lung tissue. Based on the type and duration of the inflammatory and reparative processes in the abscess wall, it appears to me that sometime in March or April she may have aspirated something or that a small area of existing bronchiectasis became infected, leading to the formation of an abscess.

This patient's course reveals that abscesses may metastasize and prove lethal just as do neoplasms. The autopsy findings showed that the infection had disseminated to the brain and to the spleen. Multiple abscesses were found throughout the brain (Fig. 4). The largest was located in the left frontoparietal region near the internal capsule and measured about 3 cm. in size. Some of the brain abscesses appeared to be several weeks in duration, while others were more acute. Bacterial colonies were found in the inflammatory exudate. Both gram-negative rods and gram-positive cocci were present (Fig. 5).

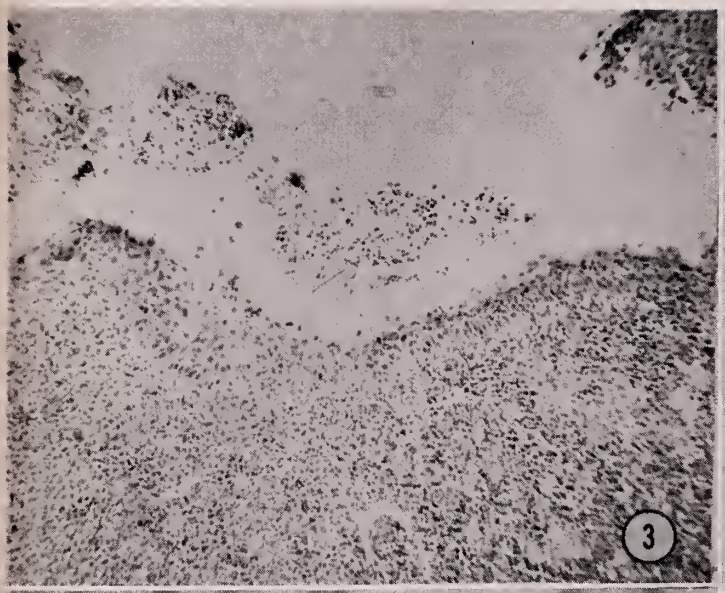
The large mass in the left upper abdominal quadrant was a splenic and perisplenic abscess (Figs. 6 and 7). Multiple abscess cavities were present in the spleen, and the spleen was surrounded by a loculated collection of pus. The splenic and perisplenic abscesses were acute and chronic in nature, and probably these lesions began sometime in June or July. The very thick "pyogenic" membrane denotes that it is of considerable duration, at least four to six weeks. Many of the splenic abscesses freely communicated with the perisplenic locule, indicating that the abscesses were originally splenic in location, and then extended through the capsule. There was also septic thrombophlebitis of the splenic vein. This thrombus extended along the splenic vein and into the portal vein (Fig. 8), and further into the portal vein branches throughout the liver (Fig. 9). In the liver there was acute inflammation with multiple small abscesses, which had formed in the connective tissue of the portal tracts (Fig. 10). The bile ducts were not involved. Many of the abscesses had the typical leaf-shaped appearance of acute hematogenous hepatic abscesses (Fig. 11).

In times past, the most frequent cause of thrombophlebitis of the portal vein (thrombopylephlebitis) has been spread from an infected appendix. With the advent of improved surgical methods, antibiotics, and rapid diagnosis this route has become rare. The most frequent pathogenesis of thrombopylephlebitis in recent years has been progression from infection of the colon and rectum (perirectal abscesses, diverticulitis or ulcerative colitis) to thrombophlebitis of the inferior mesenteric vein, and subsequent propagation into the portal vein and liver. A less frequent mechanism of thrombopylephlebitis is progression of the infection down the splenic vein from hematogenous splenic abscesses, as we have in the patient being discussed today (Fig. 12).

The patient indeed did have early Laennec's cirrhosis with intense fatty metamorphosis (Fig. 13). During the terminal episodes of shock, from which she suffered, centrilobular necrosis developed.

The heart was atrophic, but otherwise normal. An area of acute erosion of the gastric mucosa was found immediately adjacent to the large splenic abscess, which protruded against the posterolateral gastric wall. At this site, the gastric wall was thinned by the encroaching inflammatory reaction, and it was at this site in the gastric mucosa that the superficial area of ulceration of the stomach had occurred (Fig. 14).

The lungs were acutely congested. Examination of the area in the lung from which the lesion was surgically removed showed only acute congestion and pulmonary edema.



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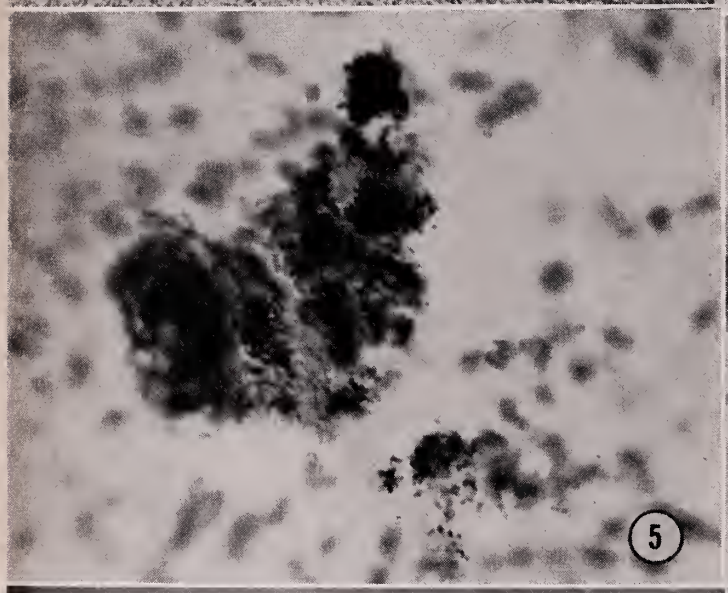

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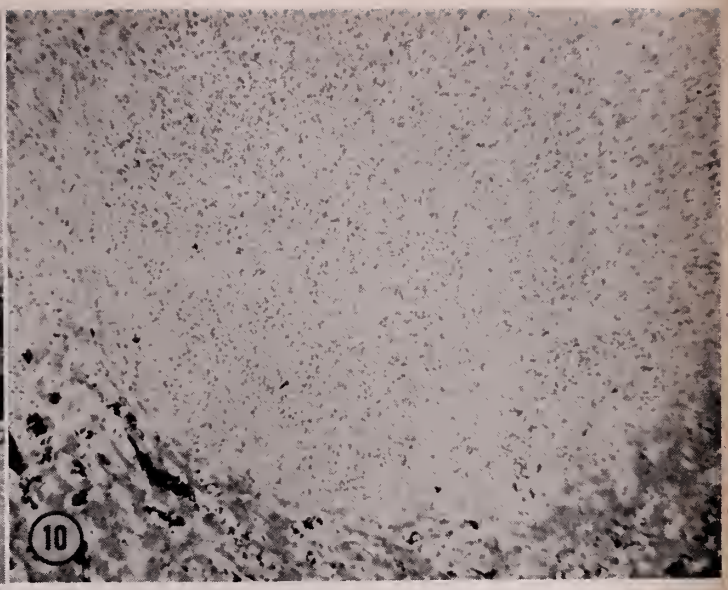


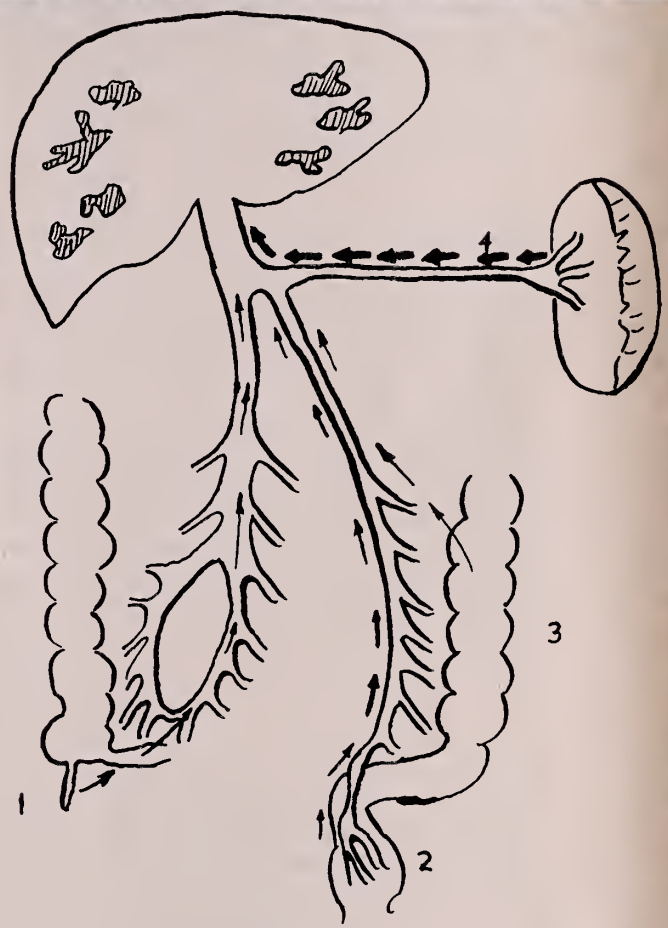


Fig. 12.

Fig. 3. Photomicrograph of the surgically resected pulmonary lesion. The pulmonary abscess is shown. It probably originated either from an area of saccular bronchiectasis or as the result of aspiration. Note the squamous as well as the respiratory epithelium lining a portion of the cavity. Fig. 4. Gross photograph of one of the brain abscesses. Multiple brain abscesses were present from dissemination of bacteria from the pulmonary abscess. Fig. 5. A gram stain revealing bacterial colonies in the brain abscess. Both gram positive cocci and gram negative rods are present. Fig. 6. Gross photograph of the large left subdiaphragmatic abscess surrounding the spleen. Fig. 7. Gross photograph of the spleen showing abscesses which had eroded through the splenic capsule and were continuous with the perisplenic abscess. Fig. 8. Gross photograph showing the portal vein opened just before its entry into the liver. A large septic thrombus occupies the vein and protrudes from the lumen. Fig. 9. Photomicrograph of liver. A large portal radical is shown in which the portal vein is filled with a septic thrombus. Note the inflammatory exudate in the wall of the portal vein and surrounding it. Fig. 10. Photomicrograph of one of the liver abscesses. The abscess is continuous with the inflammatory process in the portal vein. Fig. 11. Gross photograph revealing thrombophlebitis of a medium sized tributary of the portal vein. To the left are "leaf-shaped" liver abscesses. The abscesses follow the portal venous system and extend outward into the surrounding liver. Fig. 12. Diagram indicating that pylethrombophlebitis most commonly originates from one of four regions: (1) appendix (acute appendicitis with rupture and formation of a periappendiceal abscess), (2) anorectal region (perirectal abscess), (3) sigmoid and descending colon (diverticulitis or ulcerative colitis), and (4) pancreas and spleen (splenic abscess), as in our case. Arrows represent route of infection into the portal vein and subsequently into the liver.

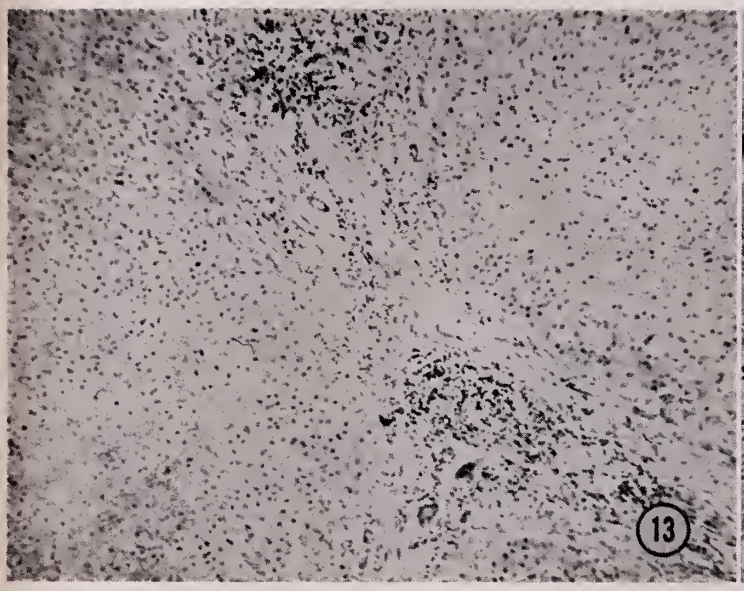


Fig. 13. Photomicrograph showing Laennec's cirrhosis of the liver. Portal fibrosis, bile canalicular proliferation, portal chronic inflammation, septation of the lobule with loss of central vein and formation of a regenerative liver nodule.

Fig. 14. Photomicrograph of the gastric erosion caused by the protruding subdiaphragmatic abscess into the left posterior lateral portion of the stomach.

No evidence of residual lung abscess or surrounding pneumonitis was found.

Bacteriologic studies carried out from the brain and splenic abscesses revealed *Pseudomonas aeruginosa,* which grew out very heavily.

In summary, the pathological studies indicate that this patient had long standing Laennec's cirrhosis which was still in a fatty stage. This is believed to have been due to a nutritional deficiency, compatibly related to chronic alcoholism. Sometime prior to the first admission she developed a pulmonary abscess, which we believe originated either as an infected bronchiectatic cavity or following aspiration. Probably even before this lesion was removed surgically, dissemination of the infection occurred. Then, with the lapse of several weeks, splenic and brain abscesses began to enlarge and extend. The splenic infection then spread, through splenic and portal thrombophlebitis, to the liver, causing multiple hematogenous hepatic abscesses to form. It appears that overwhelming infection with accompanying shock was the main mechanism causing death.

New Test for Syphilis

A new screening test for syphilis was recently reported at the annual meeting of the American Public Health Association. Claimed faster, economical, and more accurate than the standard test, the new test is known as the macroscopic latex test.

Its basis is the fact that antibody-like material in syphilitic blood agglutinates the latex particles coated with cardiolipin, fats extracted from fresh beef hearts.

The new test can be read within two minutes after two drops of blood serum are mixed with one drop of the cardiolipin-coated latex antigen. The standard test takes 30 minutes. The new test was found to be 99.8 percent accurate; the standard test, 98.2 percent accurate. The new test was reported by Kenneth A. Borchardt, Ph.D., of Letterman General Hospital, San Francisco.

Clinicopathologic Conference

George W. Zeluff, M.D.; Dolph L. Curb, M.D.; R. S. MacIntyre, M.D.;
Don W. Chapman, M.D.; R. D. Pruitt, M.D.; Philip C. Johnson, M.D.;
John Kelsey, M.D.; and Thomas L. Linn, M.D.

Baylor University College of Medicine

Protocol

History.—This 78-year-old, married white woman was admitted to the Methodist Hospital on July 11, 1962, complaining of recurrent sharp abdominal pain of four weeks' duration, usually occurring promptly after meals. Pain began in the right upper quadrant with radiation to the angle of the right scapula, and several times the patient required "hypos" for relief. She denied food intolerance and there was no associated nausea or vomiting, jaundice, dark urine, or pale stools. The pain never woke her at night. She gave no history of heart trouble or high blood pressure and was fully active with no dyspnea on exertion or angina. Thirty years ago she had had gallbladder drainage and for seven years she had been on thyroid replacement therapy, following thyroidectomy.

Physical Examination.—Blood pressure 120/70 mm. of mercury, pulse 76 and regular, respirations 20, temperature 97.8 (oral), height 5 feet 4 inches, weight 120 pounds. Neck, thyroidectomy scar; lungs, clear; heart, normal sinus rhythm with grade 1 basal systolic murmur (ejection type)—A-2 greater than P-2; abdomen, tender right upper quadrant with no palpable gallbladder and no organs or masses felt. The examination otherwise was noncontributory.

Diagnostic Studies.—The chest x-ray showed slight cardiac enlargement, and the cholecystogram showed multiple small radiolucent stones in a well-visualized gallbladder. Urinalysis showed an acid urine with specific gravity of 1.011, 30 mg. (24 hr. specimen) of protein, negative sugar, and packed WBC's with many bacteria. Hematocrit was 35 per cent, and white blood count 6,300 with normal differential. Sedimentation rate was 19 mm. per hour; fasting blood sugar, 105 mg. per 100 ml.; cholesterol, 250 mg. per 100 ml.; protein bound iodine, 3.8 μg. per 100 ml.; and VDRL, nonreactive. Myocardial blood flow was reported as normal. Electrocardiogram (ECG) July 11 showed nonspecific ST and T wave changes. Repeat ECG July 12, taken during her "usual pain," showed first degree A-V block with nonspecific ST and T wave changes. The P-R interval had increased from 0.18 to 0.22 seconds since the previous tracing.

Course in Hospital.—A cholecystectomy was planned for July 16. Before surgery, the patient experienced her usual "gallbladder" pain. At 8:30 a.m. July 12, she complained of chest pain after eating, but was relieved by A.S.A. Again at 3:35 a.m. July 14, she complained of pain in her back and arm,

Dr. S. H. Frazier, professor and chairman of the Department of Psychiatry, Baylor University College of Medicine, is guest editor for this clinicopathologic conference from that school. Dr. Pruitt is professor and chairman of the Department of Medicine; Drs. Zeluff, Curb, Johnson, and Kelsey are associate professors of medicine; and Dr. Chapman is a clinical professor of medicine. Dr. MacIntyre is a clinical assistant professor of radiology in the Department of Pathology, and Dr. Linn is a clinical assistant professor of pathology.

History and diagnostic studies indicated cholecystectomy for the patient. She had increasingly severe "gallbladder" pain until the time of surgery; conditions seemed to warrant no postponement. Pentothal Sodium was given, and the patient's heart stopped. Open heart massage was successful; shortly afterward, respiratory difficulty developed and the patient died.

stating specifically that this was her "gallbladder" pain again. It was again relieved by A.S.A. Once more she complained of similar pain at 7:45 p.m. July 14. This required Darvon for relief. The following day at 7:45 p.m. she again had pain, requiring Demerol for relief. The morning of surgery (July 16), severe chest pain recurred, and preoperative sedation gave no relief. Later, in the operating room corridor prior to surgery, she remarked to the anesthesiologist that she "felt like she was going to die." Examination at this time showed a normal blood pressure and a full regular pulse. The pain was thought to be of biliary origin and it was decided to proceed with surgery as planned. Immediately following administration of 180 mg. of Pentothal Sodium intravenously, blood pressure fell to 0. Vasopressors and external cardiac massage produced no immediate response. With artificial respiration through an endotracheal tube, open heart massage was started and adequate circulation immediately reestablished with a normal rhythm and a blood pressure of 110/60 mm. of mercury. The patient was transferred to the intensive care unit. On arrival there she was awake, talking coherently, and moving all four extremities. She then developed some respiratory difficulty which was unrelieved by 7.5 grains of intravenous aminophylline. Her color became poor, pulse disappeared, and blood pressure fell to 0. Open heart massage was unsuccessful and the patient died at 12:05 p.m.

Discussion

DR. GEORGE W. ZELUFF: We have an interesting CPC and we will assume that the audience has read the protocol. Dr. Curb will initiate the discussion.

DR. DOLPH L. CURB: The problem here is not so much why this lady died, for it is obvious the immediate cause was cardiovascular collapse—at least the "first" time she "died." The mechanism of the second is speculative, although bronchial spasm or obstruction probably played a major role. As to primary pathology, I believe in this case the pathologist can be trusted not to turn up with some mysterious or unsuspected condition such as ruptured aneurysm, fulminant pancreatitis, or massive pulmonary embolism as the reason for the cardiovascular collapse. Rather, I feel the fatal outcome centers around one or the other of two major problems, that is, gallbladder and heart disease, and their interrelationship. Which one of these was responsible for the symptoms that led to her admission and the decision to operate? Were the previous symptoms the same as those immediately prior to surgery, or was the final clinical episode of different pathologic orign?

Now, back to the protocol.

The initial description of the pain specifies location in the right upper quadrant, with radiation to the angle of the right scapula. The surgical consultant, in additional notes, describes the pain as lower of midsubsternal and refers also to pain in both shoulders, but more on the right, with the patient describing it as a "sensation like the food has stopped." Also, although no previous history of heart trouble is admitted, there is an additional chart statement that she was "believed at home to have had heart trouble." Among these notes are minor historical distinctions which we may have to pay particular attention to if objective findings fail to help in patient evaluation. At this point, Dr. MacIntyre, will you please show the x-rays?

DR. R. S. MACINTYRE: Routine posteroanterior and lateral chest films show slight cardiac enlargement, mostly left ventricular. The aorta is elongated with some arch calcification, and there is good gallbladder visualization with multiple radiolucent calculi present.

DR. CURB: We now have objective evidence of two major problems, arteriosclerotic heart disease and gallbladder disease with stones. I would now like to ask Dr. Chapman to discuss the ECG findings. Perhaps he should know first that I will use his interpretation in later discussion; this may allow me to put the blame on him if I guess incorrectly!

DR. DON W. CHAPMAN: This ECG shows a normal sinus rhythm. The P-R interval is approximately 0.20, the QRS 0.09, the QT 0.42-0.44, and there is a minor ST segment change in leads I and II. The precordial leads show minor ST segment depression in V-4, V-5, and V-6 compatible with digitalis effect or subendocardial injury, but may represent nonspecific changes. The overall QRS complex is normal. The R wave is low in V-2 and V-3, is absent in V-1,

and is large in V-4. This suggests an old anteroseptal infarction.

The second ECG again shows ST segment change, nonspecific, as if from digitalis effect. Precordial leads are the same as in the first. The P-R interval is now 0.23-0.24, suggesting prolongation and a possible first degree atrioventricular heart block. There is a small Q wave in AVL.

DR. CURB: The small increase in Q in AVL—would you consider that significant?

DR. CHAPMAN: Let's go back to the first tracing for a moment. Sometimes with a lateral wall infarction a tiny Q wave in AVL may develop. There is no Q wave in V-6, but in the second ECG tracing a definite Q wave has developed in AVL. Dr. Pruitt, would you call it a recent lateral wall infarction on the basis of these findings?

DR. R. D. PRUITT: The fact that there is a Q-S deflection in AVR in both records would lead me to think that this was unreliable evidence of lateral wall infarction. Nevertheless, one must admit that the findings in the right precordial leads, plus the intermittent Q in AVL, suggest the possibility of scarring in the anterolateral wall. This, however, is not as clear-cut as one would like.

DR. CURB: Thank you. It seems, therefore, that the electrocardiographers, even with hindsight, do not recognize any specific warning of serious or impending heart disease. Now, among the laboratory findings, some abnormalities such as pyuria and proteinuria, I believe, are of no significance except as potential postsurgical complications had she survived. Neither do I believe the slight depression of PBI significant in the outcome. Before interpreting the myocardial blood flow studies, I will ask Dr. Johnson for his evaluation of this.

DR. PHILIP C. JOHNSON: As to the significance and interpretation of the myocardial blood flow studies, one must understand that this radioisotope technique is still in the developmental stage. In the past two years, I have made many changes in the technique to a point where I believe the results are now fairly reliable and clinically useful. Since we measure total coronary flow, our results should relate more often to symptomatic heart disease or the ability of the heart to perform work than to ECG findings. The myocardial blood flow from this patient is within our normal range. This suggests that severe coronary artery disease restricting coronary blood flow is not present.

DR. CURB: So far, we have not received much objective help for the question, "Why did this lady die?" although the patient herself may have tried to answer. She said she felt like she was "going to die." It has been said that one should pay particular attention to such premonitions, presuming some of these patients might actually die of "fear." Bearing on this, some years ago at a medical meeting Dr. Frank Lahey denounced this kind of explanation of death, "People do not die because they are afraid they will die or say they are going to die; they do die because something is wrong with them and it is up to us to find out what it is." One should bear in mind, however, that a premonition of death may be part of the "alarm reaction" caused by some catastrophic visceral derangement such as massive hemorrhage with shock. In this case, attention was paid to the particular complaint.

Following careful examination, and in the presence of normal heart action, blood pressure, and a good full pulse, the symptoms at this point were considered to be from gallbladder colic, and were interpreted as additional indication to proceed with surgery. The subsequent course seems to indicate that this interpretation was wrong, and also the surgeon's retrospective chart record states that she was probably suffering from severe myocardial ischemia, if not actual infarction, during the immediate period before surgery. However, I have read the entire record carefully and my honest, objective opinion is that at the time I would have made the same decision on the basis of the evidence available. In other words, if the pain were due to an impacted cystic duct stone of long duration, as suggested by the history, there was certainly an excellent chance of impending gallbladder obstruction with gangrene, empyema, or localized or generalized peritonitis as a major complication. This might then be more disastrous than the increased risk of surgery. On this basis, good judgment seemed to dictate that surgery should not be canceled.

Now, with hindsight, I suspect this patient did have *two* types of pain. The original pain pattern was most likely caused by gallbladder colic, but later in the course of the disease it was supplanted by pain of car-

diac origin. The transition escaped recognition, perhaps because the patient herself insisted on several occasions that "it was the same gallbladder pain" she had always had. One possible point of significance is the pain radiation to the arm, particularly *if* it were the left arm, which was not specified; this would be unusual for biliary colic. Also, the alteration in the second ECG, although slight, might have indicated the need for further serial ECGs. The mechanism of cardiac arrest I will leave for other discussants, and will confine my final remarks to the possible relationship between gallbladder and heart disease, in which I developed an interest many years ago.

The idea of a "gallbladder heart" (heart disease caused by gallbladder pathology) is an old one. It begins with the empirical observations of authorities such as Drs. Mayo and Babcock, who removed gallbladders in patients with angina or other manifestations of heart disease, and saw the patients' cardiac status improve. Such empirical observations are difficult to confirm scientifically, but the idea has persisted. One valid observation is the statistical fact that there is a somewhat greater-than-chance coincidence between arteriosclerotic forms of heart disease and gallstones. This finding, in clinical case series, x-ray series, and autopsy series, may indicate some etiologic overlap in metabolic background of both conditions. Also, experimentally in animals and humans, one can at surgery produce mechanism disturbances in ECG, blood pressure, pulse, and cardiac dynamics by gallbladder stimulation such as pulling, pinching, distention, or spillage of bile into the peritoneal cavity. Therefore, disturbance of cardiac mechanism or function may be mediated through the autonomic nervous system, originating from the gallbladder. Applied to this patient, the gallbladder pathology, for example a cystic duct stone, may have stimulated the ECG abnormality and possibly sensitized the heart to a fatal rhythm disturbance, such as arrest.

Finally, in my own practice, I have seen several patients similar to this one with known gallstones and known anginal coronary artery disease, in whom specific pain distinction between these two conditions was extremely difficult. One particular patient, seen many years ago, showed this very problem. She was a poor surgical risk, but pain was so frequent and so incapacitating that the risk was taken. Following successful cholecystectomy, she had prolonged and gratifying relief of angina for some two years, after which it recurred, finally with a fatal heart attack.

DR. ZELUFF: Thank you, Dr. Curb, for your excellent discussion. Are there other comments on this case? Dr. Kelsey?

DR. JOHN KELSEY: The role of primary discussant in a CPC is always difficult and requires much preparation. That of the secondary discussant is easier since the difficult part of the conference is over and available to me as a member of the audience. In a CPC, to serve a teaching function, one must appear to criticize the clinical approach to the case in question. This I plan to do—briefly.

The reason for the cholecystostomy and drainage 30 years previously is not known. It is known that if the patient with stones has drainage only, without cholecystectomy, the likelihood of future gallbladder disease is very real, and even greater than in a patient known to have a silent gallstone. Equally, cholecystectomy in a patient in good physical condition carries a risk of probably less than 1 per cent, in contrast to a surgical risk perhaps as high as 20 per cent following emergency surgery in the presence of serious gallbladder disease. Also, age carries a greater surgical risk, but does not necessarily eliminate surgery even in the face of possible heart disease. It does require careful planning and perhaps prophylactic digitalis before surgery even though there is no definite indication for such. Prophylactic cortisone therapy may also need to be considered in older patients, particularly those who have been ill. The latter therapy of course is empirical, is geared to individual patient problems, and is not necessarily applicable to this case.

Looking back—as one not faced with the immediate decision, nor in the operating room corridor witnessing the persistence of pain and the sense of impending doom—perhaps surgery should have been canceled. However, a severe attack of gallbladder colic carries a positive danger, particularly in patients with coronary artery disease. An additional attack of colic may then be equally as serious as the surgical procedure.

DR. ZELUFF: Thank you, Dr. Kelsey,

for your interesting remarks. Is there a final comment before the pathologic revelation?

DR. CHAPMAN: We should remember the ECG tracings are dated the 11th and 12th of July and the patient died on the 16th. She complained of severe substernal distress on the morning of surgery and told a physician she was about to die. We have all repeatedly seen patients with severe coronary artery disease and known that this type of comment is frequently heard. Perhaps, looking back, another ECG might have shown further credence of a lateral wall infarction. We have often seen minimal changes initially, such as slight prolongation of PR interval with a little Q wave development in AVL, and in a tracing obtained three or four days later, these changes may be much more pronounced. Perhaps an enzyme level stat on the morning of surgery, with repeat ECG prior to surgery, might have shown that this patient did have a myocardial infarction in the process of development.

DR. ZELUFF: Thank you, Dr. Chapman. Dr. Pruitt?

DR. PRUITT: I believe the indications here are that the patient died a mechanism death, with catastrophic arrhythmia or standstill, probably the former; that she had a prolonged PR interval, and that the indication was for anticholinergic therapy, if anything, pharmacologically. Therefore, John, I must disagree with the issue of digitalizing the patient, since this might have further impaired the atrioventricular conduction.

DR. ZELUFF: Thank you, Dr. Pruitt. Dr. Linn will now present the pathology.

DR. THOMAS L. LINN: I decided this time to choose a "simple" problem for CPC. The discussants have illustrated just how simple this problem is, and have nicely covered the clinical differential issues. Now this case could be closed by labeling briefly the gallbladder disease, heart disease, and anesthetic death, but the processes involved demand more adequate definition.

First, the patient was old, with old coronaries. The intense severity of old coronary arterial disease may well explain why some patients live to be so old. Disease develops slowly, is nursed carefully over a long period of time, and demand redistribution of the myocardial vascular supply and blood flow occurs. A section of coronary artery from this patient showed massive lumen reduction by severe calcified atheroma. This diffuse process produced pipe stem vessels, sometimes with no visible lumen, most often with a very small central or peripheral channel. Absolute thrombotic occlusion is seldom demonstrated in such cases, unless one injects the coronary system with radiopaque material.

The heart, 375 gm., showed left ventricular hypertrophy with a large healed anteroseptal infarct involving the distal anterior two-thirds of the septum and the anterior wall including the apex, where it was circumferential. It also showed moderate aortic valvular disease of the type one finds in elderly people, with some cusp rigidity, slight nodular calcification deep in the sinuses, and slight incompetence. I think this is simply a degenerative atheromatous process similar to that seen in major vessels. There also was faint suggestion of a focus of recent infarction in the posterior basal left ventricular wall.

The healed infarct contained large endothelial vessels which carried a significant part of the cardiac blood flow. In other areas there were large stellate foci of replacement fibrosis representing so-called "falling-out" necrosis. This change, midway between acute infarction and slow replacement fibrosis, is most commonly seen in diabetics, and indicates small vessel disease of considerable severity. In this patient these areas were highly vascular, organizing, and contained scattered dead or dying residual muscle. This process had been slowly progressive over a long period of time, with gradual replacement of the necrotic foci by active granulation tissue. It was probably the major pathologic process; it correlates precisely with the vascular disease, and nicely explains the complex clinical story. Other foci showed progressive pyknotic abnormality produced also by severe ischemia. So far, we have evidence of a slow buildup of extensive changes without as yet frank infarction, and of the significant surgical risk in such patients even in the absence of absolute myocardial infarction. However, as the logical end point of this process, in some sections we did find a large acute myocardial infarction.

In summary, she showed severe coronary vascular disease producing continuous muscle damage, the most obvious being the old infarct. The minor but progressive damage was represented by continuous, active, "falling-out" necrosis, progressing to fibrous replace-

ment, but in some areas still active. The recent infarct was approximately 10 to 18 hours old, which coincides nicely with the episode of pain which occurred the night before surgery, was not relieved by sedation, and persisted into the morning of surgery. Despite this, it is interesting to remember that immediately prior to surgery, blood pressure was normal and the pulse full and regular. This may label the acute infarct as relatively "minor" terminal pathology.

Fig. 1 shows a portion of gallbladder and cystic duct. The contained stones, through drying, show beautifully how they are formed, and one single stone is loosely impacted in the first compartment of the cystic duct. Discussion of this finding is crystallized in the phrase, *"Res ipsa loquitur."*

Concerning the final mechanism of death —the first death—the anesthesiologists tell me that most anesthetic drugs, except ether, are powerful parasympathomimetic agents, and will produce bradycardia and hypotension. Apparently the original issue with Pentothal was that, in excessive dosage, this effect went unrecognized and hypoxia then occurred, with death in asystole. Also, all anesthetic agents are eventually myocardial depressants, but I do not believe this was an issue in this patient. The anesthesiologist believed that cyclopropane first, to eliminate the possible effect of Pentothal, followed by muscle relaxants for intubation, would promptly have created the same problem. His final comment was, "Pentothal was easier, more rapid, and much more efficient." The other pertinent comment was that with open heart massage, the heart responded immediately without pharmacologic assistance,

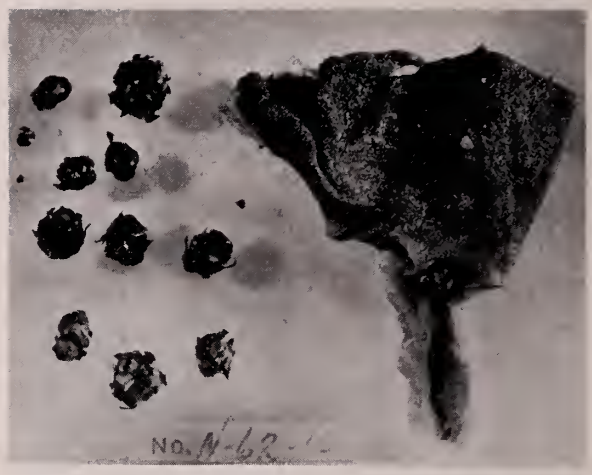


Fig. 1. Portion of gallbladder with stones. Note single stone impacted in beginning of cystic duct.

and afterward the patient was awake, alert, and complaining bitterly of pain; she had a clear sensorium and a good pulse and blood pressure despite the established infarction.

One final point: Apprehension, touched upon several times in this discussion, can be a significant issue in appropriate patients. It produces an endogenous or epinephrine effect, but the patient always shows fibrillation, indicating an irritable myocardium. In contrast, I would agree that this patient died in asystole.

DR. ZELUFF: Thank you, Dr. Linn, for your usual dynamic comments. Are there any final remarks, which would perhaps be anticlimactic at this point?

I would like to thank the participants in this discussion, particularly Dr. Curb for an excellent presentation, and Dr. Linn and Dr. Kelsey for their contributions.

♦ The authors. Baylor University College of Medicine, 1200 M. D. Anderson Blvd., Houston.

NEWS

FIVE SPECIAL SPEAKERS NAMED FOR JANUARY MEET

TEXAS HOUSE of Representatives Speaker Byron Tunnell of Tyler, Dr. Ever Curtis of Gloucester, Mass., a California public relations man, Joseph E. Burger of Pasadena, Dr. Jack Schrieber of Canfield, Ohio, and the Rev. Walter Courtenay, pastor of First Presbyterian Church, Nashville, Tenn., are scheduled to speak to members of Texas Medical Association on Jan. 16. The occasion will be the Conference on Legislation to be held in Austin.

A Symposium on State Legislation of Medical Importance has been scheduled for the afternoon of Jan. 16. Speaking on the topic of "Mental Health and Mental Retardation" will be Dr. P. C. Talkington of Dallas. Dr. Charles A. Lemaistre of Dallas will discuss "Tuberculosis." "Implementation of the MAA Program Under the Kerr-Mills Act" will be presented by Dr. Joe T. Nelson of Weatherford; "Lay-Owned Laboratories," by Dr. Floyd Norman of Dallas; and "Anticipated Legislation Calling for the Licensing or Certification of Various Health Groups," Philip R. Overton, Austin. Also to be discussed will be the battered child syndrome and thallium poisoning.

A Symposium for County Medical Society Officers will be held during the morning of Jan. 16. There will be two sections, one for county society secretaries, the other for presidents and other officers. The secretaries will have opportunity to hear about the responsibilities of their office in regard to forms, reports, and other items. The presidents will hear a presentation on the various obligations of that office.

Voters Endorse MAA

BETTER than two out of every three Texans voting on Nov. 3 cast ballots for Amendment Three, thereby issuing a mandate to the next State Legislature to implement the second phase of the Kerr-Mills program.

Endorsement of extended medical care for the aged was measured by an unofficial count of returns showing 995,197 for and 406,769 against the amendment. Percentagewise, this is approximately 71 percent for and 29 percent against.

Further indication of the strength of the TMA-supported proposal was shown by the fact that of three amendments on the ballot, Amendment Three carried with the largest majority. Outdistanced by the medical care amendment, although racking up sizeable majorities, were amendments regarding the permanent school fund and conservation districts which carried about two to one.

Texas Medical Association has long supported the extension under the Kerr-Mills Law of health care benefits for needy aged persons not eligible to receive Old Age Assistance. Health care for those on OAA has been provided in Texas by the Kerr-Mills program since 1961.

Passage of the Constitutional amendment authorizing the Legislature to extend the medical care program was expected since there was no organized opposition to the proposal.

Voters had opportunity to become well-informed on the subject through TMA-prepared news releases carried in newspapers and over television and radio. Physicians and their wives distributed material explaining the proposal through office waiting rooms, hospitals, civic clubs, and direct mail. A brochure on Amendment Three published by the Association was in greater demand than any other ever prepared by TMA. Some 279,000 copies were sent out in answer to requests. Five printings of the leaflet were required to fill the orders.

In the coming preparation of a bill for the Legislature, officers of Texas Medical Association will have opportunity to advise regarding benefits, length of hospital stay, and on other points where physicians are of special help.

'65 Dues Return to '52-'59 Level; Still Under Other States' Average

THE AMOUNT of dues paid by regular members of Texas Medical Association has been returned to the level of $50, the same as charged during the period 1952-1959. However, when Texas physicians remit their 1965 fees, the payment will be well below the average $60 dues paid by members of other state medical societies.

The House of Delegates, meeting in Houston last April, author-

ized an increase in dues not to exceed $10. At its meeting in September, the Board of Trustees, after careful study, voted to increase dues by $5, from $45 to $50 for regular members, effective in 1965. This increase partly is accounted for by a rise in the subscription price of the *Texas State Journal of Medicine* from $3 to $5. The $3 price has existed since 1927.

In reporting to the Executive Board on Sept. 20, Dr. Byron York, chairman of the Board of Trustees, stated that the Board "has planned no further dues increases at this time." He continued, "We are hopeful that the additional operation revenues which will be available starting in 1965 will prove sufficient for at least several years."

The Board of Trustees has reported that the Association's financial affairs for 1964 are better than had been believed possible. In January, it appeared that a deficit would be incurred. However, at present all sources of revenue—membership dues, *Journal* advertising, Annual Session exhibits, and investments—are producing more than was budgeted. Several economies have been initiated and investment dividends have been used to meet some expenses. The outlook is good that the Association's financial report will show some margin of revenue over expenditures for the 11th straight year.

At best, this margin will be small, and without additional revenues from the dues increase a deficit was almost certain for 1965 and future years, the Board has reported.

In choosing to call for an increase in dues, the Board of Trustees earlier ruled out two other possibilities for a balanced budget. One was to curtail existing programs and activities; the other was in invade Association reserves. The dues increase appeared the best course to follow for continued growth and development of the organization.

Support Rallied for Dr. Copeland's Nomination to AMA Board of Trustees

TEXAS DELEGATES plan to support nomination of Dr. J. B. Copeland of Austin to the AMA Board of Trustees when there is next an opening. A letter-writing campaign is in progress in which a number of Texas physicians are contacting friends in the AMA House of Delegates on behalf of Dr. Copeland.

It is possible that a vacancy on the Board may occur during the forthcoming AMA Clinical Convention Nov. 29-Dec. 2 at Miami Beach.

Attending the convention from Texas Medical Association will be Dr. J. B. Copeland, Austin, chairman of the delegates to the AMA; Dr. George Turner, El Paso, alternate delegate; Dr. Harvey Renger, Hallettsville, delegate; Dr. James H. Sammons, Highlands, alternate; Dr. John K. Glen, Houston, delegate; Dr. N. L. Barker, Paris, alternate; Dr. G. W. Cleveland, Austin, delegate; Dr. Ridings E. Lee, Dallas, alternate; Dr. John L. Otto, Galveston, delegate; Dr. E. P. Hall, Jr., Fort Worth, alternate.

Dr. T. C. Terrell, Fort Worth, delegate; Dr. Denton Kerr, Houston, alternate; Dr. M. O. Rouse, Dallas, delegate; Dr. J. C. Terrell, Stephenville, delegate; Dr. J. L. Cochran, San Antonio, alternate; Dr. R. W. Kimbro, Cleburne, delegate; Dr. J. E. Miller, Dallas, alternate;

Dr. Max Johnson, San Antonio, President; Dr. David Wade, Austin, President Elect; Dr. Travis Smith, Abilene, Vice President; Dr. A. Rex Kirkley, Dallas; Dr. Joe T. Nelson, Weatherford; Dr. Joseph Ainsworth, Houston; Dr. Hayden E. McKay, Humble; Philip R. Overton, Austin, General Counsel to TMA; Dean Davis, Austin, Assistant General Counsel; C. Lincoln Williston, Austin, Executive Secretary; and Don Anderson, Assistant Executive Secretary.

A scientific program attuned to the current needs and interests of the practicing physician is planned for the clinical convention. Immunization, depression, cardiac arrhythmias, vascular occlusive diseases, emphysema, iatrogenic diseases, and hypertension are among major areas to be explored.

More than 300 physicians will participate in a full program of lectures, exhibits, motion pictures, color television, fireside conferences, and breakfast round tables.

A new feature of the clinical convention this year is a postgraduate course on obstetrics for the general practitioner. Fifteen lectures will be presented during three sessions ranging from infertility and prenatal care through complications of labor and anesthesia to postnatal care and maternal mortality.

The entire scientific program, with the exception of the fireside conferences and breakfast round tables, will be held in Miami Beach Convention Hall. The modern, single-level structure, completed in 1959, is fully air-conditioned and boasts one of the finest sound amplification systems to be found anywhere in the nation.

The popular fireside conferences, presented as a joint session of the American College of Chest Physicians and the AMA, will be held Sunday night, Nov. 29, at the Fontainebleau Hotel. There will be 11 tables at which 50 to 60 discussion leaders will engage in an informal and free exchange of views on a variety of medical subjects.

Six breakfast round tables are scheduled at the di Lido Hotel. Topics include cancer of the thyroid, cosmetic surgery, and peptic ulcer.

In addition, 125 scientific exhibits will be on display during the meeting, including a special exhibit on fractures, and some 30 medical motion pictures will be shown in the afternoon Monday through Wednesday.

Curbstone Consultation Topics, Other Details Announced for 1965 TMA Annual Session

A VARIETY of subjects of interest to physicians have been chosen for the ten tables making up the 1965 Annual Session Curbstone Consultations.

In other news of the forthcoming meeting of the Texas Medical Association in San Antonio April 28-May 2, 1965, the program for the Symposium on Transportation Safety has been announced as have names of speakers being sponsored by the Committee on Nuclear Medicine. The list of San Antonio hotel and motel accommodations has been released, and information on how to enter a scientific exhibit is available for those interested.

Curbstone Consultation topics are "Functional Gastrointestinal Disease," sponsored by the Section on Digestive Diseases; "Diagnosis and Relief of Headache," Section on General Practice; "Pediatric Gynecology," Section on Obstetrics and Gynecology; "The New Antibotica, Uses and Dangers," Section on Pediatrics; "Diseases of Biliary Tract," Section on Surgery; "Emphysema, Pathology, Diagnosis and Treatment," Texas Chapter, American College of Chest Physicians; "Problems in Dermatology," Texas Dermatological Society; "Newer Diagnostic Tests in Nuclear Medicine," Committee on Nuclear Medicine; "Privileged Communication," Texas Neuropsychiatric Association; and "Cirrhosis of Liver," Committee on Nutrition.

The Symposium on Transportation Safety sponsored by the Committee on Transportation Safety will be held Saturday, May 1, from 10 a.m. until noon. On the program will be Dr. Horace E. Campbell of Denver who will speak on "Alcoholic Drivers"; Mr. James P. Economos, director of the Traffic Court Program of the American Bar Association, Chicago, speaking on "Legal Aspects of Traffic Problems"; Dr. Harold A. Fenner, Jr., past president of the American Association of Automotive Medicine, Hobbs, N. M., "Low Velocity Impact"; Mr. Roy Haeusler, safety engineer of Chrysler Corporation, Detroit, "Car Design and Prevention of Injuries in Accidents"; Col. John P. Stapp, deputy chief scientist, Aerospace Medical Division, Brooks Air Force Base, "Human Tolerance to Crash Impacts"; and Dr. George M. Wheatley, medical director, Metropolitan Life Insurance Co., New York, "Report of Subcommittee for President's Committee on Highway Safety—Medical Aspects of Driver's Licensing, Alcohol and Drugs, Emergency Care, and Transportation of Injured."

Dr. William H. Beierwaltes and Dr. Merrill A. Bender are speakers to be sponsored by the Committee on Nuclear Medicine. Dr. Beierwaltes is with the Department of Internal Medicine of the University of Michigan at Ann Arbor, Mich. He will give a 30-minute paper before the Section on Pathology entitled "Chromosome Abberrations Following Remote I-131 Therapy of Thyroid Gland"; a 30-minute paper before the Section on Pediatrics, "Radioisotope Diagnostic Procedures in Pediatrics"; a refresher course, "Use of Radioisotopes in Obstetrics and Gynecology"; and will be a Curbstone Consultation table participant on "Newer Diagnostic Tests in Nuclear Medicine." Dr. Bender

DR. JOSEPH B. COPELAND, Austin, right, receives a scroll recognizing his contribution to the Texas Academy of General Practice and to medicine from Dr. Haden E. McKay, Humble, outgoing president of TAGP. The scroll calls attention to Dr. Copeland's 33 years of devotion to general practice and to the fact that he was a founder and first president of the Texas organization. Presentation was made during the annual TAGP meeting in Corpus Christi on Oct. 12.

is on the staff of Roswell Park Memorial Institute at Buffalo. He will give a 30-minute paper before the Section on Internal Medicine, "Radioisotopes in Diagnosis of Abdominal Disease"; a 30-minute paper before the Section on Radiology, "Organ Scanning—Brain, Pancreas, Kidney, etc."; a paper before the Texas Chapter, American College of Chest Physicians, "Diagnosis of Pulmonary and Myocardial Infarcts with Radiopharmaceuticals"; and will be a Curbstone Consultation table participant in "Newer Diagnostic Tests in Nuclear Medicine."

San Antonio hotels and motels which are recommended to members of TMA include El Tropicano Hotel, 110 Lexington; Flamingo Hotel, 1131 Austin Highway; Granada Hotel, 309 S. St. Mary's; Gunter Hotel, 205 E. Houston; Hiway House, 96 Military Drive NE; Holiday Inn, 3862 Panam Highway; Howard Johnson's Motor Lodge, 3333 Austin Hwy.; Menger Hotel, Alamo Plaza; Ramada Inn, 333 NW Military Drive; Rodeway Inn, 1259 Austin Highway; Rodeway Inn, 900 N. Main; St. Anthony Hotel, 300 E. Travis; Town and Country Motor Lodge, 6901 San Pedro; Wayfarer Motor Hotel, 600 E. Elmira; Wayhouse Inn, Interstate Hwy. 35 and 81 N.; and Westerner, 1116 Austin Highway. The San Antonio Chamber of Commerce is coordinating housing for the Annual Session. Physicians should write to the TMA Housing Bureau in care of the Chamber office listing three hotel or motel preferences.

Persons interested in entering scientific exhibits in the 1965 Annual Session should make application for space before Jan. 14. Application forms are available from Dale R. Werner, Exhibits Manager, Texas Medical Association, 1801 N. Lamar, Austin. All applications will be reviewed by the Committee on Scientific Exhibits and are subject to its approval. The exhibit space, velour drapes or plywood required in construction of a standard booth, one table (maximum 6′ x 2′), and two plain chairs are furnished without cost for each exhibit. All charges for transportation, storage, or individual signs must be paid by the exhibitor. Exhibits will be located in the Villita Assembly Hall, and booth numbers will be assigned after applications are approved. Three awards will be given in each of four classifications.

MEDICOLEGAL NOTES

Illinois Court Holds Hospital Liable for Physician Negligence

HOSPITAL liability in Texas, as in other states, constantly is being reviewed by the appellate courts. A recent decision by the Appellate Court of Illinois has caught the eye of those who are watching closely the trends in hospital and physician liability, and the result of this case is a significant departure from the limited charitable immunity doctrine now observed in Texas.

Hospitals have been held liable for injuries to patients resulting from ineffective or faulty equipment and for maintaining on the payrolls employees known or reasonably believed to be incompetent. *No Texas courts and only a few in other states have ever charged a nonprofit, charitable, or voluntary hospital with the negligence of its attending physicians.* This is exactly what has been done in the Illinois case. The case has now been appealed to the Illinois Supreme Court, and anxious observers in this field will spend many an uncomfortable moment until the final decision is handed down.

At the trial of the case before a jury last fall, the plaintiff received a judgment for $110,000 against the hospital corporation. The physician invoved in the alleged negligence settled out of court for a sum approaching $40,000. If the case is upheld in the Supreme Court, there will be two major repercussions:

(1) At least in Illinois, the liability of a nonprofit hospital will be extended drastically; and (2) at least in Illinois, the liability of lay adminsitrators for the medical practice in their hospital will be determined, thereby sharply focusing on a reevaluation of hospital-physician organizational relationships.

These are the facts of the case:

A college athlete suffered a broken leg in a football game. After receiving emergency treatment at the stadium, he was transferred to the defendant hospital. Dr. A diagnosed the fracture as a break of both the tibia and the fibula, and with the help of an orderly and two nurses, set the fractures. Then the leg was covered with a plaster cast, and according to the plaintiff's petition, there was no underpadding to protect the leg from the quick-setting plaster.

During the first two weeks the boy was in the hospital, the nurses' notes reflect substantial suffering and the administration of narcotics and other pain medication as well as "decreasing sensation and increasing cyanosis of exposed toes," and some week and a half after admission, the presence of a "foul odor."

Some two weeks after admission to the defendant hospital, the patient was transferred to another hospital and placed under the care of an orthopedic surgeon. After several operations, the leg was amputated. The plaintiffs contended that the first hospital was liable because the doctor was a general practitioner who was not qualified to perform orthopedic surgery and that the regulations of both the Joint Commission, the hospital bylaws, and even the code of ethics of the American College of Hospital Administrators all required the administrator acting for the hospital board, to be responsible for the quality of patient care in the hospital. The physician involved testified that despite the fact there were complications he never consulted with other physicians and disagreed with various orthopedic texts as to how fractures of this type were to be handled. The hospital administrator testified, "I did nothing to see that Dr. A reviewed his operating tech-

niques for the handling of broken bones. So far as I know, Dr. A may not have reviewed his operating techniques since he first was licensed to practice in 1928. . . ."

Possibly one of the most alarming portions of the court's opinion is that the court seemed willing to take the standards of hospital care from the hospital's bylaws, rules, and regulations and to apply them *literally* in sustaining the plaintiff's cause of action.

In defense, the defendant hospital presented witnesses who testified to facts showing that such operation on the part of the hospital was "in conformity to standard practice in the area."

The conclusions reached by the court are shown in its opinion which, in pertinent parts, reads as follows:

> The Plaintiff's theory is that the Defendant hospital corporation which held itself out to the public as a licensed and accredited hospital was held to the standards imposed by law, by public regulation, and by self-regulation upon such institution; it had to maintain certain departments, such as nursing, laboratory, medical and surgical, and it was required to maintain certain standards applicable to each of them; whether the Defendant did so in this case, or not, became a matter of fact upon which the evidence was overwhelming in favor of the Plaintiff; the verdict is amply supported by the evidence; and there was no harm unless of a trivial character. . . .
>
> After considering the arguments of the Plaintiff and Defendant, and the authorities relied on, we believe that conformity with the standards of care observed by other hospitals in good standing in the same community cannot necessarily in itself be availed of as a defense in a negligence action, where the criterion relied upon is shown to constitute negligence, in that it fails to guard against injuries to the patient. . . ."

The final result of this radical departure in hospital liability will be given *Journal* readers after the Supreme Court of Illinois has reviewed the case.

—PHILIP R. OVERTON, LL.B.,
Austin.

Statewide Panel Approves New Mental Health Plan

A STATEWIDE citizens committee—including strong representation from the medical profession—has approved a long list of recommendations for a new program of mental health services for Texas.

These recommendations, approved by the 112-member planning committee at an Oct. 16 meeting in Austin, now will be put into final form and sent to Gov. John Connally and the 59th Legislature for possible legislative action in 1965.

They are contained in a thick document entitled "The Texas Plan for Mental Health Services." Various sections of the plan have been written during the past 11 months by 13 subcommittees within the statewide panel.

Among the chief recommendations:

—The 59th Legislature should establish a new Texas Department of Mental Health, to take over the mental health and hospital functions of the present Board for Texas State Hospitals and Special Schools and also the present activities of the division of mental health, State Department of Health.

—The proposed Texas Department of Mental Health should be responsible for an accelerated program of recruiting and training psychiatrists, psychologists, and other mental health personnel which now are in short supply.

—Rehabilitation of mentally ill citizens should begin as closely as possible to the time and place where the illness begins.

—More emphasis should be placed on the prevention of mental illnesses and the promotion of mental health in local communities.

New mental health research institutes should be established in Dallas, Galveston, Austin, and San Antonio. (The state now has one psychiatric research facility in Houston.)

—The Legislature should submit to a vote of the people a proposed amendment to the Texas Constitution which would allow use of state funds to assist in financing the community mental health services.

—Community mental health centers should be established as rapidly as possible in the state's larger population centers, to supplement existing hospitals and clinics.

The October meeting was the sixth session in Austin for the planning committee since it began its work in December, 1963. Two-day sessions also were held in Austin in February, April, June, and August. (A complete listing of members of the statewide committee was given in an introductory article on the planning effort in the *Texas State Journal of Medicine* for January, 1964.)

Details on the administration and coordination of the proposed new mental health plan were worked out in advance of the Oct. 16 meeting by the 17-member steering committee, made up of state officials, educators, and medical doctors. They were endorsed by the statewide committee.

The proposed Texas Department of Mental Health should be responsible, the committee said, for developing and implementing a program which would help carry out the following objectives:

—"Preserve, protect, and promote the mental health of all Texans;

—"Prevent the development of mental illness;

—"Limit the progress of mental illness once established;

—"Reduce and remove the disability of those who have been mentally ill;

—"Return them to a meaningful and productive life in their own community."

The committee further recommended that the proposed Texas Department of Mental Health be guided by policy developed by a nine-member, unsalaried Board which would include three members of the medical profession, one of them a psychiatrist. The board members would be appointed by the Governor, with Senate approval, for six-year, overlapping terms.

The recommendations also call for a mental health advisory council of 15 members, appointed by the Governor, which would advise the board on professional matters. Chairman of this council should be a psychiatrist, the committee recommended.

In addition, the committee recommended establishment, by law, of a medical committee which would "study and advise with the mental

health advisory council on medical standards and care within the Texas Department of Mental Health." This committee, as recommended, would be chaired by the advisory council chairman and also would include the heads of the departments of psychiatry from the three medical schools in Texas, three psychiatrists in private practice, two general practitioners, and one pediatrician.

Chief administrative officer of the proposed new department would be a commissioner of mental health, who should be a physician licensed in Texas and certified in psychiatry.

Judge C. L. Abernethy of Hale County, chairman of the statewide committee, said efforts will be increased during November and December to inform the medical profession, the public, and public officials about the recommendations of the Texas Plan. A summary booklet on the highlights of the plan will be distributed before Jan. 1.

Serving with Judge Abernethy on the executive committee for the planning are Dr. James E. Peavy, state commissioner of health, chairman; Dr. C. J. Ruilmann, director of mental health and hospitals for the Board for Texas State Hospitals and Special Schools, co-chairman; and Robert L. Sutherland, Ph.D., director of the Hogg Foundation for Mental Health at the University of Texas.

The four leaders pledged their full support to the new plan and the proposed new Texas Department of Mental Health. Dr. Peavy reminded the planning committee that it still had important work ahead in helping to implement the plan.

Dr. Ruilmann said the committee had been outstanding for its honesty, creativity, and willingness to work.

Judge Abernethy expressed satisfaction with the community-centered treatment philosophy which is the central theme of the mental health plan. The chairman said:

"If we work together we can treat mental illnesses, and prevent many of them, on a community basis. It is much better to treat these people at home and let them get well at home and sometimes even continue working at home, than to send them away. Rehabilitation begins immediately under the community plan, not when the patient returns."

A charm displaying the official seal of the Woman's Auxiliary to the Texas Medical Association is offered for sale by the Auxiliary with proceeds going to American Medical Association Education and Research Foundation. Actual size, ⅞ inch.

14 Carat Gold	$25.20
Gold Filled	8.69
Sterling Silver	6.16

A new charm in the shape of our State of Texas is being offered for sale by the Auxiliary with proceeds going to the American Medical Association Education and Research Foundation. Actual size, 1 inch.

14 Carat Gold	$20.16
Gold Filled	5.60
Sterling Silver	5.60

Orders for these charms should be directed to the Auxiliary Office, Texas Medical Association, 1801 North Lamar Boulevard, Austin. Checks should be made payable to AMA-ERF.

ACS Inducts Fellows Including 44 Texans

Texas physicians who are new fellows of the American College of Surgeons include:

Joe F. Robberson, Amarillo; Gaston Machado and Frank A. Rainone, Jr., Arlington; Bryan W. Forister, Jr., and Francis A. Morris, Jr., Austin; Joseph A. Dowlen, Baytown; Charles H. Fuller, Benjamin A. McReynolds, Arthur L. Sarris, Harold C. Urschel, Jr., and Otto L. Willbanks, Jr., Dallas.

Glen E. Furr and Stephan N. Schanzer, El Paso; Leeroy J. Kelly, Fort Worth; George W. Fisher, Fort Sam Houston; John B. Lynch, Galveston; Bobby R. Alford, Robert J. Bacon, Charles D. Chamberlain, Jr., James A. Chamberlin, Gerald S. Dowdy, Jr., Simon Fredricks, John G. Fries, H. Edward Garrett, Joseph C. Gathe, Grady L. Hallman, Jr., Edward J. Kirby, Alice R. McPherson, Howard E. Miller, and Joseph W. Robertson, Houston;

Lester F. Williams, Jr., Lackland Air Force Base; Richard D. Smith, Lubbock; Charles E. Nichols, Marlin; Fortunato M. Manzanero and Robert W. Milam, McAllen; Durwood N. Clader and Garland H. Lang, Midland; Leonard O. Coleman, Navasota; John L. Doyle, San Antonio; James H. Hockenberry, Sheppard Air Force Base; Tom E. Miller, Sherman; John W. O'Banion, Jr.; Snyder; Raymond H. Hedge, Jr., Tyler; and Albert Palatchi-Sharaff, Wichita Falls.

Arizona Meeting Set For International Group

The Medical Society of the United States and Mexico will meet Dec. 9-12 at the Mountain Shadows Hotel in Scottsdale, Ariz.

Both Mexican and American physicians will participate in symposiums and presentation of individual papers. Planned to appeal to those in all branches of medicine, symposiums will be held on cancer chemotherapy, allergy, pulmonary diseases, congenital defects, and diarrheal diseases. Individual presentations will include discussions of mammography, pancreatitis, pulmonary surgery, and psychosomatic medicine, and other topics.

A system of simultaneous translation will be provided for those

who are not bilingual.

Further information may be obtained from Dr. James D. Nauman, secretary, 1603 North Tucson Blvd., Tucson, Ariz.

Space Symposium Announced for November

The third international symposium on bioastronautics and the exploration of space will be held in San Antonio Nov. 16-18. Sponsored and arranged by Aerospace Medical Division, Southwest Research Institute, headquarters for the meeting will be at the Granada Hotel.

Symposium coordinator is Jack Harmon, Southwest Research Institute, P. O. Box 2296, San Antonio.

(Inside copy of card)
(Actual color not as shown, instead a turquoise blue.)

THE WOMAN'S AUXILIARY to the Texas Medical Association is taking orders for the Christmas card, pictured above, which sells for $2.50 per 25-card box. Proceeds from the sale will go to the American Medical Association Education and Research Foundation. An insert for each card, stating that the "card was purchased from the Woman's Auxiliary to the Texas Medical Association to benefit the AMA-ERF," is enclosed for your use if you so desire. Orders should be directed to the Auxiliary Office, Texas Medical Association, 1801 North Lamar Blvd., Austin.

PR Conference Talks Available on Tape

Tapes of addresses given during the Public Relations Conference in Austin Sept. 19 are available on loan from Texas Medical Association Memorial Library, 1801 N. Lamar Blvd., Austin.

Houston To Host Private Clinics, Hospitals

The Private Clinics and Hospitals Association of Texas will meet at the Sheraton-Lincoln Hotel in Houston Dec. 4-5.

Included on the program are the following addresses and speakers: "Cash Control for the Small Medical Clinic," James H. Gillis, CPA, IACPA; "Advantages of Clinics Use of the Service Bureau," John Gill, clinic manager, Scott and White Clinic, Temple; "Services Available Through Services Bureaus," O. B. Fraser; "Report from Texas Health Insurance Council," Robert L. Davis; "History of the PCHA of Texas," Dr. John B. Chester, Dallas; "Importance of the Private Hospitals to the Texas Hospitals Association," O. Ray Hurst, executive director of THA, Austin; "What's in the Future for the Proprietary Hospital?," Dr. George L. Walmsley, Baytown;

"Report on Regional Planning," Dr. J. F. Peters, Alvarado; "Retirement Programs for Doctors and Personnel," representative of Southwestern Life Insurance Co.; "Planned Insurance Program for the Proprietary Hospital," Daley Williams, representing Blain & Co.; and "Personal Financial Management," E. K. McClain, Jr., Management Service for Doctors, Waco.

Dec. 11 Date Set For Rheumatism Meet

The Texas Rheumatism Association will hold its annual meeting Dec. 11 in the Sam Houston Room of the Sheraton Hotel in Dallas.

Guest speakers will be Dr. Howard F. Polley, president of the American Rheumatism Association and chief of the section of arthritis at Mayo Clinic, Rochester, Minn., and Dr. Javier Robles Gil, president of the International League for the Control of Rheumatism, Mexico City.

Speakers and their papers will include Dr. J. Donald Smiley, Dallas, "Scleroderma"; Dr. Louis H. Paradies, Dallas, "Use of Synovectomy in the Treatment of Rheumatoid Arthritis"; Dr. John T. Sharp, Houston, "Clinical Studies in the Glomerulus"; Dr. Gil, Visceral Lesions of the Most Important Connective Tissue Diseases";

Dr. J. E. Miller, Dallas, "Interpretation of Roentgenograms in Skeletal Diseases"; Drs. Earl J. Brewer, Houston, and Willson J. Fahlberg, "Serum Factors in Children with Rheumatoid Arthritis"; Dr. Polley, "Discussion of the Morning Papers"; Dr. Edward M. Krusen, Dallas, "Treatment of the Painful Shoulder"; Dr. John Baum, Dallas, "Autoantibodies in the Connective Tissue Diseases";

Dr. Polley, "Differential Clinical Features of Connective Tissue Diseases"; and Drs. Baum, Polley, Gil, Sharp, and Charles Ashworth of Dallas, "Panel on Disseminated Lupus Erythematosus."

Personals

President-elect of Texas Academy of General Practice is *Dr. Guy T. Denton, Jr.*, of Dallas. Other new officers are the president, *Dr. J. Frank Beall*, Nacogdoches; the vice president, *Dr. Lloyd M. Southwick*, Edinburg; and the treasurer, *Dr. Durwood E. Neal*, Fort Worth.

Dr. Robert C. Berson has resigned as dean of the University of Texas South Texas Medical School, San Antonio, and has been named the executive director of the Association of American Medical Colleges.

A group of 350 friends feted *Dr. Max Johnson* of San Antonio with a testimonial luncheon on Sept. 29 at San Antonio's St. Anthony Hotel. The program was arranged by Bexar County Medical Society and the San Antonio Chamber of Commerce. Represented at the gathering were the professions, business, civic, and cultural elements, and the armed services.

Dr. W. B. Russ recently celebrated his 90th birthday. He is San Antonio's oldest practicing physician.

The Nueces County Medical Society recently honored past presidents including *Dr. George Wyche* of Alice, *Dr. McIver Furman* of Corpus Christi, *Dr. Norman T. Gibson* of Robstown, *Dr. E. T.*

DR. EDWARD R. ANNIS, immediate Past President of the American Medical Association, center, spoke in El Paso Sept. 26 to members of the El Paso County Medical Society, staff members of William Beaumont General Hospital, and physicians from District One of the Texas Medical Association. Above, left to right, are Brig. Gen. James B. Stapleton, commanding general of William Beaumont General Hospital, Mrs. Russell L. Deter, El Paso, president-elect of the TMA Woman's Auxiliary, Dr. Annis, Dr. H. D. Garrett, president of the El Paso County Medical Society, and Dr. Ira A. Budwig, El Paso, president-elect of District One of the TMA.

Bickley, Dr. C. P. Jasperson, Dr. Ralph St. John, Dr. Turner Moller, Dr. John Pilcher, Dr. Foy Moody, Dr. Leslie Garrett, Dr. Franklin Yeager, Dr. M. C. Kendrick, Dr. Hugh Kennedy, Dr. W. E. Morris, Dr. Gerald Ahern, Dr. James Barnard, Dr. Harold Griffin, Dr. Paul Gray, Dr. James Riley, Dr. Walter Brown, Dr. Jack McKemie, and *Dr. James Gabbard,* all of Corpus Christi.

Dr. Gordon P. Baker of Dallas spoke on "Treatment of Scoliosis" at the October meeting of the Hill County Medical Society in Whitney.

Dr. Betty Pearce Stephenson of Houston has recently become a fellow of the American College of Anesthesiologists.

Dr. Truman G. Blocker, Jr., of Galveston was named on the program of the 15th National Conference on Disaster Medical Care held in Chicago Nov. 7-8. His topic was "The Medical Aftermath of the Texas City Disaster."

Dr. George F. Crawford of Beaumont has been elected a director of the Flying Physicians Association.

Dr. C. J. Ruilmann of Austin is the subject of a feature, "High-Flying Texan," in the Sept.-Oct. issue of *SK&F Psychiatric Reporter,* published by Smith Kline & French Laboratories.

Members of the Tom Green Eight Counties Medical Society heard *Dr. William Spencer* describe the problems and misconceptions of rehabilitation at its September meeting in San Angelo.

A 1965 TMA Annual Session speaker is the author of an article on alcoholism in the September issue of *Reader's Digest.* He is *Dr. Marvin A. Block* of Buffalo, for ten years chairman of the AMA Committee on Alcoholism.

New officers of the Texas Industrial Medical Association are *Dr. Roy E. Joyner,* Texas City, president; *Dr. Robert R. Dugan,* Houston, first vice president; *Dr. James K. Stewart,* Beaumont, secretary-treasurer; *Dr. Max E. Johnson,* San Antonio, *Dr. Lee Lloyd Pickett,* Houston, *Dr. Charles O. Bruce,* Port Neches, *Dr. J. K. Leverett,* Fort Worth, *Dr. Robert J. Potts,* Dallas, *Dr. George E. McGlothlen,* Victoria, and *Dr. Robert L. Raleigh,* Longview, members of the board of directors; *Dr. J. G. Burdick,* Pasadena, counsellor, Southern Region; and *Dr. Frederick Fink,* Dallas, counsellor, Northern Region, Dallas.

Laredo is believed to be one of the few communities in the nation in which women physicians hold two key health positions. *Dr. Margaret G. Cigarroa* is city public health officer and *Dr. Ruby Lowry* is city school physician.

Dr. William A. Spencer of Houston has been designated the 1964 Horowitz Visiting Professor of the Department of Physical Medicine and Rehabilitation, New York University School of Medicine.

New officers of the Panhandle District Medical Society are *Dr. Joe Arrington, Jr.,* Lubbock, president; *Dr. Wilbur Q. Budd,* Amarillo, president-elect; *Dr. Ray Freeman,* Hale Center, first vice president; *Dr. Walter Brooks,* Quanah, second vice president; and *Dr. Louis Devanney,* Amarillo, secretary.

Dr. James Y. Bradfield of Dallas has been elected vice president of the Flying Physicians Association.

Three physicians who have completed residencies at the Mayo Foundation in Rochester, Minn., have left that city and will be located in Texas. *Dr. R. D. Cunningham,* an ophthalmologist, goes to Temple; *Dr. B. H. Warren,* pathologist, to San Antonio; and *Dr. R. C. Gasser,* radiologist, will practice in Wichita Falls.

Four Texas physicians were among 35 recipients of Outstanding Achievement Awards from the University of Minnesota on Sept. 17. They are *Dr. Nicholas C. Hightower, Jr.,* of Temple, and *Dr. R. Lee Clark, Jr., Dr. Stanley W. Olson,* and *Dr. Raymond D. Pruitt,* all of Houston.

Dr. Herman L. Gardner of Hous-

ton is the first Texan to become president of the Central Association of Obstetricians and Gynecologists.

New officers of the Texas Academy of General Practice are *Dr. J. Frank Beall*, Nacogdoches, president; *Dr. Lloyd M. Southwick*, Edinburg, vice president; and *Dr. Durwood E. Neal*, Fort Worth, treasurer.

Officers of the Texas Heart Association are *Dr. Robert H. Mitchell*, Plainview, president; *Dr. Julian H. Acker*, Wichita Falls, vice president; and *Dr. Saul B. Appel*, El Paso, secretary.

The Postgraduate Medical Assembly of South Texas has announced the following officers: *Dr. T. J. Fatherree*, Houston, president; *Dr. William R. Knight, III*, Houston, president-elect; *Dr. C. I. Shult*, Columbus, vice president, District 8; *Dr. George V. Pazdral*, Somerville, vice president, District 9; *Dr. George J. Pruitt*, Lufkin, vice president, District 10; *Dr. James Allen Chamberlin*, Houston, secretary; and *Dr. Elmer K. Sanders*, Houston, treasurer.

Dr. R. S. Sparkman, Dallas, is the new president of Texas Surgical Society; *Dr. Sam G. Dunn*, Lubbock, is first vice president; *Dr. John P. Heaney*, San Antonio, second vice president; *Dr. Walter B. King, Jr.*, Waco, secretary; *Dr. W. S. Lorimer, Jr.*, Fort Worth, treasurer; and *Dr. R. W. Crosthwait*, Waco, archivist.

Dr. E. E. Anthony, Jr., Fort Worth, is the new president of the American Association of Physicians and Surgeons.

Mrs. Marilyn Baker, managing editor of the *Texas State Journal of Medicine*, sustained fractures of the pelvis and ankle in a car accident near New Orleans, Oct. 16.

Texas physicians named on the program of the 23rd annual meeting of the American Academy of Dermatology Dec. 5-10 in Chicago include the following: *Dr. John M. Knox*, *Dr. Leopold F. Montes*, and *Dr. Everett R. Searle* of Houston; *Dr. J. Fred Mullins* of Galveston; *Dr. J. Lewis Pipkin* of San Antonio; *Dr. Edmund N. Walsh*, Fort Worth.

The International Atomic Energy Agency (IAEA) of the United Nations has appointed *Dr. Thomas Powell Haynie* of the University of Texas Medical Branch faculty as a technical advisor in nuclear medicine to the Hospital Center in Casablanca, Morocco.

Officers for the Texas Pediatric Society are *Dr. Tom W. Black*, San Antonio, president; *Dr. Paul R. Meyer*, Port Arthur, president-elect; *Dr. Clarence E. Gilmore*, Paris, secretary; *Dr. Donald H. McDonald*, Abilene, treasurer; and *Dr. A. L. Alfieri*, Dallas, chairman of District 2.

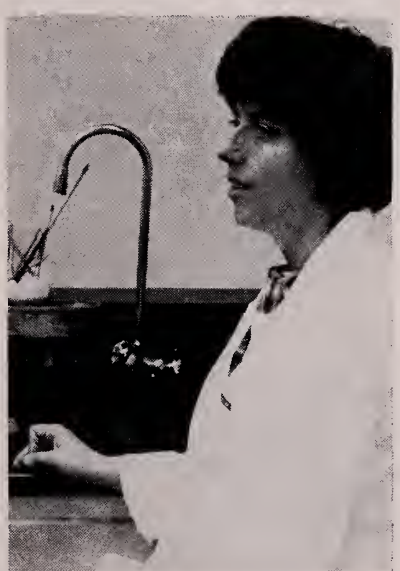
Those attending the meeting of the section on ophthalmology at the SMA meeting will hear three Texas physicians — *Drs. Louis J. Girard, Milton Boniuk*, and *L. Leighton Hill* of Houston.

The program for the meeting of the section on otolaryngology for the SMA meeting includes the names of *Dr. Louis J. Girard, Dr. Frederick R. Guilford*, and *Dr. S. Donald Greenberg* of Houston and *Dr. Claude D. Winborn* and *Dr. John T. Mallams* of Dallas.

The Southern Medical Association section on pathology includes on its program for the forthcoming meeting the names of *Dr. Robert J. Rowe* of Dallas and *Dr. William J. McGanity* of Galveston.

Dr. Don P. Morris and *Dr. William C. Grater* of Dallas, *Dr. James W. H. Rouse* of San Antonio, *Dr. Anthony A. Fernandez* of Houston, and *Dr. Stephen R. Lewis* and *Dr. Duane L. Larson* of Galveston will appear on the pediatrics section program of the SMA.

Dr. Stephen R. Lewis and *Dr. Duane L. Larson* of Galveston will speak before the section on plastic and reconstructive surgery at the meeting of the Southern Medical Association.



DR. JARA MOSER of Prague, Czechoslovakia, the first recipient of a one-year fellowship grant from the University of Texas Medical Branch supported by the Shriners Hospitals for Crippled Children, will work with Dr. Curtis P. Artz in an exchange of ideas and methods of burn treatment and research at the Shriners Burn Institute in Galveston.

Taking part on the program of the section on proctology at the SMA meeting will be *Dr. O. P. Griffin* of Fort Worth, *Dr. James M. Hampton* of Houston, and *Dr. Robert J. Rowe* of Dallas. *Dr. Rowe* will also appear before the section on radiology.

Dr. Watts R. Webb of Dallas is vice chairman of the SMA section on surgery. He is scheduled to appear on the program as is *Dr. Robert N. McClelland* of Dallas.

Speakers from Texas at the SMA section on urology will include *Dr. Russell Scott, Jr.*, of Houston and *Dr. Terry D. Allen* of Dallas.

Dr. Thomas R. McElhenney of Austin is chairman of the section on allergy of the Southern Medical Association, *Dr. Theodore J. Haywood* of Houston is secretary, and *Dr. Frederick W. Grover* of Dallas is secretary-elect. Appearing on the allergy section program at the forthcoming meeting of the Southern Medical Association in Memphis Nov. 16-19 will be Texans *Drs. Anthony A. Fernandez, Herbert L. Fred, and William H. Lauderdale* of Houston; *Dr. James W. H. Rouse* of San Antonio; *Dr. William C. Grater* of Dallas; and *George J. Seibold* of Wichita Falls.

Anesthesiologists from Texas on the Southern Medical Association section program are *Dr. William A. Boyd of Houston* and *Dr. Joseph P. Jones* of Temple.

Appearing before the SMA section on dermatology will be *Dr. Robert G. Freeman* and *Dr. John M. Knox* of Houston, *Dr. Earl B. Ritchie* of Galveston, *Dr. W. R. Hubler* of Corpus Christi, and *Dr. M. Allen Forbes, Jr.*, of Austin.

Dr. Marcel Patterson of Galveston is chairman of the Southern Medical Association section on gastroenterology. Appearing on the program of that section are *Drs. Charles W. Barrier, Jr.*, and *N. C. Hightower, Jr.*, of Temple, and *Dr. Patterson.*

Drs. Max A. Stoner, Anthony A. Fernandez, and *Lewis A. Leavitt* of Houston, *Drs. James W. H. Rouse* and *Eugene T. Standley* of San Antonio, *Drs. William C. Grater, Watts Webb*, and *Robert N. McClelland* of Dallas are to appear before the SMA section on general practice.

Among those listed on the program for the meeting of the Radiological Society of North America

to be held in Chicago Nov. 29-Dec. 4 are the following: *Drs. Francisco Alaniz Camino, Gilbert H. Fletcher, Vincent P. Collins, Norah DuV. Tapley, Philip T. Hudgins, Herman D. Suit, R. D. Lindberg,* and *Edward B. Singleton* of Houston; and *Dr. F. J. Bonte,* Dallas.

Drs. Arthur C. Beall, Jr., Denton A. Cooley, Michael E. DeBakey, Don C. Quast, John B. Fitzgerald, George C. Morris, Jr., and *Grady L. Hallman,* Houston; *Drs. Watts R. Webb, Ben F. Mitchel, Jr., Maurice Adams, Milton V. Davis, Fouad A. Bashour, Carleton B. Chapman, Harold C. Urschel, Jr., James W. Finney, Alan K. Pierce, William L. Kraus,* and *Jacob L. Kay,* Dallas; and *Dr. Per H. Langsjoen,* Temple, will take part on the program of the American College of Chest Physicians, Southern Chapter, meeting in Memphis Nov. 15-16.

Dr. James Y. Bradfield of Dallas will speak at the meeting of the Flying Physicians Association in Memphis Nov. 16.

Houston physicians who will have scientific exhibits in Memphis for the Southern Medical Association meeting include the following: *Drs. B. R. Alford, George N. Gorman,* "Osteoplastic Surgery of the Frontal Sinus"; *Drs. Arthur C. Beall, Jr., Denton A. Cooley,* and *Michael E. DeBakey,* "Prosthetic Replacement of Cardiac Valves"; *Dr. Robert B. Elliott,* "The Hagie Pin"; *Drs. Gunyon M. Harrison* and *Franklin James Harberg,* "Surgical Complications and Their Management in Cystic Fibrosis of the Pancreas"; *Drs. John Kinross-Wright, K. D. Charalampous,* and *Najia H. White,* "An Economic and Effective Way of Providing Follow-Up Care"; and *Drs. L. R. Malinak, G. V. Miller, A. M. Faris,* and *T. G. Gready, Jr.,* "Conization —Hysterectomy Time Interval."

Galveston physicians to show a scientific exhibit at the Southern Medical Association meeting are *Drs. Marcel Patterson, John McGivney, Arnold Drake,* and *Helen Ong.* The exhibit is "Topical Steroid Therapy of Ulcerative Colitis —A Ten Year Experience."

Dallas physicians who will have scientific exhibits at the Southern Medical Association meeting include *Drs. Jesse E. Thompson, Dale J. Austin, C. Gene Wheeler,* and *R. Don Patman,* "Aorto-Iliac Surgery"; and *Drs. Watts R. Webb* and *Unal Degerli,* "Clinical Evaluation of a New Mucolytic Agent, Acetylcysteine."

Vacation with Service

IT IS NOW POSSIBLE for doctors to have a vacation in a foreign country and at the same time practice their profession," reports Dr. Otto Heins of Raymondville.

Last winter Dr. Heins, now 62, spent two months with the Aymará Indians in Bolivia under the auspices of the Laymen's Overseas Service of Jackson, Miss.

"It was a wonderful experience," he says, going on to tell something of this South American country. "Bolivia has an area about the size of Texas and California combined. It is referred to as the Tibet of America, as most of the people live at a level of 12,000 to 14,000 feet. Of over 3,000,000 Bolivians, nine-tenths are Indian or mestizo. The majority are hungry and diseased; only 30 percent can read or write. There is one physician to about 3,850 people, and most physicians are in the large cities. Bolivia has a sick economy, and political turmoil keeps the natives on the abyss of Communism."

His time was spent around beautiful Lake Titicaca which has an altitude of 12,500 feet, and is one of the highest large lakes in the world. The Aymará Indians who live there are more aggressive than those of the lowlands, perhaps because of the cold climate.

"While I was there I was chilled all the time, although it was their midsummer," Dr. Heins recalls.

The natives make a bare existence by farming poor, rocky, exhausted soil. They live in stone or mud huts, with the most primitive type of sanitation and hygiene. All chronic diseases are found here —tuberculosis, intestinal parasites, skin ulcers, arthritis, leprosy, vitamin deficiency, and dental decay. Their main diet consists of potatoes, barley, and beans. The meat of cattle and sheep, however, help a poor protein diet. The adults are fairly well developed with a large rib cage due to the high altitude. The children seem sickly and undernourished, with an estimated mortality of 40 percent the first year.

Dr. Heins found the people very friendly and appreciative. Many walked hours to avail themselves of modern medicine. The village *curanderos* and medicine men still treat most of the sick because they, as a rule, have no competition.

A Lutheran, Dr. Heins organized a clinic at the Methodist mission in Warisata at the base of snow-covered Mt. Iliampu (about 23,000 feet). The village of Warisata has a rather modern teachers college of about 300 students. The college and the mission home are the only two buildings there not built from mud and stone. There was no doctor besides Dr. Heins for many miles for an estimated population of 30,000 to 50,000.

"It was very gratifying to find missionaries of all denominations working together for the good of the natives," says Dr. Heins. "Whenever an emergency arose, I was 'borrowed' by Baptists, Lutherans, and Catholics."

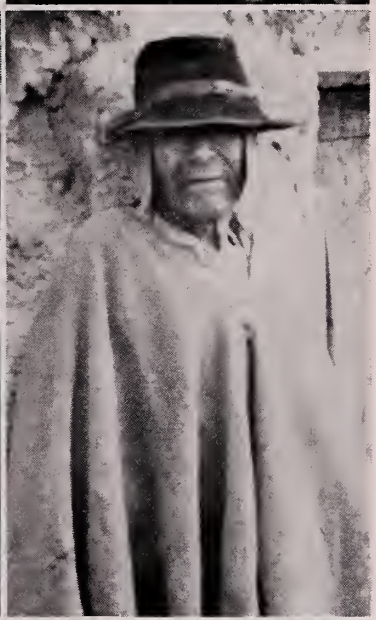
Part of Dr. Heins' time was spent with the Baptist mission at Huatajata from which he also made calls to the islands in Lake Titicaca. He reports that the Baptists have a fine primary school with more than 1,000 pupils, and a good clinic and nurse, but there is no doctor.

In Bolivia, Dr. Heins found it frustrating to attempt the practice of modern medicine with outdated samples and hardly any instruments. Since his return, he has been speaking and showing his slides of Bolivia to interested groups in a fund-raising effort to meet some of the needs of the Warisata clinic.

Out of his experience in Bolivia, Dr. Heins has accepted the philosophy that "if, instead of foreign aid, we would use a fraction of this money to help all mission and peace corps clinics, we would again be loved and respected in foreign lands."

"There are several lay organizations which will welcome Texas doctors who will give a hand to work for a short while in foreign countries," Dr. Heins says. "The need is great, and the people are grateful."

A Raymondville physician
spends two months with Indians in Bolivia.



Dr. Otto Heins (at center) and scenes taken near Lake Titicaca

Radio, TV Added To A. Jones Awards

Excellence in reporting health information to the public via radio and television will be recognized next year as a part of the Anson Jones Award program. This brings to six the number of categories in the contest which for several years has offered competition for three classes of newspapers and for magazines.

The awards are presented annually by the Texas Medical Association as an expression of appreciation and gratitude to the news media of Texas. Each award consists of a copper plaque mounted on a mahogany base which is presented to the winning publication or station, and a cash award and certificate which goes to the individual writer or producer. Winners for 1965 are expected to be announced about Feb. 1.

Entries are submitted to the county medical society where the publication or station is located. The county society then forwards the entry to Texas Medical Association Headquarters, 1801 N. Lamar, Austin, no later than Dec. 10.

Contest rules may be obtained from county medical societies or by writing to Jon R. Hornaday, Public Relations Director, Texas Medical Association, 1801 N. Lamar, Austin.

Pediatric Researchers To Meet at Baylor

The fifth annual meeting of the Southern Society for Pediatric Research will be Dec. 4-5 in Houston. Host will be Baylor University College of Medicine. The headquarters hotel is the Hotel America, and the site of the meeting will be Baylor University College of Medicine, Jesse Jones Library Auditorium at Texas Medical Center.

Dallas To Host Texas Internists

The Texas Academy of Internal Medicine will meet Dec. 12 in Dallas at the Sheraton-Dallas Hotel.

Speakers and their subjects will be Dr. John S. Chapman, Dallas, "Theoretical Mechanism for Phlebitis and Infarction"; Dr. Seymour Eisenberg, "Changes in Blood Viscosity in Subjects with Congestive Heart Failure"; Dr. Floyd C. Rector, Jr., Dallas, "Mechanism of Action of Newer Diuretics"; Dr. Robin Johnston, "Differentiation of Supraventricular from Ventricular Tachycardias"; Dr. Burton Combes, Dallas, "Problems of Jaundice in Pregnancy"; Dr. Watts Webb, Dallas, "Experimental and Clinical Aspects of Pulmonary Transplantation";

Dr. Eugene P. Frenkel, Dallas, "The Refractory Hypochromic Microcytic Anemia"; Dr. John Fordtran, Dallas, "Carcinoid Syndrome"; Dr. Rolland C. Reynolds, Dallas, "The Pathogenesis and Electron Microscopy of Pulmonary Emphysema"; Dr. Jay P. Sanford, Dallas, "Inhalation Therapy Equipment; A Potential Source of Hospital-Acquired Infections"; Dr. Steven Schenker, "Pathogenesis of Hepatic Coma"; and Dr. Holbrooke Seltzer, "The Effect of Prolonged Administration of Orinase on Insulin-Secretory Reserve."

Familiar Medical Quotes To Be Compiled

The publishers Little, Brown and Company of Boston are compiling a book of quotations about medicine. It is planned to be "a great collection of wit and wisdom from and about physicians of the ages."

The editors have asked for suggestions for entries. Anyone who has a favorite medical quotation may have it considered for publication. The collection will include not only quotes from physicians, teachers, and investigators but also from lay writers, including the scriptures, that pertain to disease and treatment or even doctors and medical life.

Proposed entries should be sent to Familiar Medical Quotations, Little, Brown and Company, 34 Beacon Street, Boston 6, Mass.

New Books In TMA Library

Anderson, W. A. D.: **Synopsis of Pathology**, 6th ed., St. Louis, C. V. Mosby Co., 1964.

Bagge, L.: **Psychosomatic Approach to Cystic Glandular Hyperplasia of the Endometrium**, Sweden, Almqvist and Wiksells, Boktrycheri Ab, 1963.

Beard, G., and Wood, E. C.: **Massage, Principles and Techniques**, Philadelphia, W. B. Saunders Co., 1964.

Bergler, E.: **Basic Neurosis, Oral Regression and Psychic Masochism**, New York, Grune & Stratton, Inc., 1949.

Bergler, E.: **Superego Unconscious Conscience**, New York, Grune & Stratton, Inc., 1952.

Black, M., and Wagner, B. M.: **Dynamic Pathology**, St. Louis, C. V. Mosby Co., 1964.

Chatton, M. J., and Sanazaro, P. J.: **Current Medical References**, 3rd ed., Los Altos, Calif., Lange Medical Publications, 1963.

Ciba Foundation Symposium: **Cellular Injury**, Boston, Little, Brown & Co., 1964.

Ciba Foundation Symposium: **Animal Behaviour and Drug Action**, Boston, Little, Brown & Co., 1964.

Clark, R. L., and Cumley, R. W., eds.: **Year Book of Cancer, 1963-64**, Chicago, Year Book Medical Publishers, 1963.

Commission on Drug Safety: **Report of the Commission on Drug Safety**, Washington, D. C., Commission on Drug Safety, 1964.

Duke-Elder, S., and Cook, C.: **System of Ophthalmology**, vol. 3, "Normal and Abnormal Development," pt. 1, "Embryology," St. Louis, C. V. Mosby Co., 1963.

Ehrenwald, J.: **Neurosis in the Family and Patterns of Psychosocial Defense**, New York, Hoeber Medical Division, Harper & Row, Publishers, 1963.

Evans, F. T., and Gray, C., eds.: **Modern Trends in Anaesthesia**, series 2, "Aspects of Hydrogen Ion Regulation and Biochemistry in Anaesthesia," Washington, Butterworths, 1962.

Farberow, N. L., and Shneidman, E. S., eds.: **Cry for Help**, New York, McGraw-Hill Book Co., Inc., 1961.

Flint, T., Jr.: **Emergency Treatment and Management**, 3rd ed., Philadelphia, W. B. Saunders Co., 1964.

Fox, S. A.: **Ophthalmic Plastic Surgery**, 3rd ed., New York, Grune & Stratton, Inc., 1963.

Gambrell, H.: **Anson Jones, The Last President of Texas**, 2nd ed., Austin, University of Texas Press, 1964.

Gammelgarrd, A.: **On Rare, Weak A Antigens in Man**, Washington, D. C., Walter Reed Army Institute of Research, Walter Reed Army Medical Center, 1964.

Garrett, G.: **American Story**, Chicago, Henry Regnery Co., 1955.

Girard, L. J.: **Corneal Contact Lenses**, St. Louis, C. V. Mosby Co., 1964.

Hadley, L. A.: **Anatomico-Roentgenographic Studies of the Spine**, Springfield, Charles C Thomas, 1964.

Hamilton, J. A.: **Postpartum Psychiatric Problems**, St. Louis, C. V. Mosby Co., 1962.

Harvey, W. P., et al., eds.: **Year Book of Cardiovascular and Renal Diseases, 1963-64**, Chicago, Year Book Medical Publishers, 1963.

Imperato, P. J.: **Doctor in the Land of the Lion**, New York, Vantage Press, 1964.

Krugman, S., and Ward, R.: **Infectious Diseases of Children**, 3rd ed., St. Louis, C. V. Mosby Co., 1964.

Lief, H., et al.: **Psychological Basis of Medical Practice**, New York, Hoeber Medical Division, Harper & Row, 1963.

National Congress of Internal Medicine: **Atherosclerosis, Rheumatic Carditis, Malignant Reticulopathies**, Bucharest, Academy of Rumanian Peoples Republic, 1964.

Nelson, W. E.: **Textbook of Pediatrics**, 8th ed., Philadelphia, W. B. Saunders Co., 1964.

Newerla, G. J.: **Medical History in Philately**, Wisconsin, American Topical Association, 1964.

New Orleans Academy of Ophthalmology: **Infectious Diseases of the Conjunctiva and Cornea**, St. Louis, C. V. Mosby Co., 1963.

Paper, E. M., and Kitz, R. J.: **Uptake and Distribution on Anesthetic Agents**, New York, McGraw-Hill Book Co., Inc., 1964.

Ross Conference on Pediatric Research: **Report of the 47th Conference, International Child Health**, Columbus, Ohio, Ross Laboratories, 1964.

Sato, S.: **Pathophysiology of Toxemias of Late Pregnancy**, Tokyo, Igaku Shoin Ltd., 1964.

Strauss, M. B., and Welt, L. G., eds.: **Diseases of the Kidney**, Boston, Little Brown & Co., 1963.

Turner, R. D., ed.: **Office Urology**, New York, Blakiston Division, McGraw-Hill Book Co., Inc., 1963.

U. S. A. F. School of Aerospace Medicine, Aerospace Medical Division, Air Force Systems Command: **Lectures in Aerospace Medicine**, Brooks Air Force Base, Tex., U. S. A. F. School of Aerospace Medicine, 1964.

U. S. Department of Health, Education and Welfare: **Proceedings of World Forum on Syphilis and Other Treponematoses** (Public Health Service, Publication no. 997), Washington, D. C., Government Printing Office, 1962.

NEW JOURNALS

International Anesthesiology Clinics, vol. 2, no. 2, "Shock," Boston, Little, Brown & Co., 1964.

International Ophthalmology Clinics, vol. 4, no. 1, "Affections of the Lids," Boston, Little Brown & Co., 1964.

Modern Treatment, vol. 1, no. 4, "Treatment of Infectious Diseases," New York, Hoeber Medical Division of Harper & Row, 1964.

Radiologic Clinics of North America, vol. 1, no. 2, "Diagnostic Roentgenology of the Chest," 1963; vol. 1, no. 3, "Pediatric Radiology," 1963; and vol. 2, no. 1, "Radiologic Diagnosis of Acute Abdominal Diseases," 1964, Philadelphia, W. B. Saunders Co.

Revue Roumaine de Biochimie, vol. 1, no. 1, 1964, Bucarest, Roumanie, De L'academie De La Republique Populaire Roumaine, 1964.

Revue Roumaine de Medecine Interne, vol. 1, no. 1, 1964, Bucarest, Roumanie, Academie De La Republique Populaire Roumaine, 1964.

Recent Deaths

DR. V. ROHRER

Dr. Vern Rohrer of Fort Worth, a practicing radiologist, remained active in his field until his death from a heart attack on July 21.

He was born in Springtown, Tex., Aug. 16, 1913, and was graduated from Weatherford High School. He earned the B. S. degree at Texas Technological College, Lubbock, and the M. S. degree at North Texas State Teachers College, Denton. He received his M. D. degree at Southwestern Medical School and interned at Gallinger Municipal Hospital, Washington, D. C., in 1944. From 1948 to 1951 he served a residency in radiology at the U. S. Veterans Hospital in Dallas.

DR. V. ROHRER

In 1951 he began his radiology practice in Lubbock but moved to Fort Worth in 1952 where he continued to practice until his death.

Dr. Rohrer was certified by the American Board of Radiology on April 13, 1953. He was a member of Tarrant County Medical Society, Texas Medical Association, American College of Radiology, Radiological Society of North America, Texas Radiological Society and the Dallas-Fort Worth Radiological Society.

Dr. Rohrer was a captain in the United States Army in World War II, a Mason, and a member of the Presbyterian church.

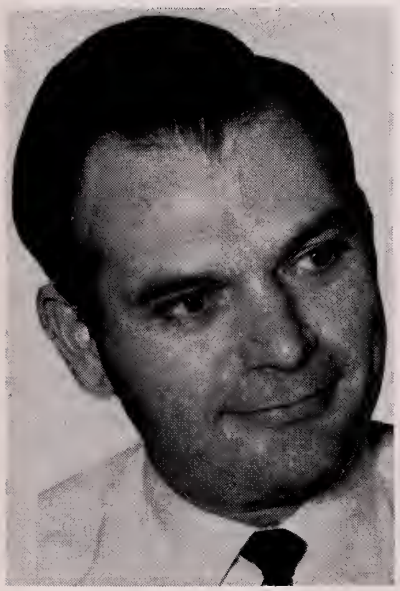
His interests included the Texas Tech Ex-Students Association, fishing, and golf.

Survivors include his mother, Mrs. William Rohrer; his sister Mrs. Helen Rohrer Hinkle, both of Springtown, Tex.; and his brother, Chas. S. Rohrer, Ph.D., Commerce; two nieces; and two nephews.

DR. H. E. SHRADER

Dr. Harold Eugene Shrader of Pasadena died on July 22, at the age of 38.

A native Houstonian, he received his B.A. from Rice Institute in



DR. H. E. SHRADER

1947 and was graduated with honors from Baylor University College of Medicine in 1951. He interned at Brooke Army Hospital, Fort Sam Houston, the following year and was then stationed at Fort Hood until 1953.

Dr. Shrader began general practice in Pasadena in 1954 and served as chief of staff at Southmore Hospital and Clinic for several years.

Among his memberships were Alpha Omega Alpha, Texas Academy of General Practice, Harris County Medical Society, Texas Medical Association, and the American Medical Association. A member of the official board at Sunset Methodist Church, he was also chairman of the board of trustees there. He was also in Rice and Baylor University College of Medicine alumni groups.

Dr. Shrader is survived by his wife, Dorothy Ann; one daughter, Karen Elizabeth; four sons, Jeffery Glenn, Gregory Lynn, Scott Eugene, and Mark Joel, all of Pasadena; and his sister, Mrs. James E. Scott, Houston.

DR. E. H. CHAPIN

Dr. Elisha Hughes Chapin of Houston died Sept. 5. He was physician for four downtown Houston hotels, with his office in the Rice Hotel.

He was born July 20, 1884, in Milan, Ohio. His parents were Leonard B. and Helen H. (Hughes) Chapin.

He attended Milan, Ohio, schools,

DR. E. H. CHAPIN

Kenyon College, Ohio Northern University, and received his M.D. degree from Starling Medical College, Columbus, Ohio, in 1909. From 1909 to 1950 he practiced surgery in Columbus, Ohio. He then moved to Houston where he practiced until his death.

He was a cofounder of the Columbus Radium Hospital in Columbus, Ohio, in 1918, now the Doctors Hospital.

Dr. Chapin had published papers in the Ohio medical journal. He was a member of Harris County Medical Society, Texas Medical Association, American Medical Association, Southern Medical Association, and the Texas Railway and Traumatic Surgeons Association. He was a fellow of the American College of Surgeons.

He was an Episcopalian, a member of Christ Church Cathedral.

Dr. Chapin is survived by his wife, Lucille; a daughter, Mrs. Mary Louise Chapin Burks; and two grandchildren, Mrs. Bettina Burks Brannan, Jr., of Norman, Okla., and Chapin Burks of Long Beach, Calif.

DR. G. W. CROSS

Dr. George Washington Cross, who practiced medicine for 42 years in Yorktown, died Thursday, Sept. 3, in Austin. He was 96.

Dr. Cross was born March 13, 1868, at Nolanville, Tex. His parents were William B. and Elizabeth Cox Cross. The boy grew up in Brown County and was graduated from the Ragsdale Academy at Brownwood. He received his degree in medicine from the University of Kentucky.

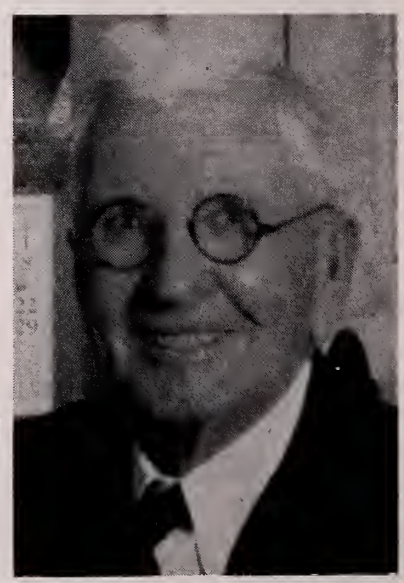
He entered practice in Fayette County and later practiced in Flatonia and Eagle Lake. He moved to Yorktown in 1916.

He married Louisa Allen at Thrifty, Tex., on April 28, 1889. She died July 14, 1955.

A 32nd degree Mason, Dr. Cross often conferred degrees in lodges over a wide area of Texas. He was a past master of the Cameron lodge at Yorktown and a past district deputy of the 34th Masonic District. He was a past Worthy Patron and charter member of the Eastern Star chapter at Yorktown.

The son of a Methodist minister, he was a lifetime member of the Methodist church. He and Mrs. Cross were organizers and charter members of the Yorktown Methodist Church.

Dr. Cross, an honorary member of Texas Medical Association through the DeWitt County Medical Society, is survived by three daughters, Mrs. Cleo Martin and Mrs. Ruthabel Lander of Austin and Mrs. Louisa Esther Cabaniss of Three Rivers; a son, George Cross of Waco; eight grandchildren; and 27 great-grandchildren.



DR. G. W. CROSS

COMING MEETINGS

American Medical Association, Clinical Meeting, Miami Beach, Fla., Nov. 29-Dec. 2, 1964. Annual Meeting, New York City, June 20-24, 1965. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 24-26, 1965; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

NOVEMBER

Gastrointestinal Research Group, Chicago, Nov. 6, 1964. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Radiological Society of North America, Chicago, Nov. 29-Dec. 4, 1964. Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

Southern Medical Association, Memphis, Tenn., Nov. 16-19, 1964. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Texas Dermatological Society, Waco, Nov. 8, 1964. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Division, American Cancer Society, San Antonio, Nov. 18-19, 1964. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Society on Aging, Austin, Nov. 15, 1964. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas State Board of Examiners in Basic Sciences, Fort Worth, Nov. 13-14, 1964. Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

DECEMBER

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 7-9, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of General Practice, San Francisco, April 12-15, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Association of Obstetricians and Gynecologists. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 14-19, 1965. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society. Mr. G. Whittlesey, 521 W. 57th, New York 19, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, New York City, June 17-21, 1965. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition. Dr. Robert A. Peterman, 3 Craig Court, Totowa Boro, N. J., Sec.

American College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Atlantic City, Oct. 18-22, 1965. Dr. Samuel P. Harbison, Pittsburgh, Sec.

American Congress of Physical Medicine and Rehabilitation, Philadelphia, Pa., Aug. 22-27, 1965. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Boca Raton, Fla., Feb. 25-March 2, 1965. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society, New York, May, 1965. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Hospital Association, San Francisco, Aug. 30-Sept. 2, 1965. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Heart Association. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Sec.

American Medical Women's Association, Chicago, Nov. 10-14, 1965. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Neurological Association, Atlantic City, N. J., June 14-16, 1965. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., Chicago, Oct. 18-22, 1965. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Denver, Oct. 23-27, 1965. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Socicty of Clinical Pathologists. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., Chicago, April 9-10, 1965. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons, Las Vegas, Nev., April 25-29, 1965. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Medical Society of United States & Mexico, Phoenix, Ariz., Dec. 9-12, 1964. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Psychiatric Association. Dr. Sullivan Bedell, 1103 Barrs St., Jacksonville, Fla., Sec.

Southwest Allergy Forum, New Orleans, La., March 14-16, 1965. Dr. W. P. Coleman, 1828 Calhoun St., New Orleans, La., Sec.

Southwestern Medical Association. Dr. Zigmund Kosicki, 1220 North Stanton St., El Paso, Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of General Practice, Dallas, Oct. 4-6, 1965. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association for Mental Health. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. Leonard R. Robbins, 3618 Yoakum, Houston, Sec.

Texas Heart Association, Austin, Sept. 17-19, 1965. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, San Antonio, April 28-May 2, 1965. Dr. James K. Stewart, P. O. Box 3311, Beaumont, Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Pediatric Society, San Antonio, Sept. 23-25, 1965. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. Charles Peterson, 1333 Moursund, Houston, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Society of Anesthesiologists, San Antonio, May 1-2, 1965. Dr. Fred P. Thomas, 2342 Maroneal, Houston, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society of Pathologists, Inc., Houston, Jan. 29-31, 1965. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas Surgical Society, Dallas, April 4-6, 1965. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, Sec.

United States-Mexico Border Public Health Association, Los Angeles, April 26-29, 1965. Dr. U. Blanco, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Lubbock, Oct. 9, 1965. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 9-10, 1965. Dr. J. E. Albright, 2481 Medical Center, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 12-14, 1965. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

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Systemic Clues To Occult Neoplasms

IN THE ABSENCE of a magic "cure-all" for cancer, the best available therapy is to detect malignancy as soon as possible. Unusual systemic manifestations are being associated with occult neoplastic lesions with increasing frequency, and early recognition and correlation of these symptoms and signs should help the physician to direct definitive investigation in the "possible" cancer patient. Some clues to occult neoplasms are described herein.

Diseases of Skin.—The accepted incidence of cancer in patients with dermatomyositis is about 15 percent. This relationship increases considerably in patients over age 40, with lesions of the stomach, breast, lung, and ovary being the most frequently diagnosed neoplasms. Other skin diseases which often are associated with an internal cryptic neoplasm include herpes zoster, acanthosis nigricans, Bowen's disease, and erythema gyratum repens.

Vascular Manifestations.—A recurrent or migratory phlebitis which is resistant to therapy and which involves unusual or multiple sites suggests an underlying neoplasm. Lung cancer in men and carcinoma of the reproductive system in women are the malignant lesions reported to be associated with vascular disturbances most frequently.[1] Verrucous endocarditis secondary to an occult neoplasm without evidence of a bacterial disease process may produce arterial embolism in the brain as well as elsewhere in the body.

Skeletal Changes.—Any patient over age 50 who develops an acute exacerbation or onset of rheumatoid arthritis which is resistant to treatment should be investigated for cancer, especially for carcinoma of the lung. Other skeletal clues to an underlying neoplasm are clubbing of the fingers with associated pain, synovitis, and effusion of the joints. These signs may be part of the arthritic-

like syndromes which are found in patients with malignant lesions of the breast, prostate, ovary, stomach, and colon.

Gastrointestinal Clues.—Patients whose presenting symptoms include severe watery diarrhea and hypokalemia should be investigated for an underlying villous adenoma of the rectum or an islet cell adenoma of the pancreas. Although not of gastrointestinal origin, hepatomegaly as well as pulsating osseous metastasis have been reported in patients with hypernephroma.[2]

Metabolic Signs.—Hypercalcemia without evidence of hyperparathyroidism or osseous metastasis may be a rare manifestation of breast, lung, or renal cancer. Similarly, other nonendocrine neoplasms may produce the picture of Cushing's syndrome secondary to an increased production of an ACTH-like substance. A middle-age man who develops hyperglycemia with a severe hypokalemic alkalosis and elevated blood cortisol levels should be investigated for bronchogenic carcinoma and carcinoma of the pancreas. In contrast, recurrent symptoms secondary to episodic hypoglycemia may be noted in patients with a wide variety of occult mesodermal neoplasms such as fibrosarcoma and mesothelioma.

Neuromuscular Symptoms.—In the patient with an occult neoplasm, any level of the nervous system may be involved. The acute onset of symptoms from cortical cerebellar degeneration, manifested by ataxia and incoordination, has been described in patients with carcinoma of the lung, breast, or ovary. Peripheral neuropathies associated with malignancy may be either of the sensory or sensory-motor type. The sudden occurrence of polymyositis in an older person carries the same significance as dermatomyositis. A myasthenia-like syndrome has been observed in patients for several years prior to any clinical evidence of bronchogenic carcinoma.[3]

Although the cause for these syndromes cannot be explained on the basis of our current knowledge of the neoplastic process, the list of unusual symptom complexes that are associated with specific neoplasms continues to grow. Some, undoubtedly, are coincidental. Nevertheless, the physician's awareness of these systemic clues may lead to an earlier diagnosis and prompt treatment of cancer—and subsequently to a better understanding of these heretofore seemingly unrelated symptoms.

—Robert L. Palmer, M.D., Temple.

REFERENCES

1. Lieberman, J. S., et al.: Thrombophlebitis and Cancer, JAMA 177:542 (Aug. 26) 1961.
2. Sultar, R. S., and Levine, H.: Pulsating Metastasis of Bone from Hypernephroma, Conn. Med. 27:11 (Jan.) 1963.
3. Rooke, E. D., et al.: Myasthenia and Malignant Intrathoracic Tumor, M. Clin. North Amer. 44:977 (July) 1960.

The Texas Physician And The American Cancer Society

As PHYSICIANS we are "built in" to the American Cancer Society; we have been from the beginning and we will be until the problem and threat of cancer is finished.

We were the beginning of the Society. In the early 1900's, against a public backdrop of fear, fatalism, and ignorance, committees from the American Medical Association and the American Gynecological Society began working on the problem of cervical cancer. To implement this work, a group of doctors and laymen organized an agency in 1913 for the study and prevention of cancer; they called it The American Cancer Society for the Control of Cancer. This group became the American Cancer Society.

In 1945 the leaders of the Society faced a decision about the group's future. Was it to be almost entirely medical, or was it to reflect the will of both the public and the medical profession? Thus the American Cancer Society, Inc., was formed and the board of directors was enlarged to provide truly national representation.

The Texas Division of ACS was formed during a meeting of the Texas Medical Association. Its bylaws provide that the state board of directors be composed of an equal number of laymen and physicians who are members in good standing of TMA. These state bylaws also set rules of operation which require medical membership at all levels of organization and in all areas of the program.

Thirteen percent of money raised by the American Cancer Society in Texas is spent on professional education to inform physicians of services to their profession. Concrete examples of this are cancer conferences, lectureships for medical schools, traveling teams, program at the TMA annual

meeting, tumor clinics with professional education aspects, professional publications, films and exhibits, fellowships, and special grants.

The theme of the American Cancer Society's extensive public education program is, "See your physician at least once a year for a complete check-up." All of us want to see our patients at least that often.

Three of the Society's main films—"Breast Self-Examination," "Time and Two Women" (dealing with cervical cancer), and "Life Story" (dealing with cancer of the colon and rectum)—must be approved for showing by the county medical society before they may be used, and a physician must be present when they are shown.

Another major area of work by the Society is service to cancer patients. Here the cardinal rule is that no patient may be served without written permission and instructions from the physician (again, providing he is a member in good standing of TMA) in charge of the patient.

"Built in," indeed, the American Cancer Society is "built on" the medical profession. Physicians are active, and must be active, on every policy-making, decision-making level from the local units through the national organization. If we dodge this responsibility we will create a vacuum of leadership which might be filled by "pseudomedical" men.

We want the American Cancer Society "built on" us, because cancer is a health problem, and health problems are our business.

—W. D. SEYBOLD, M.D., Houston,
President, Texas Division,
American Cancer Society.

Medicine's Responsibility To Industry and Its Workers

WHEN INDUSTRY first began to burgeon in Texas it spawned the first rudimentary programs of occupational medicine in the state. As industry became a major factor in the economy of the state, in-plant medicine became a major factor in the health of the worker.

It was fortunate that in Texas we could use to advantage the experiences of the older industries of Europe and of the northeastern United States in applying a program of preventive medicine within the plant; thus we could avoid the major health disasters that typified the rapidly expanding industrialization in other sectors and in earlier times. We started with the important advantage of prior knowledge and we added the results of our own research as the years went by. Today, where industry has recognized its prime responsibilities in providing a safe work environment, that prior knowledge has been utilized to create an enviable record of safety and freedom from toxic hazards. The phrase, "*where industry has recognized its prime responsibilities,*" is of utmost importance. Medicine cannot be expected to function well in areas where problems are hidden or where irresponsible ignorance holds sway. Industry should be encouraged to ask for help when help is needed. Wherever it has asked, the needs have been met by qualified physicians with sane programs of occupational medicine.

Today, more than 100 Texas physicians are active members of the Texas Industrial Medical Association, united by the bond of a common interest in the worker, his environment, and his health. This interest derives from full-time connection with industry, through referral associations with industry, or simply through the general practice of modern medicine. Many times the general practitioner is the first physician to see cases of industrial injury or illness. Whether we are consciously aware of it or not, almost all of us today are engaged in some type of industrial practice. Only about 10 percent of the members of the Texas Industrial Medical Association are engaged in full-time occupational medicine. The other 90 percent are in part-time or referral practice. They make up the backbone of an organization that seeks to provide for the workers of Texas a safe environment and a long, healthy, and rewarding tenure of employment. Through the fulfillment of these aims the worker will benefit, industry thrive, and the state prosper.

Medicine can and should exercise leadership in instituting and improving occupational health programs. The medical profession has a responsibility to bring to the attention of management and labor the intrinsic values that such programs possess.

—R. E. JOYNER, M.D.,
Texas City, President,
Texas Industrial
Medical Association.

Anesthesiology in 1964

IN TEXAS, anesthesiology has grown to a respectable specialty, numbering 268 active practitioners and 37 residents-in-training, but this number still is inadequate. A big increase has occurred since the close of World War II. The War, in fact, was indirectly responsible for many current practitioners. Physicians who had never thought of anesthesia as a specialty had been drafted into taking short courses in anesthesia while in military service and assigned to do anesthesia. These same physicians returned home to give thought to anesthesia as a new specialty; they took further postgraduate work to become qualified anesthesiologists, and some, without further training, practiced part-time.

Surgeons began to appreciate the difference between good and poor anesthetic management and to seek the added assistance of anesthesiologists. Many advances in surgery in the last two decades could not have been achieved without corresponding advances in anesthesiology, such as muscle relaxants, hypothermia, hypotension, extracorporeal circulation, newer nonexplosive anesthetics, and monitoring devices. With each advance in modern surgery, it is more and more necessary to have a physician-anesthesiologist at the head of the table managing the anesthetic. Physicians, with their prior knowledge of pharmacology and physiology, make apt students for learning the essentials of good, safe anesthetic management.

Most anesthesiologists practicing today in the state do so in the larger cities. The Texas Society of Anesthesiologists hopes to increase the number of anesthesiologists to improve anesthetic care and coverage in smaller cities. In May the Society approved the idea of formulating plans for postgraduate refresher courses in anesthesiology to be offered by various medical schools for physicians interested in learning competent anesthesia for at least part-time practice. Announcement of the final plans are expected in early 1965. A survey of anesthesia needs of hospitals in Texas is now being prepared by the Society.

Meanwhile, the goal of anesthesiologists in Texas is to offer the patient a qualified physician to administer his anesthetic needs. By instructing interested physicians in anesthesiology in the smaller Texas cities, quality anesthetic care and coverage can be given to all patients.

—EUGENE L. SLATAPER, M.D., Houston,
President, Texas Society of
Anesthesiologists.

Physicians Have Obligation To Train Successors

EVERY PHYSICIAN currently in practice was at one time a student, resident, or trainee. The medical profession as a whole is certainly responsible for perpetuating and training future physicians. The training of our residents and interns, whether in the university hospital, the charity hospital, or the private institution, has considerable implication for the future. We should make every effort for each intern and resident to receive all the educational experience possible, even to the point of considerable inconvenience and delay of our daily activities.

Approximately half the patients in the country are well cared for in institutions not having residents or interns. The remaining patients are therefore to a greater or lesser degree classifiable as teaching patients (100 percent in pathology and radiology). It is in this area that, having taken responsibility for postgraduate education, we should make the effort above and beyond to insure that the house officer is paid in knowledge if not in money.

The essential ingredients for good training are (1) interest of the physician-teacher, (2) time of the physician-teacher, and (3) utilization of interesting instructive cases including the scientific supporting data.

When we accept the responsibility for excellence in teaching, we insure that the medical profession will continue to advance both from the scientific and the humanitarian points of view.

—G. J. RACE, M.D.,
GENE H. MOORE, M.D., Dallas.

personal opinions and views

Can't Medicine Pick ONE Winner?

PERHAPS IT IS WELL if words of dissent are spread on the pages of our *Journal* lest future historians be misled to believe that doctors of the mid-20th century were all of one persuasion, and that what now may seem controversial and untrue may then be taken as axiomatic truth. We who dissent must have the courage to stand by our minority point of view if for no other reason than to prove the fiber and worth of the men who practiced medicine during this era.

Once I took a personal problem to the great Dr. Felix Miller of El Paso, then Councilor of District 1, and I shall never forget his comment: "Young man, you are a doctor of medicine. You know right from wrong. You do what you believe to be right, and no man who is a doctor of medicine will harm you!"

In the decades that will come, historians, doctors, and statesmen may marvel that the medical profession almost always took the conservative stand against progressive measures. For example, 20 years ago we opposed Blue Cross, fearing that this would lead to socialized medicine. We took an untrusting look at such institutions as the state department of health and took measures to hobble them. Here was a "foot in the door" toward "socialism." Yet today these great institutions are generally recognized as bastions preserving the private practice of medicine.

At a TMA Conference of Medical Society Officials in Austin in January, 1955, an economist, Frank G. Dickinson, Ph.D., Chicago, was explaining why social security was "fiscally unsound." Later that year Dean Clarence Manion, South Bend, Ind., an editorial advisor to the official John Birch Society publication, *American Opinion*, was the principal speaker at a Symposium on Legislation, and railed at the federal government and its sinister designs to sell us out to the communists with socialized medicine as a great first step. At a 1963 session, the speaker was one-time "Life Line" commentator, Wayne Poucher, Washington, D. C. At our District 1 meeting in Pecos a few years ago, the then President of TMA concluded a condemnation of the government and President of the United States of America with these words: "Teach your children to count in rubles! They will need to know how!" He was applauded with a standing ovation.

Most of the doctors I know believe in the good purposes of the government of the United States of America. They all have some misgivings and divergence of opinion on any matter, but deep down there is loyalty and confidence in our government.

Why does our state medical association take such a strongly conservative position? There are, of course, many reasons. First, it is because of a prevailing deep conviction that that course is correct and just. Each of us, as we go through life, should pause occasionally and question seriously the integrity of our thinking. Are the ideas we have today really true, or are we bending with some prejudice? Perhaps, in our college years, we doctors were so loaded with scientific courses that we were exposed to only a smattering knowledge of economics and government. Perhaps activity in medical politics requires the affluence that permits colleagues at home to "take care of the patients" for the participant, and affluence predisposes to conservatism.

Today most Americans accept social security and the graduated income tax, and most serious students of economics and government hold that these systems are necessary to spread purchasing power and give prosperity to all elements of our society. Our government, with its fortunate leadership, has during the past three decades avoided a bloody revolution—the lot of less fortunate people—and has given us instead a healthy balance between private enterprise, government enterprise, and government control and regulation that has yielded more genuine prosperity and personal freedom for American people than at any time throughout all history. We should be grateful. And this is only the beginning. This prosperity can spread throughout the world, ending war and famine and reducing poverty, ignorance, and prejudice. We doctors must recognize this great program and support the only hope for survival in a world

subject to possible total destruction by modern technology.

We doctors opposed federal aid to medical education, although we knew that medical schools had inadequate financing to meet the problem and that the state did not, and perhaps could not, provide adequate schools. We attempted a pusillanimous program of voluntary contributions that eased our conscience and provided perhaps a dollar when a hundred were needed. Today we silently accept the fact that most of the financial support for medical education is federal. We will sink or swim with the United States of America!

We are now opposing medical care for the aged under social security. Actually this is a conservative measure that will become law and will be accepted by the American people. We are backing a program that is not adequate, which has large gaps in service and application, and which requires a pauper's oath that will not be acceptable to American people. Why cannot medical leadership pick a winner in *one* race? If we doctors continue in the role of obstructionists, going down in defeat in race after race, our image will diminish with the charge of selfishness.

What is said here does not propose that the medical profession should forego leadership in the political arena in preservation of those principles necessary for the most effective application of medical science and morality to the needs of people. It does propose that medical leadership should check its hole card occasionally and, as Davy Crockett said so well: "Be sure you are right. Then go ahead!"

—W. E. LOCKHART, M.D., Alpine.

COMMENT

The foregoing opinion does not reflect the thinking of the majority of American physicians; however, if we are to adhere to the principles of democracy, the minority should be heard. I must take exception to Dr. Lockhart's statement that we are backing an inadequate program. Factual analysis of our experience with the application of the Kerr-Mills Act in Texas during the past two years reveals that the true need has been met far more adequately than could be expected from the King-Anderson bill, a token measure and a political football. Since the constitutional amendment providing for the Medical Aid to the Aged program was approved by the voters of Texas in November, it is obvious that organized medicine has sought and found the solution to the problem of financing the health care needs of our senior citizens.

—ROBERT MAYO TENERY, M.D., Waxahachie,
Past President, Texas Medical Association.

Testing Drugs in Animals Not Conclusive for Man

Metabolic differences between man and animals complicate the extrapolation of animal data to man. Therapeutic and toxic doses of a drug may vary from species to species, points out Dr. Harold M. Peck, a Merck Sharp and Dohme researcher.

If it requires a lesser amount of a drug on a milligram per kilogram of body weight basis to bring about the desired biological activity in an animal than it does in man, it would be unrealistic and misleading to use the greater human dose in evaluating the safety of the drug in animals. Also, if the therapeutic dose in animals is higher than for man, preclinical studies would result in a false sense of security regarding the safety of the dosage when used in man. A good example of this is the drug thalidomide which required a much greater dose to bring about birth defects in animals than it did in humans.

Hard Work, Opportunity Await County President

A man chosen as a county society president must have a comprehensive knowledge of the policies of the county as well as the Texas and American Medical Associations, and be in accord with them. If he cannot conscientiously and wholeheartedly endorse these policies, he should never accept the office.

He should have a well developed plan, outlining everything he hopes his society can accomplish during the coming year. After careful deliberation and consultation, he must be able to make decisions and then follow through. One who has been active in many phases of his society's program and has served on TMA committees and councils will be better qualified to develop and carry out an effective program.

The selection of capable committee chairmen, who will help accomplish the stated goals, is of major importance. It is advisable to allow each chairman to choose those with whom he can work best. No member should serve on more than one committee, and the president should encourage maximum participation of all society members.

Since committee members are appointed for specific duties, they should be replaced if they do not perform these duties, and committees that are no longer needed should be dissolved.

The president's program should be planned and his committee chairmen selected while he is president-elect. To delay until after his installation will cost valuable time, resulting in a less effective program.

Responsibility of leadership in all community medical affairs rests with the president of the county medical society. He must be well informed on medical subjects and issues, and available at all times to represent the profession and to speak for his society. He has many opportunities to improve the image of the doctor by personal participation in the political, civic, and religious life of his community and by encouraging his colleagues to do so.

Mat E Johnson

President
Texas Medical Association

Treatment Planning in Radiotherapy

REVIEW ARTICLES

BEFORE MEGAVOLTAGE equipment came into use, the radiotherapist was often required to devise elaborate multiport plans to effectively irradiate deep internal structures and spare skin from severe radiation injury. Although the increasing availability of megavoltage equipment gives the therapist a skin-sparing effect and better depth penetration, the burden of precise dosimetry has not been lifted. On the contrary it has become increasingly important since the warning signals of skin reaction are often not present, and lurking under an almost normal-appearing skin there may be profound radiation effects which constitute a severe hazard to the patient. Therefore, treatment must be planned to give maximum radiation effect upon tumor where it is desired while avoiding the severe complications of radiotherapy to normal tissue.

Two factors which strongly influence the planning of treatment must be considered prior to undertaking the mechanics of the treatment plan. First, the therapist must establish a diagnosis and define the goal of therapy. Second, he must establish the extent and location of the disease. Most patients come to the therapist with a histologic diagnosis from biopsy material or cytologic studies. Cytologic diagnoses are less reassuring and must have further confirmation by radiographic or clinical appraisal of the disease. Occasionally diagnosis must rely on radiographic and physical examination alone. However it should be emphasized that errors are most likely to be made when reliance is placed upon clinical evaluation. The success or failure of treatment is more likely to depend upon the therapist's ability to determine extent and location of disease than upon any other factor. In instances in which the extent of cancer defies definition because its locus is inaccessible to examination, or because there are metastases which are still so small that they escape detection, the chances of successful treatment are indeed remote.

Methods of Investigation

Since x-ray therapy can be effective only if directed to known disease, methods of investigation of the extent of disease must be fully utilized. Methods of such evaluation are physical examination, endoscopic procedures, radiographic examination, and surgical exploration. The investigation may be concluded simply if disease is located superficially, and inspection and palpation may suffice both for localization and beam directing. Endoscopic procedures adequately demonstrate and localize a few lesions and if incorporated with some means of opaque marking, localization films can confirm placement and adequacy of treatment portals. Particularly useful has been the suspension laryngoscope for outlining lesions of the hypopharynx and supraglottic regions. Radiographic examinations are most helpful especially since field localization may be recorded simultaneously. Special views may be needed to show a mass in profile, and tomography will reveal depth or extent of disease not appreciated on routine films. X-ray contrast studies are particularly helpful in defining the extent and location of disease. Cholangiography has demonstrated obstructions of the biliary ducts. Lymphangi-

In planning radiotherapy, the diagnosis and the extent and location of cancer are first determined, then a goal for therapy is made. On a contour plan, the tumor and important structures are designated, and the therapist selects fields and notes the amount of radiation which each level of tissue will receive. The dose should give maximum radiation within the individual's tolerance.

PHILIP T. HUDGINS, M.D.
VINCENT P. COLLINS, M.D.

ography demonstrates disease either by obvious direct nodal metastases or by displacement of lymphatic channels revealing an extrinsic mass. In the lymphomas grossly enlarged nodes, undetected by palpation, may be demonstrated by lymphangiography in inguinal and femoral regions. Similarly the excretory urogram demonstrates disease by displacement or obstruction of opacified structures. Barium studies in esophagus and bowel may demonstrate that there is an obstruction or displacement. Air contrast and arteriography are useful in demonstrating the extent of disease in the bladder (Fig. 1).

Occasionally nothing short of surgical exploration will adequately define the limits of disease, and surgeons should be encouraged to assist in helping with the problem of localization. When surgery is chosen as the primary approach to the cancer problem and the disease is found to be nonresectable, considerable advantage can be gained by the accurate placement of opaque markers or surgical clips to be used later in beam directing (Fig. 2). If radon or radioactive gold seeds are available, they can be used both to outline the mass at surgery and to obtain at least part of the desired tumor dose by interstitial therapy, thereby preserving radiation tolerance of surrounding normal tissues.

Two problems come to attention in the surgical exploration. One is to define the extent of disease and another is to define anatomical relationships. The surgeon is often oriented to surgical atlases designed in the anteroposterior (AP) and lateral planes, and at times he may blindly explore the abdomen and define structures by palpation without visual concept of their precise relationships.

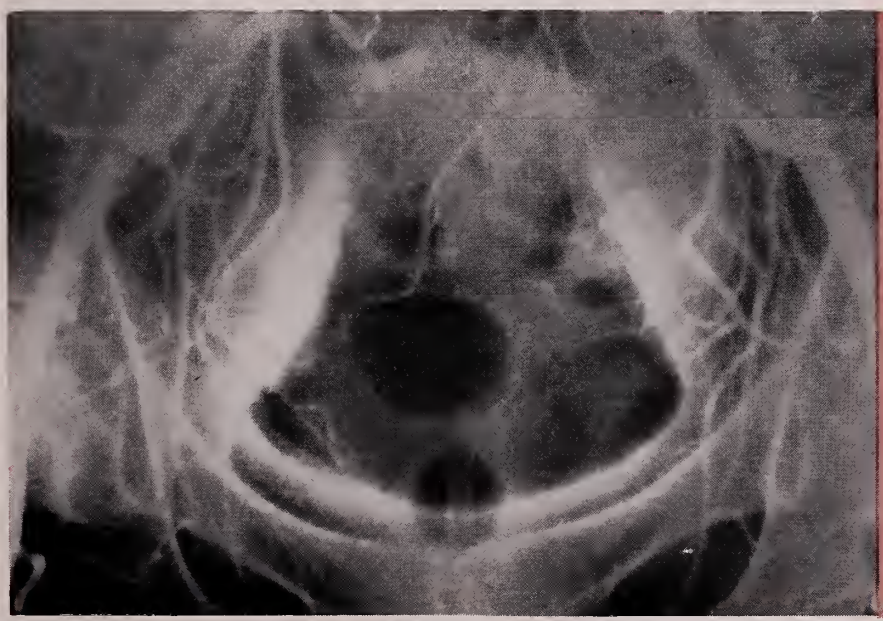

Fig. 1. This angiogram with air in the bladder defines the extent of cancer on the lateral wall.

On the other hand, the therapist must often relate these structures in planes other than the AP and lateral since direction of the x-ray beam must be done through planes and angles other than those routinely visualized. Hence the placement of opaque markers, or the presence of the radiotherapist at the time of exploration, may be extremely helpful. Too often we must rely on a surgeon's note which is more concerned with the types and numbers of sutures closing the various fascial layers than with accurately evaluating the extent of disease. Since the patient's life may be dependent upon the radiation he receives, nothing can be more important than defining the extent of disease. One of the most difficult problems is the evaluation in the postoperative patient with brain tumor. Adequate descriptions and preferably opaque markers placed by the surgeon are desperately needed since there is often reluctance to perform arteriography and air studies between the interval of surgery and the time of treatment planning.

Fig. 2. Radiogold seeds, placed at thoracotomy, define the extent of disease.

Goals of Therapy

Diagnosis having been established, a definite goal of therapy should be decided upon since it will have a direct bearing upon the treatment plan. The goal will influence the volume of tissue to be irradiated and time dose factors.

The goals become the standards by which the results of therapy are measured. Useful terms which define goals and the connotations of these terms are as follows:

Cure.—Although the radiotherapist may be hesitant to claim cure in the face of a diagnosis of cancer there should be no reluctance in having this as a goal of therapy, since it encourages all-out effort. Such a goal must necessarily be considered only in a well circumscribed lesion that can be adequately encompassed within a treatment volume that can be raised to dosage levels that are likely to be effective.

Local Eradication.—When the evident disease is life-threatening and the patient is apt to live beyond a brief interval if such disease is controlled, the tissue volume and level of irradiation should be adequate to eliminate this threat from becoming a recurrent problem.

Local Control.—When life expectancy is short because of extensive local disease or the presence of distant metastases, to eradicate disease in any one particular site may be to give unwarranted additional morbidity and prolonged treatment time which is not justified. In such instances we would choose temporary control for the dominant manifestations of the disease as they become troublesome.

Prophylactic Treatment.—When therapy is offered postoperatively without evident disease, the likelihood of occult disease must be weighed against the likely effectiveness of treatment. The probability of benefit from treatment must be weighed against possibility of complication of therapy when the actual need for such therapy has not been shown. On the other hand a form of prophylactic therapy in a more productive sense would be to offer treatment for asymptomatic bone metastases in an effort to prevent fracture. Also, the dominant or life-threatening metastasis should be treated before a severe clinical problem becomes evident.

Palliation.—This is a commonly used term

which unfortunately connotes futility and implies that only token treatment is to be offered. If such treatment is to be effective it must be well planned. Since cure or even increased survival is not the goal of treatment it is all the more important when palliation is offered to be more specific as to the purpose, e.g., regression of mass, pain relief, or control of hemorrhage. Only by stating and achieving such goals can treatment be justified and evaluated.

Case Reports

Case 1.—A 67-year-old man had primary carcinoma of the hepatic duct with obstructive jaundice of one month's duration. At exploratory laparotomy the surgeon palpated a 3 cm. mass in the porta hepatis invading the liver. A T-tube was placed distal to the obstruction. This did not allow egress of bile but did provide for contrast localization of the tumor with simultaneous placement of treatment portals. Having the radiographic and surgical definition of the extent of disease, a goal in therapy was chosen to achieve local control of disease and relieve jaundice and pain. The patient received small field therapy, 3,500 roentgens in three weeks, directed to encompass the site of obstruction and believed extent of disease. Jaundice and pain were relieved and the patient was free from symptoms for nine months before progression was evident.

Case 2.—A 27-year-old woman had extensive Hodgkin's disease involving the abdomen and mediastinal and superficial lymph nodes. She had received multiple treatments to the abdomen and superficial nodes for one year and then developed bilateral chylothorax (Fig. 3a). Thoracentesis had been required biweekly because of extreme dyspnea. She returned to radiotherapy and a lymphangiogram disclosed an extrinsic pressure mass on the thoracic duct with actual leakage from the thoracic duct demonstrated (Fig. 3b). It was believed that local control of the disease could be achieved by multiple small fields directed to the defect of the thoracic duct, and that if the obstructive phenomena were relieved spontaneous healing of the duct might occur. Therapy was directed to the lesion and within three weeks thoracenteses were no longer required. There has been no local recurrence in over a year, and roentgenograms of the chest are normal (Fig. 3c).

Case 3.—This 71-year-old man had obstruction of the esophagus caused by extrinsic pressure from a carcinoma of the lung (Fig. 4a). The lesion was small and well defined and it was believed that local eradication could be achieved. Small field therapy was directed to the lesion, 4500 r in four weeks. The esophagus opened and remained patent until the patient died of disseminated disease eight months later (Fig. 4b).

Case 4.—A 23-year-old man had teratocarcinoma of the testis and multiple metastases. He had an orchiectomy six months before being referred for radiotherapy, and when first seen by the radiologist, the patient had severe left hip pain with limitation of motion and a lytic lesion involving the acetabulum (Fig. 5a), in addition to multiple pulmonary infiltrates. Life expectancy was believed to be short but local control of the hip lesion was needed. A radiogold implant was performed in the left hip giving 3500 r to the area of lysis. The lesion recalcified (Fig. 5b) and the patient became ambulatory. Two months later acute high intestinal obstruction developed. A gastrointestinal series demonstrated the site to be in the third portion of the duodenum (Fig. 5c, d, e). As palliation, "cross-firing" fields were directed to the site of obstruction and 500 r was given in a single dose. The next day the patient was taking food without difficulty. The disease remained under good control for another three weeks when the tumor invaded the cord at the third cervical vertebra level and the patient died.

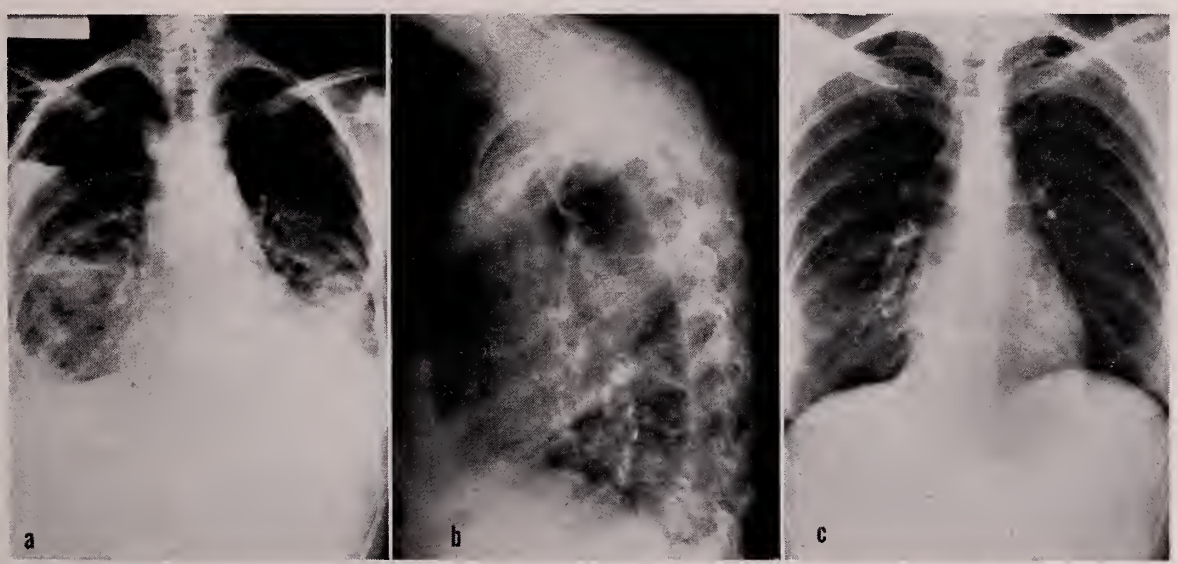

Fig. 3a. Bilateral chylothorax. b. Mass is shown to deviate the thoracic duct. c. Follow-up film one year after therapy.

Other instances of ureteral, vena caval, and bronchial obstruction may be treated in much the same way, and symptom free intervals can be expected although cure is not achieved. Central nervous system symptoms in metastatic or primary brain tumor often subside following therapy and the patient may return to normal occupation and social life.

Techniques of Planning

Once the prerequisites to treatment planning have been fulfilled, application of technique is begun. A volume of tissue is chosen to which an x-ray dose is to be given. The volume of tissue is such that it will accomplish the objective of therapy and yet be as small as is practical. There is less chance of success if dose must be decreased because a large volume is chosen. The side effects are lessened by choosing smaller volumes and decreasing irradiation of normal tissue.

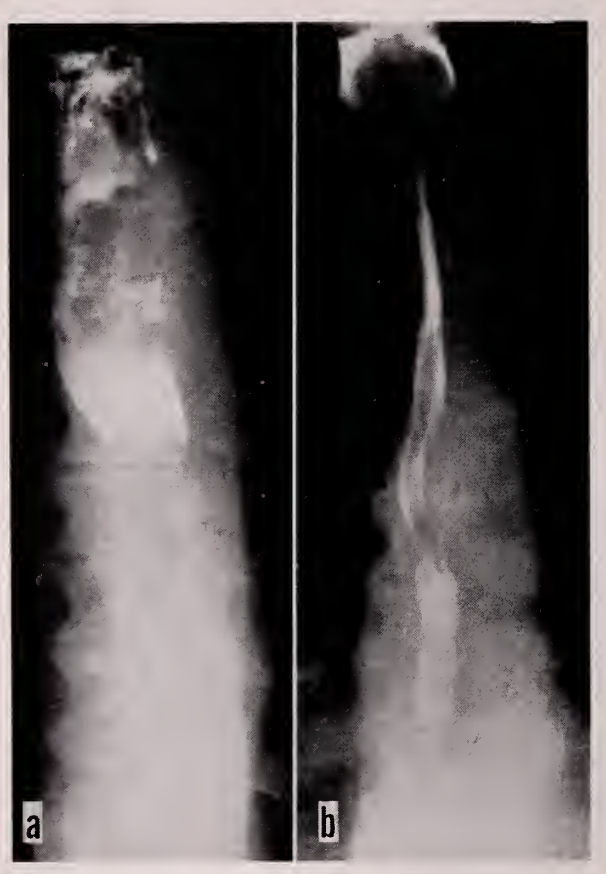

Fig. 4a. Esophageal obstruction before therapy. b. Seven months after therapy, the esophagus remains open.

When smaller volumes are chosen, directing of the x-ray beam must be precise if geographic misses are to be avoided. The preliminaries of diagnosis with definition of the extent of disease and its location having been determined and a goal of therapy decided upon, the specifics of treatment planning technique are undertaken.

A very simple device for the use of multiple fields on superficial lesions is the wobble field. Here tumor localization is by palpation of the mass, and x-ray beam directing is by observation of the x-ray beam light localizer. The incident x-ray beam is approximately 15 degrees from the perpendicular to the skin surface. The calculated daily dose is interrupted two to three times while the patient is rotated about the perpendicular axis. This gives two to three fields for therapy and has the advantage of dispersing the exit dose (Fig. 6).

In planning therapy either with fixed or moving beam techniques, a contour of the patient at the level of the tumor and in the plane of treatment is made. Plaster of paris-impregnated gauze is a convenient contour material and has sufficient rigidity to maintain its shape when removed from the patient. The contour is then traced on to the planning sheet. It is important to note that the patient must be in the position of treatment when the contours are made, otherwise sagging and shaping of tissue may affect dimensions and contour significantly.

Next, from the x-ray film the position of tumor relative to the contour is determined and the tumor is sketched into its proper position. Measurements can be made directly from the film if the proper magnification factor is available. This factor is easily obtained if radiographs have been made with a magnification ring of known dimensions and placed in the tumor plane which is parallel with the film cassette. The ring has the advantage that it cannot be foreshortened in all dimensions and the greatest diameter projected on the film will be the true magnification factor. In addition to tumor placement, important anatomic structures should be placed on the plan since they may influence the placement of the fields as well as the dosage prescribed. It is especially important to know the relative positions of spinal cord, bowel, bladder, and kidney so their functions will not be jeopardized by excessive irradiation.

The contour having been completed, selec-

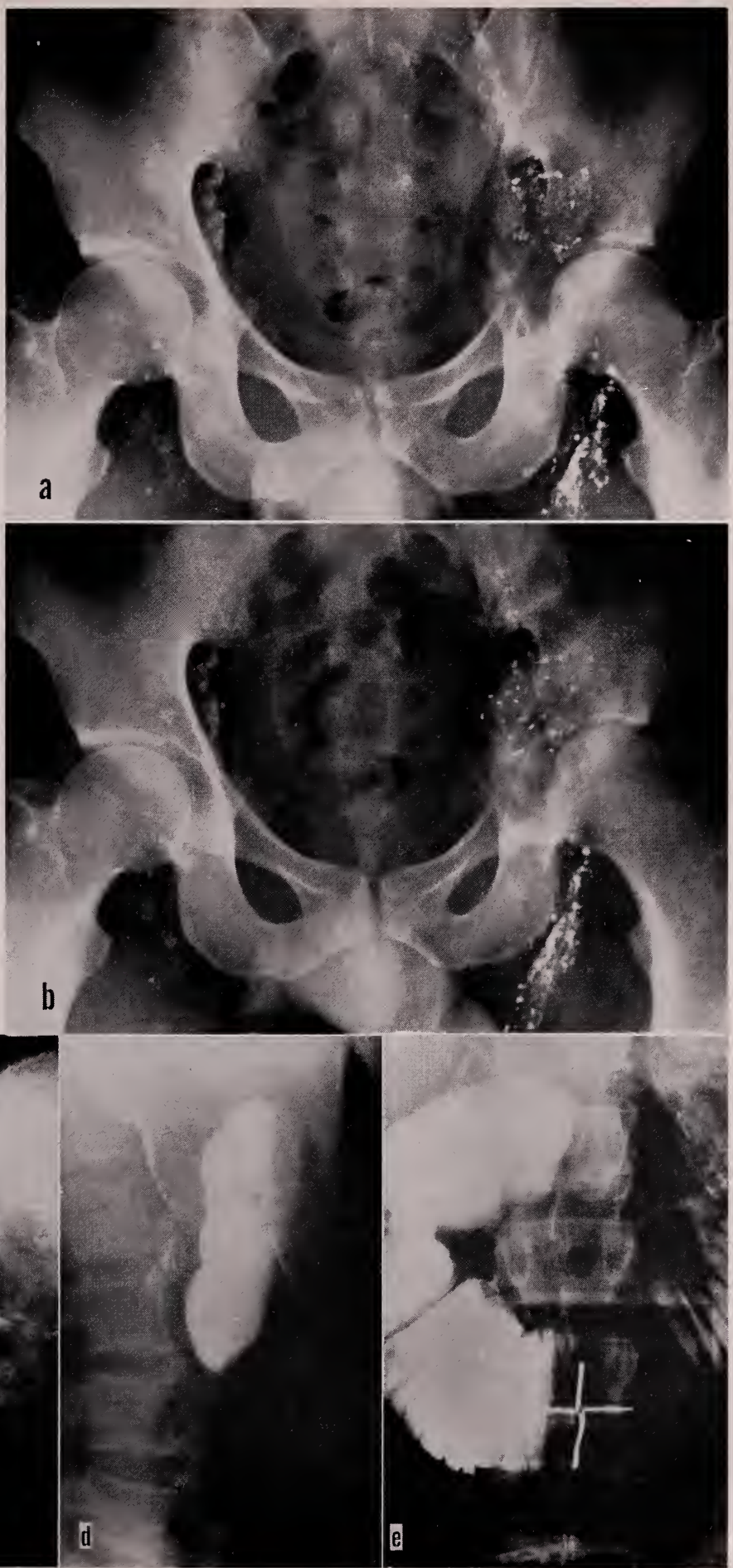

Fig. 5a. Lytic lesion is seen in the left acetabulum. b. Four weeks after implantation with radiogold, the lesion is recalcified. c. Anteroposterior view of obstruction of the duodenum. d. Lateral view of obstruction of the duodenum. e. Localization film of the area treated by x-ray.

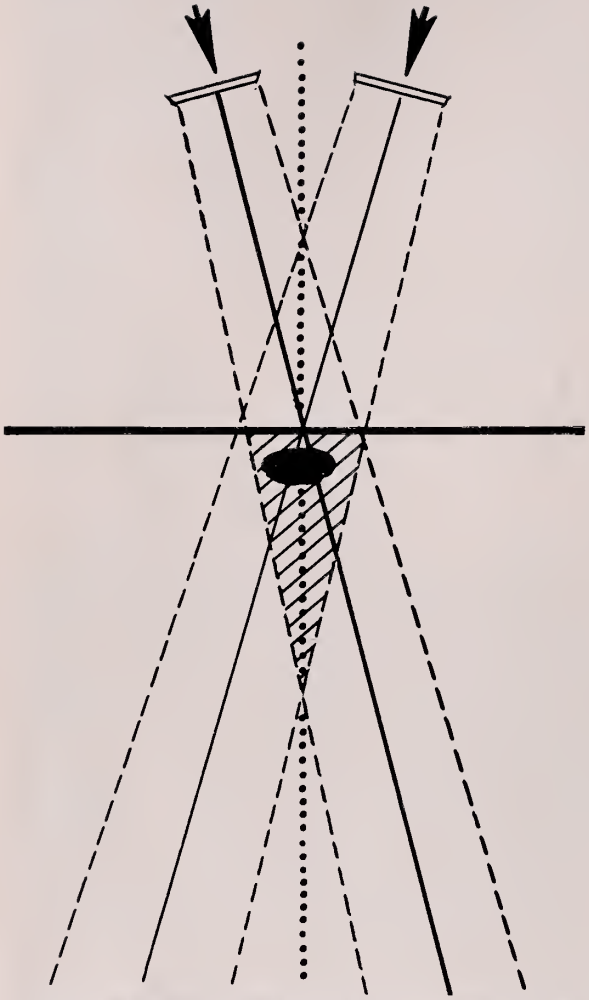

Fig. 6. The effect of dispersing exit dose from a wobble field. The heavily shaded area represents the area included in the combined fields.

tion of treatment fields is considered. In general, the smallest field which will adequately encompass the tumor volume is chosen. The number of fields and their placement will depend upon the relative position of the tumor as well as the equipment available. In those tumors which are relatively centrally located in the body contour, a "cross-firing" technique or rotation is chosen.

In "cross-firing," three or more fields are directed toward the tumor. By altering the angles or the points of convergence of the beams, the shape of the central isodose pattern can be molded (Fig. 7). Arrangement of fields is also made to avoid excessive radiation to vital sensitive structures as depicted in the contour plan. Transparencies of isodose curves are placed beneath the planning sheet in the most advantageous position and the dose contributions from each field are summed and recorded on the treatment plan.

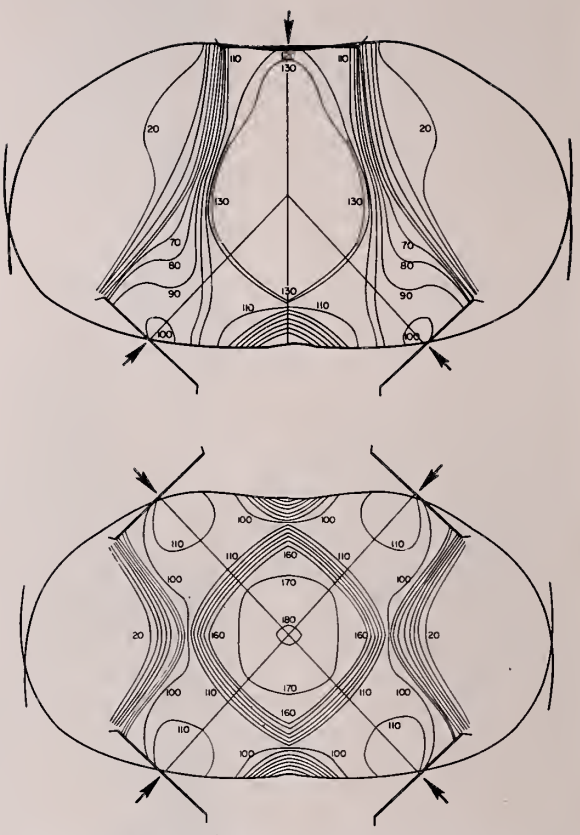

Fig. 7. The effect of three-field (top) and four-field (bottom) arrangement on the shape of the central isodose curve.

By using multiple fields it becomes evident that a desirable tumor-skin dose ratio can be attained. Measurements can then be taken directly from the plan which will give the angle of the x-ray beam entrance and the measurements for rod and pin settings when pin and arc beam directing is used. The pin and arc device assures consistency in beam direction in each treatment session. The only other requirement is to determine a fixed reference point on the patient for placement of the pin. The patient must be placed in the treatment position for localization of the reference point since shifting of skin and internal organs may displace the field away from the tumor. X-ray film documentation of the treatment field through the reference point and including the tumor gives assurance of proper placement of the reference mark. A small dot with silver nitrate on the skin or relation to fixed bony landmarks obviates the need for large, unsightly painted field entrances. Further assurance of proper depth for the pin setting is provided by taking a localization film with the treatment machine and using the proper port size with pin and arc settings.

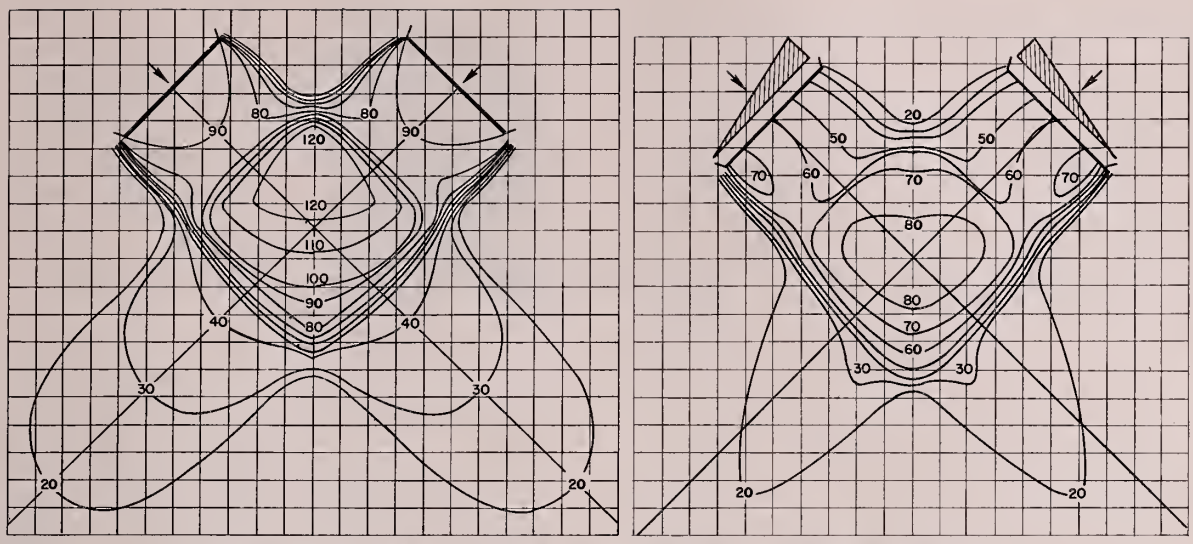

Fig. 8. Shown is the effect of a wedge filter on the shape of the central isodose curve. The numbers represent percent of metered dose. (5 by 5 cm. fields with 10 cm. separation.)

Many rather elaborate techniques for rotation therapy have been described. These consist of spiral, conical, pendulum, and scanning procedures—all of which have limited application in special situations. For the most part, rotation has been adapted for treating lesions which are situated within the central two-thirds of the body contour. Vertical rotation means that the x-ray beam is fixed and the patient is placed on a rotating chair. Horizontal rotation means that the patient is positioned on a treatment couch and the radiation source rotated about him. With either of these two methods treatment planning is essentially the same. A reference mark on the skin is utilized in centering the x-ray beam over the tumor. By measurements from radiographs, the tumor depth is determined and the tumor is placed at the axis of rotation. Isodose distributions are recorded on the plan by any one of several methods, one being to calculate the depth dose distributions from multiple radii and correcting for varying source-to-skin distances.

Eccentrically placed lesions within the body contour often can be treated to advantage with the use of wedge filters. Such filters are designed to alter the isodose distribution curve, generally at 45 degrees, and when multiple fields are used a relatively large zone of homogeneous radiation can be achieved for the eccentrically placed lesion (Figs. 8 and 9).

In summing the isodose distributions from multiport therapy or in the treatment of lesions through irregular areas, compensation should be made for irregular body contour or severe tissue inhomogeneity. The use of tissue compensators is designed to correct for the descrepancies in body tissues and irregularities of contour.

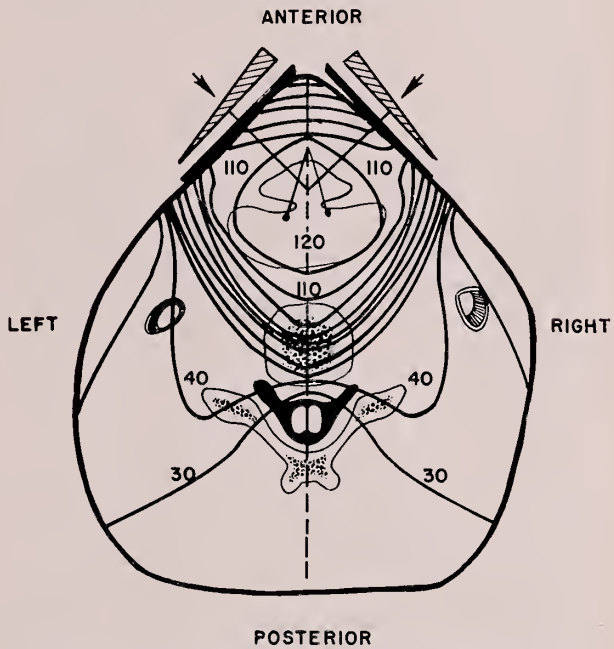

Fig. 9. Shown is the completed plan for irradiation of a carcinoma of the larynx, with important structures defined.

Prescribing the Dose

The most troublesome feature of treatment planning is the prescription of dose. There is a tendency to reduce the answer to a number of roentgens or rads, but any such

straightforward simplicity is illusory since it is hedged with essential qualifications, whether these are described in detail or taken for granted by the initiate.

Dose is the distilled experience of predicting the relation between a medical agent, whether drugs or radiation, and the biologic effect produced. Neither the bare numbers from the pharmacopoeia, nor the bare numbers from the tables and charts of the physics text will provide adequate information to prescribe drugs or radiotherapy. The training consists of providing an orderly approach to organizing experience so that at first simple, and then more complex, relationships can be predicted with useful certainty. The danger of adopting the experience of someone else is expressed in an admonition put forth by Boden, "Your roentgen may be hotter than mine."[1] He was cautioning that from one institution to another, techniques of dosimetry and treatment may vary in unrecognized details so that a dose giving satisfactory results in one institution may be disastrous in another.

The principles in establishing dose schedules in a department are simple. Identify the possible variables and reduce them as far as possible by adopting a limited range of factors, times, doses, and techniques. Approach the maximum therapeutic doses by gradual increments based on conservative estimates of tolerance.

The Strandqvist chart[3] is the classic example of time-dose relationship but it applies directly only to skin cancer and does not take surface area into account. Paterson[5] provides graphs for skin tolerance doses varying with field size. The more complex relationship between volume, dose, and time has been described by Garcia[2] but no widely accepted convention has developed.

The concept of tolerance is a useful guide to prescribing dose. The tolerance dose takes into account not only the amount of radiation and time for delivery, but also the volume and sensitivity of normal tissues irradiated. If cure is the goal of treatment, there is no tumor so predictably sensitive to radiation that anything less than the tolerance dose can be relied upon. If the sensitivity of the tumor is less than that of normal tissues, then deliberately exceeding the dose will not assure cure, and at best may only substitute an unacceptable complication for the tumor being treated.

Dr. Hudgins, who is with the Department of Radiology, Baylor University College of Medicine, presented this paper before the Section on Radiology at the Texas Medical Association's 1964 Annual Session in Houston.

When dose is stated as a number of roentgens or rads, or even if it is elaborated upon in meticulous degree, something must be left unstated. This is commonly encompassed by adhering to some conventions, for example, the rules governing radium needle implants under the Patterson and Parker system. Only a constant effort to improve one's understanding of radiation, biology, and the disease under treatment can diminish the gap that always exists in human communication.

Summary

Two factors which strongly influence the planning of treatment must be considered: (1) The diagnosis must be established. This includes more than merely determining the histology. The extent and localization of cancer must be defined. (2) A goal of therapy must be determined.

Once these prerequisites have been fulfilled a plan is constructed. Contours of the patient are made and drawn on a planning sheet with localization of tumor and important structures on the plan. Advantageous field selections are made and the isodose summations are recorded. A dose prescription is then made based upon experience. This dose is designed to give maximum radiation within the tolerance of the individual.

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Intravenous Abdominal Aortography

MELVYN H. SCHREIBER, M.D., and FRED J. WOLMA, M.D.

CLINICAL MEDICINE

THE ABDOMINAL aorta may be rendered radiographically opaque in several ways. Each has certain hazards and advantages. This article will define the indications for and describe the technique of intravenous aortography.

The object of this procedure is to introduce contrast material into a peripheral vein or veins rapidly enough to assure sufficient concentration to opacify the abdominal aorta and permit accurate diagnosis of any abnormality. Variables that must be controlled or known are speed of injection, volume and concentration of contrast material, circulation time, and speed and timing of film exposures.

Injection

A large bore cannula is necessary if the sizeable volume of contrast material is to be introduced rapidly. A 10 or 12 gauge Robb-Steinberg cannula with a two-way stopcock is ideal. It may be introduced percutaneously (through a tiny nick in the skin to reduce friction), or a cutdown may be performed and the cannula tied in place with ligatures which are later removed (Fig. 1). Local anesthesia is adequate, and the cannula may be kept open with a slow drip of saline solution while preparations for the injection of contrast material are being made.

It is not necessary to use an automatic power injector to obtain a satisfactory study. Manual pressure on a syringe plunger permits delivery of the contrast agent in one to two seconds. If the patient has been properly selected and a cannula of large bore is used, a sufficiently rapid injection can be anticipated. When necessary, injection time may be cut in half if the contrast agent is divided into two parts and injected by two operators simultaneously into two peripheral veins.[3] Although rapid injection is important, one injection site usually is adequate for a properly selected subject.[1]

Contrast Material

Most of the aqueous iodinated contrast agents will produce a satisfactory study. We have used Renografin (76 percent) and Renovist almost exclusively, with satisfactory results. Hypaque-M (90 percent) also has

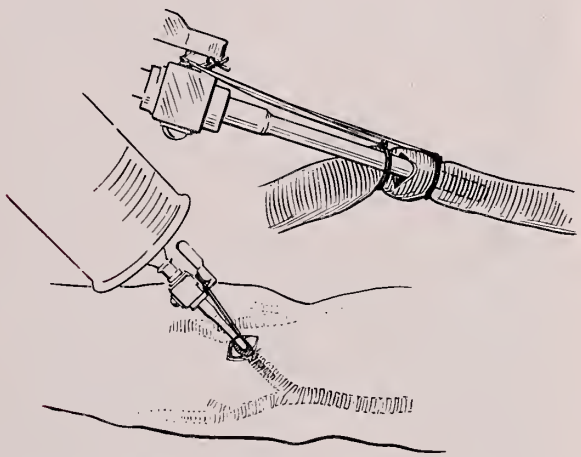


Fig. 1. The Robb-Steinberg cannula, in place within an antecubital vein, is secured by ligatures which are later removed.

The technique of intravenous abdominal aortography is described. It is simple and effective for studying the morbid anatomy in selected patients; hazards are minimal. A variety of vascular structures can be visualized by this method.

been suggested, and several articles indicate the probable acceptability of Angio-Conray.[2]

A small intravenous test dose of the contrast agent is given routinely. If no reaction occurs in five to ten minutes the examination is performed. The usual dose of contrast agent is 1 cc. per kg. of body weight. Although this measure is convenient for estimating an acceptable dose, in practice we rarely use less than 80 cc. and usually use between 80 and 100 cc. of Renografin, depending on the patient's size. Patients weighing less than 130 lb. receive 80 cc.; those weighing between 130 and 160 lb. receive 90 cc.; and those weighing more than 160 lb. receive 100 cc. No patient has received more than 100 cc. at one time. We have not hesitated to repeat the injection one time if necessary to obtain a satisfactory study.

Circulation Time

The arm-to-tongue circulation time is determined using 5 cc. of Decholin, which may be diluted to 10 or 15 cc. with normal saline solution. It is injected as rapidly as possible into the cannulated peripheral vein. An assistant with a stopwatch observes the patient's reaction and records the circulation time. If a sharp "end point" is not obtained, or if the circulation time exceeds 15 seconds, a satisfactory study is unlikely.

Timing Film Exposures

For best results a film changer of the Sanchez-Perez type is desirable. A preliminary or "scout" film is exposed, and after examining this initial film necessary corrections in exposure factors or positioning are made. Films are ordinarily exposed at the rate of one per second during the actual examination. The first film is exposed approximately four seconds before elapse of the measured circulation time, and exposures continue at the rate of one per second for about 12 seconds thereafter. Thus, if the circulation time is 10 seconds, the exposures begin about 6 seconds after the injection is completed and continue for 12 seconds, thereby covering the time of maximum opacification of the abdominal aorta and also allowing some conclusions to be drawn regarding the speed and adequacy of "run off" or drainage.

Selection of Patients

Intravenous aortography provides satisfactory opacification of the abdominal aorta and its major branches and finds its greatest usefulness in patients suspected of having gross aortic lesions such as aneurysms or obstructions (Figs. 2, 3, and 4). Essential to obtaining an examination of high quality are the following factors:

1. The patient must not be obese. Best results are obtained on average-size or thin subjects.
2. The circulation time must be reasonably rapid, and a sharp "end point" must be obtained. Patients with cardiac failure or with aortic or mitral valvular lesions are not satisfactory candidates.
3. The heart must not be enlarged.

Patients who do not fulfill these criteria are best examined by retrograde femoral or brachial catheter aortography, or by translumbar aortography.

Advantages and Hazards

Of all the methods of opacifying the abdominal aorta, intravenous aortography is the simplest and safest. The examination may be performed on outpatients and rarely requires more than an hour. There is no danger of arterial thrombosis or bleeding as with the other methods, and there is no danger of dislodging an arteriosclerotic plaque with catheter or needle. Equipment needed is simple and easy to obtain and use, and fluoroscopy is not needed to confirm proper catheter placement. Versatility in patient positioning is great.

The only hazard of any consequence is the contrast material. Large volumes are used, and in the hypersensitive patient adverse reactions such as cardiovascular or respiratory collapse may occur. Steinberg[3] reported two deaths in 500 cases in which the bilateral simultaneous injection technique was used. Using Renografin or Renovist, we have found no serious reactions in over 100 cases. Transient flushing and a feeling of intense warmth regularly follow the injection, and some patients complain of headache lasting

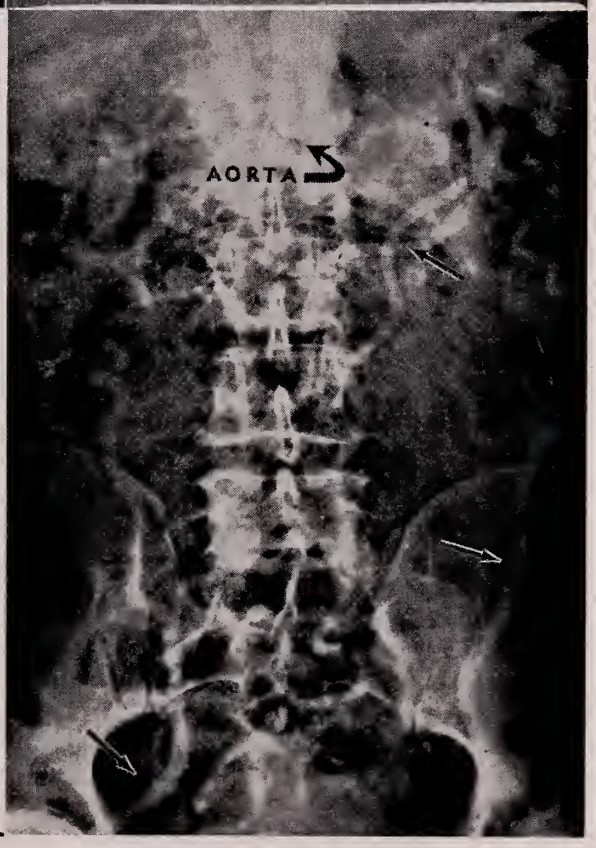


Fig. 2. The patient, an 84-year-old white woman, had a palpable pulsatile abdominal mass in the left lower quadrant, with increasing abdominal discomfort. The aortogram shows a saccular and cylindrical abdominal aortic aneurysm; the renal arteries are not involved. The aneurysm was resected and replaced by an aorto-iliac graft.

Fig. 3. The film shows a saccular aneurysm of the abdominal aorta in a 73-year-old white woman. At operation a 5 by 6 cm. aneurysm was found about 2 cm. below the renal arteries and 3 cm. above the bifurcation of the aorta.

Fig. 4. The patient, a 60-year-old white man, had low back pain and numbness in his legs. The aorta is completely obstructed below the level of the renal arteries, and huge collateral channels supply blood to the trunk and lower extremities (arrows). The x-ray findings were confirmed at operation, and a sympathectomy was performed.

for 10 to 15 minutes thereafter. Occasionally a patient reacts with bouts of coughing and nausea, but rarely with vomiting. None of these minor reactions has required specific treatment, and spontaneous recovery always has ensued within a short time.

Interpretation

With any radiographic examination, thorough familiarity with the normal anatomy of the part examined is essential to correct interpretation. Study of the intravenous aortogram requires a knowledge of the normal shape, course, and branches of the abdominal aorta and of the collateral circulation which develops in the case of obstructions. Of particular importance is the knowledge that many, if not most, abdominal aortic aneurysms contain a sizeable laminated clot within the enlarged lumen. This clot may be only millimeters thick, or may reach 3 to 4 cm. or more in size. Consequently, contrast material may outline a lumen smaller in caliber than the actual aneurysm, and the aortogram may be falsely interpreted as normal if the opacified lumen approaches normal size. However, most such aneurysms will show some enlargement and irregularity of the lumen, permitting the diagnosis to be made. The aorta normally narrows just distal to the origin of the renal arteries (Fig. 5); if it does not, aneurysm should be suspected. In some cases calcium will partly outline the lateral extent of the aneurysm, and an idea of the thickness of the aortic wall can be obtained from the aortogram (Fig. 6).

Variations

By slightly modifying the described method the anatomy of a variety of vascular structures may be demonstrated. While intravenous aortography is not the method of choice for demonstration of small arterial lesions, it can be used successfully for pelvic arteriography (Fig. 7). In the third trimester of pregnancy the technique may be used to demonstrate the placental site in suspected cases of placenta praevia. In such cases, one film of the abdomen is exposed at the circulation time plus two seconds after the end of the injection of contrast material to opacify the placental sinusoids (Fig. 8).

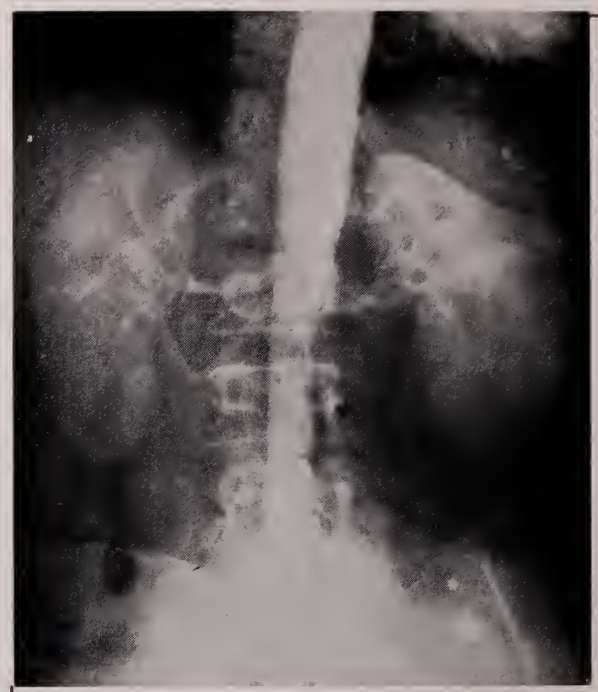


Fig. 5. Normal intravenous aortogram. The abdominal aorta and its major branches are shown. A dense nephrogram is obtained on both sides. Observe the normal narrowing of the aorta distal to the origin of the renal arteries.

Fig. 6. a. Plain film shows curvilinear calcifications outlining a lower abdominal aortic aneurysm.

b and c. Intravenous aortogram shows widening rather than narrowing of the aorta below the renal arteries. A sizeable portion of the lumen of the large aneurysm is filled with a laminated organized clot, preventing filling of the entire aorta.

d. Radiograph of a portion of the resected aortic aneurysm shows an extremely thick aortic wall. The lumen (arrows) is outlined by air.

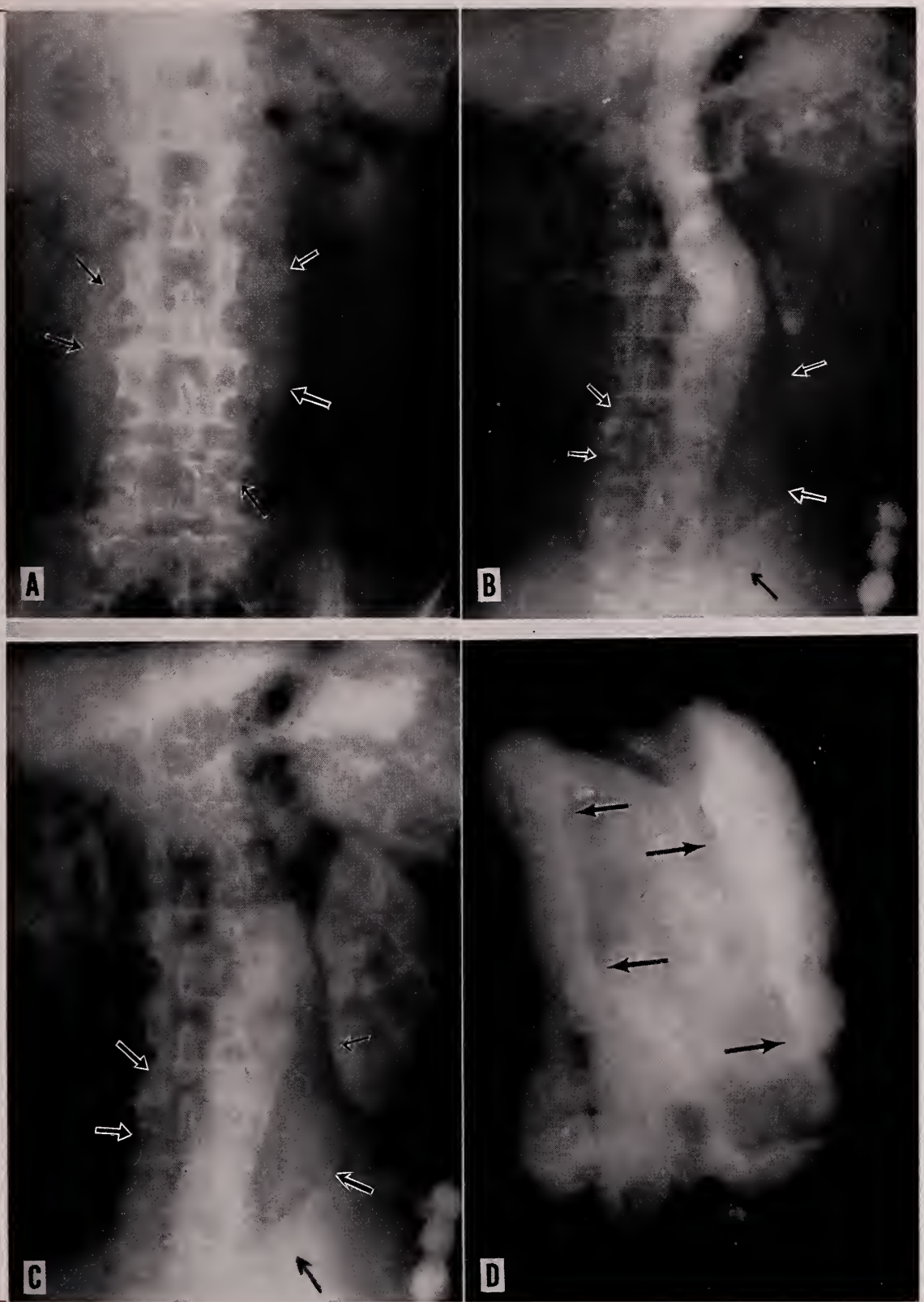
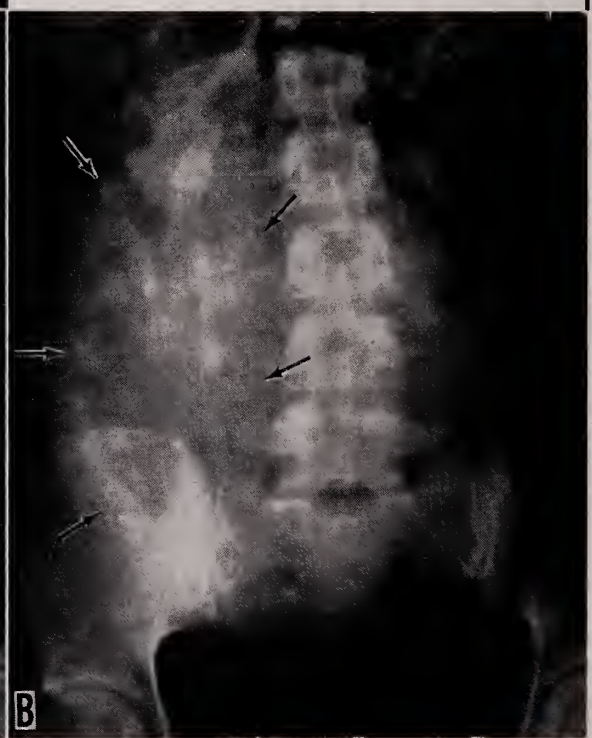

A
B
C
D

Fig. 7. The patient, a 49-year-old colored woman, had a palpable lower abdominal mass about the size of a 24 to 26 weeks' fetus. The mass was inseparable from the uterus on pelvic examination. Intravenous aortogram showed a normal abdominal aorta and major branches. A very large and tortuous ovarian artery (white arrow) was seen on the left. In the pelvis, the large, dilated artery may be the ovarian branch of the uterine artery (black arrow). Operation revealed a large multiloculated pseudomucinous cystadenoma of the left ovary.

Fig. 8. Below. Intravenous placentogram in a case of suspected placenta praevia.
a. Preliminary or "scout" film provides a baseline study with which the subsequent film is compared.
b. Film exposed at the patient's circulation time plus 2 seconds (8+2=10 seconds in this case). Intravenously injected contrast material has traversed the cardiopulmonary circulation and aorta and is seen as discrete densities (arrows) opacifying the placental sinusoids (compare with "scout" film). The placenta is thus located on the right wall of the uterus, and placenta praevia is excluded.



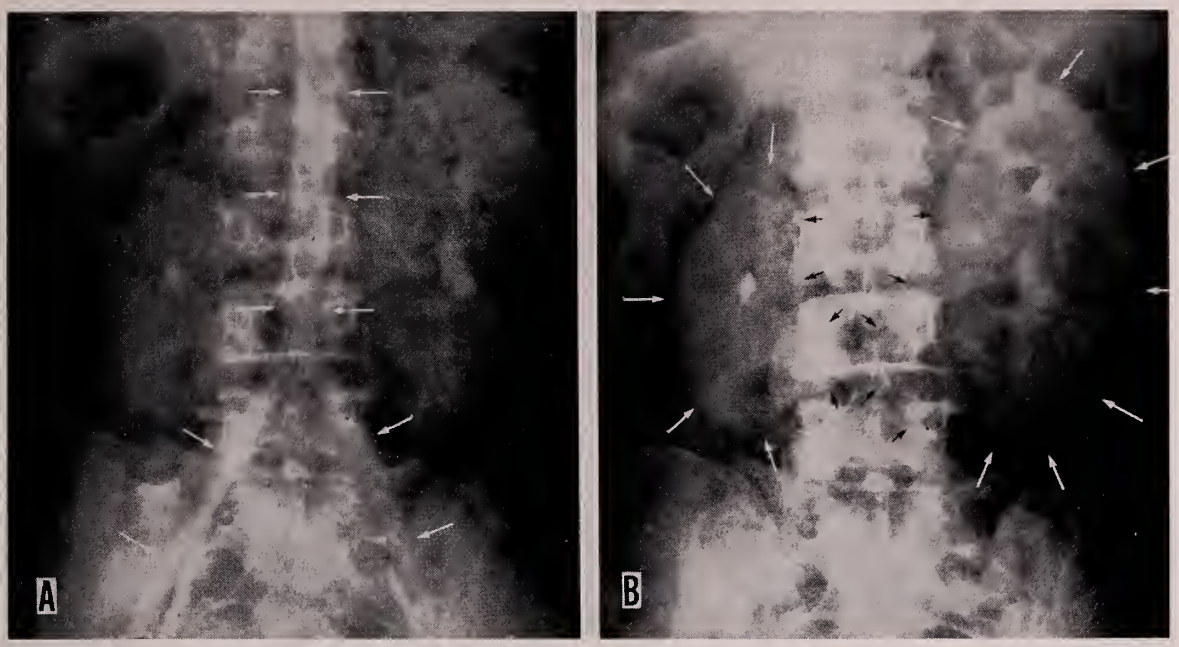


Fig. 9. a. A film exposed at the circulation time clearly shows the aorta and its major branches. No vascular abnormality is seen.

b. A delayed film shows opacification of the renal parenchyma on both sides, permitting a study of renal shape and location. Case of horseshoe kidney.

In intravenous aortography a nephrogram of high quality is obtained routinely and may be used to study suspected areas of segmental renal ischemia or even to define the shape of an abnormal kidney (Fig. 9). In selected cases, tomography may enhance detail and permit precise localization of arteriosclerotic occlusive disease of the abdominal aorta and aorto-iliac system.[4]

Conclusions

Intravenous aortography is a simple, safe, and effective means of demonstrating the morbid anatomy of the abdominal aorta. It is not as satisfactory as translumbar or catheter aortography for elucidation of anatomical details, but it is efficient for study of the gross pathology of the aorta and its branches. Intravenous aortography is particularly useful when aortic aneurysm or occlusive disease is suspected. The method permits numerous variations and has few of the hazards of other forms of aortography. Careful selection of patients and meticulous radiographic technique are essential.

Dr. Schreiber is associate professor of radiology, University of Texas Medical Branch, Galveston, and Dr. Wolma is assistant professor of surgery there. This paper was presented at the Oct. 8, 1963, meeting of the Texas Surgical Society.

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Radiation Treatment In Reflux Esophagitis

RESEARCH & INVESTIGATION

IN SELECTED cases of reflux esophagitis, roentgen irradiation has been an effective method of treatment. The purposes of radiotherapy in such cases are to reduce the incidence of hemorrhage from esophageal varices, in which I believe reflux esophagitis is an accessory factor, and to treat reflux esophagitis per se by reducing the associated pain, spasm, ulcer, and hemorrhage.

To my knowledge, the use of roentgen irradiation in such cases has not been suggested or reported, but I think that it is essentially reasonable and logical for properly selected patients.

Roentgen Irradiation of Stomach

Roentgen irradiation of the stomach for treatment of ulcers has been used for many years. Ricketts and associates have reported the effects of radiation therapy on peptic ulcer in 334 patients.[6] Of these, 298 (89.2 percent) were considered healed. Four radiation techniques were used, and ranges of total depth doses were 666 to 1000, 1100 to 1500, 1600, and 1600 to 2500 roentgens. The recovery rate for patients who received 666 to 1000 r was 70 percent; for those who received 1600 to 2500 r it was 93.8 percent. The presumed reason for this difference is that patients who received the smaller x-ray dosage had less reduction in gastric acidity than those who received the larger dosage.

Although the report from which these figures are taken was published in 1948, radiotherapy for peptic ulcer is still being used.

In a preliminary report in 1959, Kiefer and Smedal[2] gave results of radiation therapy in 23 patients who developed a duodenal ulcer opposite the stoma following subtotal gastrectomy. The treatment was intended to lower the acidity in the gastric remnant, thereby relieving pain and allowing the anastomotic ulcer to heal. Patients received approximately 1800 r during ten treatments given over a two-week period. Results were clinically satisfactory in 15 patients after only one course of radiotherapy, and in 3 patients after two or three courses of radiotherapy. In 3 patients the treatment resulted in failure, but these patients did not have adequate supervision nor was there opportunity to repeat or supplement their therapy. Two patients died of intercurrent disease.

Technique and Effects Of Gastric Irradiation

The technique of gastric irradiation therapy is simple, and this treatment is safer and better controlled than gastric freezing. The patient ingests barium and the outline of the anterior abdominal wall can be visualized. The fundus and corpus are then marked off in a 15 by 15 cm. portal. Ten treatments are administered over a two-week period, with a total depth dose to the gastric fundus of 2000 r.

Ricketts and associates[5] have reported that moderate irradiation of the stomach leads initially to acute inflammation characterized by hyperemia, edema, hemorrhage, and exudation; erosions and ulcerations are rare. This gastritis may persist in diminishing degree for several months and is directly proportional to the decrease in gastric secretion. Inflammation usually is followed by atrophy —consistently present in patients with anacidity of two or more years' duration. Irradiation gastritis is not accompanied by symptoms.

In selected cases of reflux esophagitis, radiotherapy can be used to reduce the gastric acidity which produces pain, ulceration, and bleeding, and interferes with the healing process.

RALPH D. EICHHORN, M.D.

There is divided opinion regarding the efficacy and wisdom of using high doses of radiation on any benign disease. Also, there are those who doubt that treatment of the fundus is either desirable or satisfactory. Nevertheless, I believe radiotherapy can be a useful adjunct to the treatment of bleeding esophageal varices and reflux esophagitis.

Bleeding Esophageal Varices

Selection of Patients.—In the treatment of bleeding esophageal varices, I propose that roentgen irradiation be used for patients who meet the following criteria:

1. Are over 35 years old.
2. Have varices proven by x-ray, esophagoscopy, or both.
3. Have not improved with conservative management.
4. Have contraindications for surgery, or refuse it.
5. Have gastric acidity shown to be either normal or high.
6. Are not considered to be in a terminal state.

Rationale.—The rationale for radiotherapy in esophageal varices following hemorrhage is based on my belief that the bleeding results primarily from peptic digestion of the esophageal mucosa and the venous wall. Other factors are an increased pressure within the dilated veins of the varix of the esophagus, and weakening of the venous wall caused by dilation. I believe the latter two factors are less important than peptic digestion. Linton states that although the pressure is considerably greater in the veins of the leg, rupture rarely occurs.[3] This is true even though the trauma is far greater. Furthermore, in fatal cases of bleeding varices, postmortem usually shows ulceration or erosion of a varix. It may be argued that occasionally patients will bleed with esophageal varices following vagotomy and resection, but residual acidity may remain as in patients who have recurrent stoma ulcers.

Reflux Esophagitis

Selection of Patients.—I believe that radiotherapy should be considered for patients who have reflux esophagitis when the following conditions are present:

1. The patient is over 35 years old.
2. There is associated severe pain, bleeding, or ulceration.
3. Other medical management has failed or is unsatisfactory.
4. Surgery is either contraindicated or refused.

Rationale.—Pain and spasm, secondary to reflux esophagitis, usually associated with hiatal hernia, are also caused by the reflux of gastric juice into the terminal esophagus leading to inflammation, erosion, and frequently to ulceration. Just as recurrent stomal ulceration is the result of persistence of free acid in the gastric remnant, so I believe that pain, ulceration, inflammation, and erosion are caused by gastric reflux of free hydrochloric acid and peptic digestion of the distal esophagus. (The rare case of achylia associated with esophagitis is an obvious exception.) Esophagitis is accompanied by dysphagia, retrosternal discomfort, heartburn, regurgitation, frequently by bleeding, and occasionally by cough and pulmonary symptomatology in cases in which achalasia and stenosis occur.

The fall in gastric acidity which follows adequate (1600 r) roentgen irradiation of the stomach is illustrated by Ricketts' find-

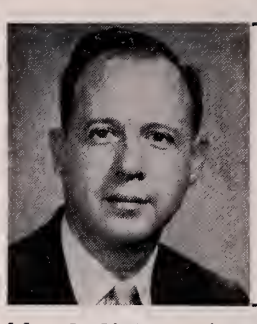
ings in 120 cases: achlorhydria occurred in 56 (47 percent), and acidity was reduced by 50 percent or more in 36 (30 percent).[6] The acidity gradually returns, but in most cases it does not return to the original level. Most important, the free acidity generally diminishes more than the combined or total acidity. In occasional cases, radiotherapy may produce complete gastric atrophy; however, this is unusual. In a study of 116 patients receiving roentgen irradiation for the treatment of peptic ulcer and including numerous cases in which gastric atrophy occurred, no increase in the incidence of carcinoma over that of the general population has been noted.[1]

Treatment

Recent evidence resulting from the vigorous diagnostic approach of Palmer,[4] and the use of early esophagogastroscopy during the variceal bleeding episode, has shown that massive upper gastrointestinal hemorrhage is caused by distal esophagitis and gastritis far more frequently than had been observed previously. It is logical to assume that gastric reflux and digestion play a role in the etiology of this bleeding.

The present day treatment of bleeding esophageal varices consists of emergency treatment utilizing the Sengstaken tube, blood replacement, and other measures; treatment of underlying liver disease; and the various surgical therapies, such as shunt procedures. With the exception of emergency treatment, which stops the immediate bleeding, there is little specific treatment of varices. Therefore, I suggest that we add two simple measures in the treatment of selected cases: (1) Elevation of the head of the bed a minimum of 8 to 11 inches to prevent reflux of acid and pepsin into the distal esophagus. This can be part of both the emergency treatment in the hospital (unless the patient is in shock) and the care of the patient at home. (2) Irradiation therapy to provide a relatively more permanent reduction in gastric acidity. Less acid will activate less pepsinogen to pepsin, and peptic digestion of the distal esophagus will be diminished. Irradiation can be done in those patients who satisfy the criteria already outlined.

Similarly, in patients with reflux esophagitis, in addition to standard therapy of



Dr. Eichhorn presented this paper before the Section on Digestive Diseases at the April, 1964, TMA Annual Session in Houston.

bland diet, antispasmodics, liquid antacids, and elevation of the bed, I would include radiotherapy for those patients who do not respond to conservative medical management and in whom surgery is either refused or contraindicated.

Nine patients who had hiatal hernia with reflux esophagitis and intractable pain have received radiotherapy; they had almost immediate reduction in pain, and relief continued for follow-up periods of several months to three years. Two patients with esophageal varices have been treated with radiotherapy; they did not have further bleeding, but both died later from other causes. One patient received radiotherapy for severe reflux esophagitis following surgical removal of a distal esophageal carcinoma. He received immediate relief of his symptoms persisting for the duration of his life.

Summary

I do not advocate radiotherapy as a universal type of treatment for bleeding esophageal varices, but I believe it should be tried when surgery is contraindicated, unwise, or refused, and when medical treatment fails to produce improvement.

Similarly, I think irradiation has promise as an adjunct to surgery and accepted conservative medical treatment of simple reflux esophagitis in cases which meet the stated criteria for selection of patients.

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Hyperfunction of the Parathyroids

A. O. SINGLETON, JR., M.D., and THOMAS KIRKSEY, M.D.

REVIEW ARTICLES

A PARATHYROID ADENOMA was found in 1903 at autopsy of a patient with osteitis fibrosa cystica, but it was not until 1925 that a clinical cure was effected by removal of a parathyroid tumor.[5] It has been repeatedly demonstrated, in patients without parathyroid function, that the high serum phosphate and low calcium with little phosphorus or calcium in the urine could be converted to a low phosphorus and high serum calcium by administering parathyroid hormone. Phosphate is excreted in the urine and later, calcium is excreted as the serum calcium rises above the renal threshold. Since the blood calcium in nephrectomized animals can be raised with parathyroid hormone, it is assumed that two mechanisms exist in hyperparathyroidism: renal excretion of phosphorus and mobilization of calcium from bone. Although originally associated with bone disease, now it is realized that hyperfunction of the parathyroids may involve many systems. With the greater awareness of this entity, an increasing number of cases are recognized.

In 26 cases studied at the University of Texas Medical Branch in the past ten years, 18 have been recognized since 1960. Similarly, at the University of California Medical Center in San Francisco, the 10 cases collected up to 1954 had increased to 90 cases six years later.[5]

The condition is seen more commonly in women. In our series there were 16 women and 10 men. Their ages are shown in Table 1. There were 18 white and 8 Negro patients.

TABLE 1.—*Ages of Patients.*

Age	Number of Patients
11-20	3
21-30	1
31-40	1
41-50	5
51-60	12
61-70	4

Diagnosis

Although the alert physician may suspect hyperparathyroidism, elevated blood calcium levels or bone changes demonstrated by roentgenogram must be the determining factors for diagnosis. The classical osteitis fibrosa cystica is not often seen in this country because of the high calcium intake. Only 5 of our 26 cases showed bone changes. Huth,[6] however, has reported that 25 percent of patients will show cortical erosion in roentgenograms of the hands. Other changes, including stippling of the skull, may be seen. Alkaline phosphatase changes are as sensitive in detecting bone changes as x-ray. Biopsy and tracer techniques may demonstrate bone changes, but are not specific.

The most common symptom suggesting parathyroid disease consists of calculi in the urinary system, which are present in about 70 percent of cases. Seven percent of patients with urinary stones have hyperparathyroidism. Eighteen of our patients had renal stones or nephrocalcinosis. Only 1 to 2 percent of patients with nephrocalcinosis have hyperparathyroidism, and infection is probably a more important etiologic factor than hypercalcemia. Renal failure and hypertension probably are the most serious

The authors used radioisotopic selenomethione to demonstrate a parathyroid tumor. A review of 26 cases of hyperparathyroidism is presented; various intercurrent symptoms are noted and discussed.

complications of hyperparathyroidism; in advanced cases the changes may be irreversible, and the patient succumbs to the complication even though his hyperparathyroidism is cured.

An increasing number of cases, 9 to 12 percent, have been discovered by serum calcium determinations alone in the absence of any specific symptoms. None of our patients fell in this category, though one patient had an unsuspected parathyroid adenoma found on exploration for a thyroid adenoma.

Peptic ulcers also have been associated with hyperparathyroidism, but it must be realized that abdominal pain associated with hypercalcemia may be confused with ulcer symptoms. Five of our patients had proven peptic ulcers. It is not known whether the parathyroid hormone or the hypercalcemia are ulcer-stimulating factors. Ulcerogenic lesions of the pancreas are associated with multiple endocrine tumors and the question must arise as to whether an undiscovered pancreatic lesion is also present in the patient with hyperparathyroidism and peptic ulcer. We had one patient with hyperparathyroidism whose peptic ulcer did not heal following removal of a parathyroid adenoma. At abdominal operation, ulcerogenic lesions of the pancreas were found. In none of our other four patients with ulcerogenic pancreatic adenomata was hyperparathyroidism detected.[8] Many patients with peptic ulcer will have hypercalcemia from the ingestion of large amounts of milk and soluble alkali.

We have seen a number of patients with lesions of the thyroid and parathyroids. One patient had an associated Hürthle carcinoma found incidentally at time of exploration for the parathyroid lesion. A total thyroidectomy was performed. Ellenberg[2] has reported that in a study of 93 patients with parathyroid adenomas, 49 percent had thyroid nodules while 7.5 percent had carcinomas of the thyroid. A high serum calcium level is probably a goitrogenic agent. Most parathyroid tumors will not be palpated, and masses palpated in the area will usually represent thyroid adenomas. Gordan[5] has also described this finding. Hyperthyroidism also may cause hypercalcemia, which may lead to confusion in this area. The temperature, pulse, and respiration (TRP) are elevated in Graves' disease in contrast to their lowered values in hyperparathyroidism.

Pancreatitis is found in a larger number of cases of hyperparathyroidism than one would expect. The condition may be concealed because neutralization of fatty acids formed by fat necrosis may give normal calcium blood levels. Checking such cases after the acute attacks of pancreatitis may reveal higher levels. There were no cases of pancreatitis in our series.

Hypercalcemia, then, becomes the most important factor in making the diagnosis of hyperparathyroidism, and though the condition may be present in the absence of such a demonstrable finding, few such patients will be subjected to surgical exploration. Repeated determinations and restriction of dietary phosphate intake may finally demonstrate elevations in cases with previously normal levels.

Unfortunately, there are many causes of hypercalcemia. Gordan[5] has found that malignancies are the most common cause for such elevations in his laboratory, occurring even in patients without roentgenographic evidence of bone involvement. Myers[7] has noted that although nephrocalcinosis is a common finding in cancer patients, renal lithiasis is not seen clinically or by roentgenogram. Calcium levels are higher in cancer patients.

Although it has been suggested that parathyroid or vitamin D-like substances are formed by certain tumors, Stone[10] believes that some unknown substance produced by the tumor causes parathyroid stimulation. If this is true, then subtotal parathyroidectomy may be justified for selected patients as a palliative measure.

The value of ionized calcium in diagnosis is debatable, especially if the serum protein is not depressed.[5]

Electrophoretic analysis of serum proteins is important in the differential diagnosis, being diagnostic in myeloma or sarcoidosis.

Cortisone, 150 mg. for ten days, will lower the calcium to normal levels in sarcoidosis, hyperthyroidism, vitamin D intoxication, and Addison's disease, but rarely in hyperparathyroidism.

Hypercalciuria is present in 95 percent of cases, but also may be present with kidney stones and osteolytic disorders.

Hypophosphatemia and hypercalcemia suggest hyperthyroidism, although this is

also found less commonly in malignancies and hypervitaminosis D. Gordan[5] found that 60 percent of his cases of hyperparathyroidism had normal phosphorus levels, however. By demonstrating a renal phosphate leak (TRP < 76 percent) with or without phosphate loading, one may find a hyperparathyroid abnormality; however, such leaks are not specific for hyperparathyroidism.

This may also be seen in secondary hyperparathyroidism (osteomalacia, renal insufficiency, congenital disorder or tubular transport, tubular nephropathy, sarcoidosis, myeloma, and urolithiasis with pyelonephritis).

Induced hypercalcemia is no longer commonly used as a test as parathyroid tumors may show a normal response. Abnormal responses may have some significance, however.

Detection by Radioscanning

In two cases, deformities caused by the tumor were noted on esophagrams.

It has been demonstrated that methione and selenomethione are concentrated in the parathyroid glands. Using radio-tagged material and scanning the neck, Bierwalters has been able to demonstrate a parathyroid adenoma. Dr. Thomas P. Haynie has carried out similar studies at our institution, and has been able to demonstrate a lesion in a recent case of Drs. Fowler, Wright, and Wolma (Figs. 1 and 2). In other cases the lesion was not visualized.

In all of our 26 patients explored for hyperparathyroidism, pathologic parathyroid glands were found. In two patients, multiple lesions were present and three patients had hyperplasia rather than adenomas. One patient had a carcinoma. All four glands were identified in 21 patients. In two patients this was not done, and later re-exploration revealed adenomas. In one case, five glands were isolated. No mediastinal explorations or total thyroidectomies were necessary although in some clinics as many as 28 percent of lesions have been found outside the neck. All patients had relief of evidences of hyperparathyroidism following surgery.

The question arises, in the absence of negative explorations, as to whether an adequate number of cases were being subjected to exploration, as many series show a large number in which no lesion was found. Gordan had three negative explorations in 90 cases but only one of these probably had true hyperparathyroidism.[5] Although Chamberlin[1] originally had as many as 29 percent negative explorations, with more careful tests and criteria this had dropped to none in recent years.

The location of lesions has chiefly been in the lower parathyroid glands. Nine of our adenomas were in the right lower gland but only four in the left. There were four in the right upper gland and four in the left.

The histologic diagnosis of the lesion may be difficult on frozen section. In one of our cases a lesion thought to be a parathyroid adenoma was diagnosed by the pathologist as carcinoma, probably a papillary carcinoma of the thyroid. The patient's hyperparathyroidism was relieved, however, and later it was concluded that this was a parathyroid carcinoma. This was the only malignancy in our series, and the patient died several years later of a ruptured aneurysm of the cystic artery. No evidence of a neoplasm was seen at postmortem.

The treatment of hyperplasia has been subtotal parathyroidectomy which was effective in our cases. None of our patients have had further evidence of hyperparathyroidism. There were no postoperative complications of note. No patients experienced tetany or the low magnesium syndrome which has been described recently.

The apparent absence of other parathyroid tissue should never frighten one into leaving portions of an adenoma behind. We are familiar with one such case in another institution; the symptoms recurred and an extensive carcinoma was found later.

Although hyperparathyroid crisis has not been a problem in our institution, many authors have described this entity.[9] We have had two patients with fairly acute symptoms. Unless surgical intervention is prompt, the condition may be fatal. In some cases other operative procedures may initiate the crisis in chronic hyperparathyroidism. The usual features are rapid onset of nausea, vomiting, constipation, pain in the extremities, back or abdomen, polyuria, polydipsia, and coma. Many patients have fever. Hypercalcemia may not be present. An elevated BUN may be present because of renal failure.

There were four acute cases in a series of 41 parathyroid lesions at the Peter Bent

Brigham Hospital. Atrophy was not present in the uninvolved glands in many cases.[11]

Fink,[3] in a collected series of 56 cases of hyperparathyroid crisis, found a mortality rate of 43 percent. In almost all fatal cases the diagnosis was unsuspected before postmortem examination. Pancreatitis was present in 34 percent and peptic ulcer in 29 percent. An elevation of the serum calcium of 17 mg. per 100 cc. or over, with an increase in the serum phosphorus, should make one suspect a parathyroid crisis.

In elderly people it has been suggested that hyperparathyroidism may run a more acute course.

Summary

Review of 26 cases of hyperparathyroidism seen from 1952 to 1963 at the Univer-

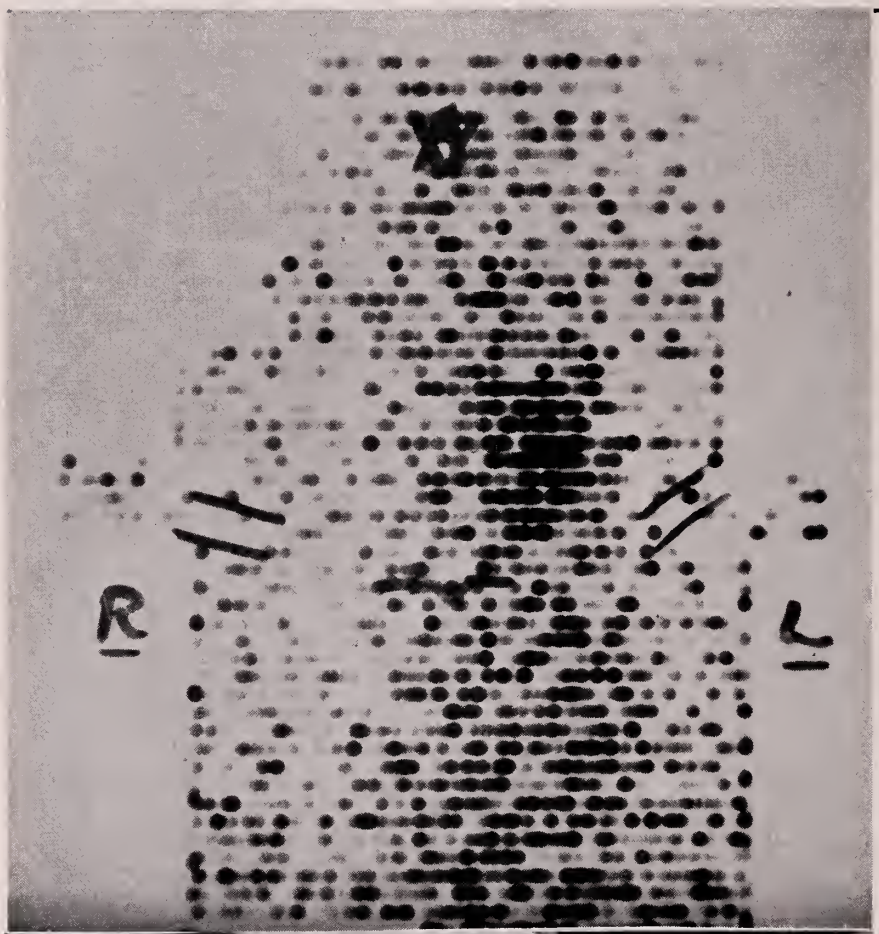

Fig. 1. Neck scan of patient with parathyroid adenoma using radio-tagged selenomethione. Note concentration in left lower parathyroid gland. This material is taken up by the thyroid as well, but a later radioiodine scan showed diffuse general uptake without concentration in any area, ruling out a "hot" thyroid nodule.

Fig. 2. Same patient as in Fig. 1. Large adenoma of left inferior lobe seen at operation.

Drs. Singleton and Kirksey are associated with the University of Texas Medical Branch, Galveston. This paper was first presented before the Texas Surgical Society April 6, 1964.

sity of Texas Medical Branch Hospitals, shows that the incidence of recognition of hyperparathyroidism is increasing. In this series, the condition was more common in women and in the sixth decade of life. Although all patients showed elevated serum calcium determinations, only five showed bone changes. Seventeen of our patients had urinary stones or nephrocalcinosis. Five patients had proven peptic ulcers. One of these also had an ulcerogenic lesion of the pancreas. Two parathyroid adenomas could be demonstrated by esophagram, but most so-called palpable lesions turned out to be associated thyroid adenomas. In a recent case at our institution, a parathyroid tumor was detected by neck scan after administration of radioisotope selenomethione. In 26 patients explored for hyperparathyroidism, 23 had adenomas, 9 in the right lower gland. Two patients had multiple lesions. There were 3 patients with hyperplasia. Frozen section histologic diagnosis may be difficult. All patients have done well since surgery, though 2 had to have two explorations before the condition was resolved. No lesions were present in the mediastinum or within the thyroid itself.

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Virus-Leukemia Relationship Expected To Be Proved

Medical scientists found some strong evidence for the viral theory of human leukemia in 1964, stirring hopes for early proof of a virus-leukemia relationship.

One of the most dramatic developments in the virus-leukemia story comes from England where the first apparent transmission of human cancer to laboratory animals and apparent success in growing a virus-like particle that is assumed to cause the disease has been reported.

Investigators have been testing the viral theory of leukemia in animals for more than 50 years, ever since the discovery of fowl sarcoma virus in 1911. But, as the British medical journal, *The Lancet*, pointed out editorially this year, "It is fair to say that the immense expense of effort, time, and treasure on the study of fowl and mouse leukemias has brought little help to our understanding and management of human leukemia."

Radiographic Diagnosis

Intramural Diverticula Of the Gallbladder

REVIEW ARTICLES

DIAGNOSIS OF intramural diverticula of the gallbladder, or the so-called Rokitansky-Aschoff sinuses, by x-ray is not a common occurrence. This entity has often been called by other names such as adenomyoma, cystadenoma, adenofibromyoma, cholecystitis glandularis proliferans, and others.

Rokitansky was the first to describe these hernia-like outpouchings in the mucosa of the gallbladder. Aschoff, many years later, noted the association of the outpouchings with muscular hyperplasia and cholecystitis, and he concluded they were a direct result of infection. These two primary investigations account for the frequently used name of Rokitansky-Aschoff sinuses for these intramural diverticula of the gallbladder. This is a relatively common entity from a pathologic standpoint. Robertson and Ferguson[5] in 1945 found Rokitansky-Aschoff sinuses in 50 percent of apparently normal gallbladders in patients over age 30, and in 93 percent of gallbladders which were removed surgically. King[3] in 1953 found that 90 percent of diseased gallbladders contained Rokitansky-Aschoff sinuses although only 10 percent showed gross changes. Jutras[2] found evidence of radiographic changes of Rokitansky-Aschoff sinuses in 5 percent of 6,082 cholecystograms; however, he reported an incidence of 33.3 percent in 2,490 surgical specimens following cholecystectomies. Colquhoun found 34 cases in 1,200 unselected cholecystograms, an incidence of 2.8 percent. It is apparent that this entity is relatively common from a pathologic standpoint, but is not as easily recognized on radiographs. The first radiographic report of this entity did not occur until 1948 by March.[4] Better technique of examination and newer contrast media appear to be the factors in the increasing demonstration of this entity on roentgenograms.

Radiographic Features

Roentgenograms may show a combination of findings which consist primarily of halo or bead-like appearance around the filled gallbladder. This may surround the whole gallbladder or only a segment of it. There frequently is narrowing of the gallbladder, either at its fundus, midportion, or tip. Calculi are often noted.

Fig. 1 demonstrates a halo or bead-like appearance around the filled gallbladder, with the narrowing in the midportion giving a so-called hourglass appearance.

Fig. 2 again demonstrates the halo or bead-like ring, however the narrowing in this case is at the tip of the gallbladder and was best noted on the post-fatty meal film. Calculi are also evident.

Fig. 3 shows that the halo or bead sign is faintly visible, but definite, when the film is closely scrutinized. There is a narrowing at the cystic duct noted on the post-fatty meal study.

Discussion

The presence of intramural diverticuli of the gallbladder with the so-called Rokitansky-Aschoff sinuses on roentgenogram are almost tantamount to the diagnosis of chronic gallbladder disease with or without clinical symptoms.

The pathologic findings of these three cases showed chronic cholecystitis despite a good cencentration of the dye on the routine cholecystogram. All three patients had small calculi.

Intramural diverticula of the gallbladder are not uncommon. When demonstrated on roentgenogram they are indicative of gallbladder disease, even though there may be good concentration of dye in the organ.

LESLIE L. LEMAK, M.D.
JAMES M. KEEGAN, M.D.

The origin of cause of intramural diverticula or Rokitansky-Aschoff sinuses has been reviewed by many authors. A combination of an increase of the intracystic pressure during contracture of the gallbladder associated with disease of the wall of the gallbladder has been accepted as the most likely etiology. Others have thought that the causative condition was hyperplasia of glandular structures, hence the name, cholecystitis glandularis proliferans. The etiology of constriction, which apparently is often seen with this disease entity, has not been definitely established; it is noted that there is a hypertrophy of the muscularis mucosae in the region of the constriction. Also noted is that the intramural diverticula appear to project into the thickened portion of the gallbladder wall, with the outer margins of the gallbladder being undisturbed. This

Fig. 1. Shown is the bead-like appearance surrounding the lower two-thirds of the gallbladder. Narrowing of the mid-portion of the gallbladder, giving the so-called hourglass appearance, is also evident.

Fig. 2. The narrowing of the tip of the gallbladder, and several radiolucencies, are evident. Small diverticula are demonstrated in the medial aspect of the distal end of the gallbladder, typical of intramural diverticula.

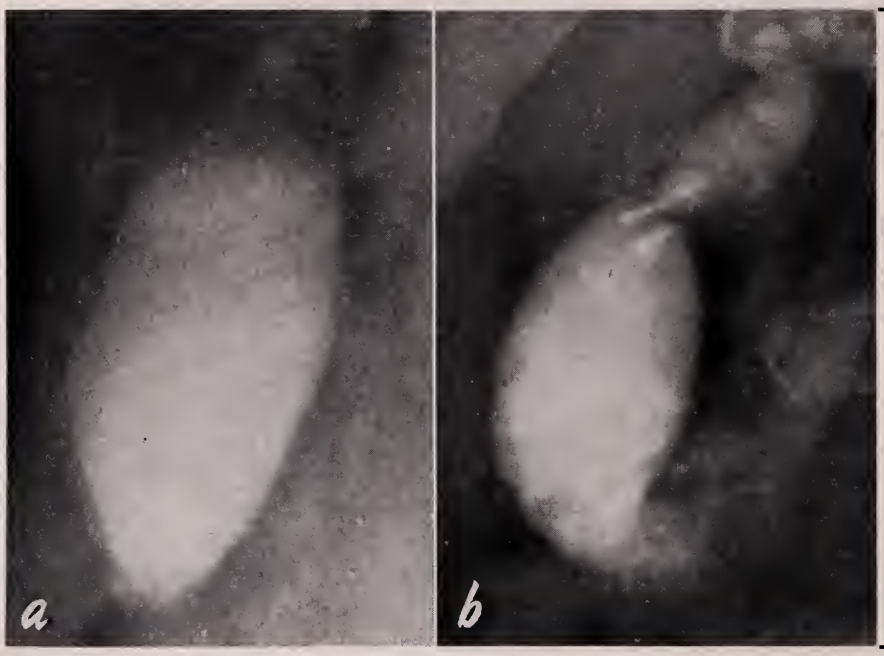

Fig. 3a. Well filled gallbladder containing multiple small calculi, with bead-like projections surrounding most of the entire gallbladder, is seen.

b. This post-fatty meal film demonstrates abnormal contracture of the gallbladder and a significant narrowing of the upper one-third of that structure.

tends to substantiate the formation of hypertrophy of the muscularis mucosae toward the lumen of the gallbladder. Disease may involve only a small portion of the gallbladder or be generalized in type. In this study, the diverticula appear to be distal to the area of the stenosis of hypertrophy. The increase in the intercystic pressure is most likely caused by neurogenic dysfunction of the extrahepatic biliary system in most of the cases, as suggested by Colquhoun.

Dr. Lemak and his coauthor, Dr. Keegan, practice radiology in Houston.

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Mammography

Report of 1,000 Examinations and Evaluation of Criteria

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RESEARCH & INVESTIGATION

ONE THOUSAND consecutive roentgenographic examinations of the breast, performed and interpreted at the University of Texas M. D. Anderson Hospital and Tumor Institute between July, 1962, and May, 1963, have been reviewed. The series was comprised of 514 patients.

Of these patients, 138 were admitted to the hospital with lesions subsequently proved by biopsy. Of these, 81 had malignant lesions and 57, benign lesions. Not all patients admitted to the hospital's breast service were referred for mammography. The difference in the incidence of malignant and benign lesions in Dr. Egan's series and in the present series indicates that patients with clinically obvious carcinomas were not referred for mammographic examination.

In this respect, this series more nearly approximated the experience expected in private practice. The patients were selected, and those referred for mammographic study were patients who presented a clinical problem of differentiation. Of these, 376 interpreted to have normal breasts or benign lesions were not biopsied.

Insufficient time has lapsed since the study of this group began to permit making completely reliable conclusions. Clinical agreement and the fact that none of this group has returned with malignant conditions suggest that the mammographic interpretations are correct.

In this series, there was no deviation from the routine methods and technical factors established by Dr. Egan and employed in his analysis of 2,000 cases.[2] The only significant variable is the interpreter. The high degree of accuracy of the mammographic examination invites skepticism. More series should be studied to determine the validity of mammography.

Tabulations of the results of 1,000 consecutive studies are grouped in Table 1. The six cases of carcinoma coded as negative or benign included two which previously were examined by Dr. Egan and reported as negative for carcinoma. Both of these patients had fibrocystic disease and adenosis. One previously was examined in 1957 and the other, in 1960. In neither case could a significant difference be detected, nor was there evidence of carcinoma.

Carcinoma is obscured easily by dense adenosis. Since adenosis is considered precancerous it is a recognized limitation of mammography. One patient had a small carcinoma in the upper outer quadrant not projected on the film study. The three remaining carcinomas coded as benign lesions included one small carcinoma adjacent to a recognized fibroadenoma; a large, dense, lobulated carcinoma with coarse calcification in the background of residual glandular tissue which was considered a large calcified fibroadenoma; and in the tail of the breast, a large tender mass with only slight blurred margination and no associated secondary change,

TABLE 1.—*Diagnosis of 1,000 Mammographic Studies as Reported and Coded, July, 1962, to May, 1963.*

Type Lesion	Total	Coded		
		Benign	Negative	Malignant
Biopsy				
Malignant	81	3	3	75
Benign	57	50	0	7
No Biopsy				
Benign	376	376		

Much credit for mammography belongs to Dr. Robert L. Egan, who adapted radiographic techniques and diagnostic criteria from a few earlier investigators and later statistically analyzed 2,000 studies to prove the value of mammography. Since Dr. Egan's appointment to the staff of Methodist Hospital in Indianapolis, Dr. Swearingen has assumed responsibility for the mammography program at the University of Texas M. D. Anderson Hospital and Tumor Institute.

which was interpreted as an abscess.

Benign conditions of the breast coded malignant included two cases with diffuse skin thickening indistinguishable from inflammatory carcinoma and two with regional skin thickening associated with an underlying abscess. Two of the remaining three benign conditions coded malignant were considered indeterminate, but for coding purposes the interpretation of malignant was favored. The other case demonstrated punctate calcification in a small nodule in a patient with carcinoma, primary lesion unknown.

Dr. Egan previously had examined 121 patients in this series. Thirteen of these had biopsy-proven carcinoma. The earlier mammograms were interpreted as negative for carcinoma.

Bilateral carcinoma was present in three patients at the initial examination. Contralateral carcinoma was present in 24 patients who had undergone mastectomy.

In this series 376 patients were not biopsied. Most were interpreted to have benign lesions, agreeing with the clinical impression. In none of these patients have subsequent biopsies proved to be malignant. Several mastectomized patients with widespread metastases and mammographic evidence of a second primary lesion or cross metastases were not biopsied, but received radiotherapy or other modes of therapy.

Evaluation of Criteria

A dominant nodule visualized in the breast in a background of fat is easily identified as benign or malignant. The benign lesion is round, oval, or lobulated in shape and has smooth, well-circumscribed borders. It is homogeneous in density. Often, a fine rim of lucency representing fat displacement outlines the nodule. Occasionally, coarse calcification is seen within a benign lesion, indicating a calcified fibroadenoma. Clinically, the mass measures the same size by palpation as it appears on the film study (Fig. 1).

The malignant lesion shows variable, ragged, and spicular shape and margination. The lesion appears heterogeneous because of infiltration and fibrotic response. The malignant lesion also appears more dense at the center than in its peripheral portions. Occasionally, numerous punctate calciferous densities are seen within the lesions which are characteristic of carcinoma (Fig. 2).

A lesion that is a clinically palpable mass frequently is obscured completely because of fibrocystic disease and adenosis. When a dominant nodule is visualized clearly in a predominantly fat background, the shape of the lesion, margins, size, and infiltrative and fibrotic characteristics can be evaluated adequately, and diagnostic accuracy approaching 100 percent can be rendered (Fig. 3).

When the clinically palpable nodule is obscured, calcification may be a clue in determining the nature of the lesion. Calcification typical of carcinoma and of benign fibroadenoma has proved a highly accurate criterion when applied according to strict definition. The calcification of carcinoma is small, punctate, and like grains of sand too numerous to count. Calcification must be visualized from both views not only to determine its position within the breast but also to show that the calcification actually is confined to a small area and is not the calcification seen in arteriosclerosis or the scattered benign ductal calcification. Other authors have stated that calcification of carcinoma occurs in approximately one-third of

Fig. 1. Cranio-caudad view of both breasts. Typical calcified fibroadenoma located in the outer portion of the right breast.

the cases. In this series of cases, the incidence of calcification of carcinoma approximates 10 percent and consideration of it was seldom needed to arrive at the diagnosis of carcinoma; other criteria were present which eliminated the importance of identifying the calcification (Fig. 4).

Calcification of carcinoma has helped identify lesions not visualized in a background of fibrocystic disease and adenosis when there are no other secondary characteristics of carcinoma.

Coarse calcification occurs occasionally in carcinoma. This calcification has none of the criteria generally considered indicative of malignant lesions and must not be confused with benign calcification (Fig. 5).

Skin thickening has been a useful criterion in the diagnosis of carcinoma. The acute or inflammatory carcinoma may not appear as a dominant mass within the breast. Diffuse skin thickening, predominant in the medial quadrant, with diffuse increased density in that breast compared with the opposite breast, and stranding attributable to engorged and blocked lymphatics, are sufficient to establish the diagnosis of acute carcinoma. When skin thickening caused by carcinoma is regional or localized, there must be a dominant nodule beneath the involved skin. Also fibrotic response and evidence of lymphatic engorgement generally are present, as well as retraction. Regional skin thickening without retraction most often represents a

Fig. 2. Medio-lateral view of both breasts. Scirrhous carcinoma in the lower portion of the left breast. Note the distant extension to the inferior areola with retraction and skin thickening.

Fig. 3. Medio-lateral view of both breasts. Scirrhous carcinoma in the upper portion of the left breast.

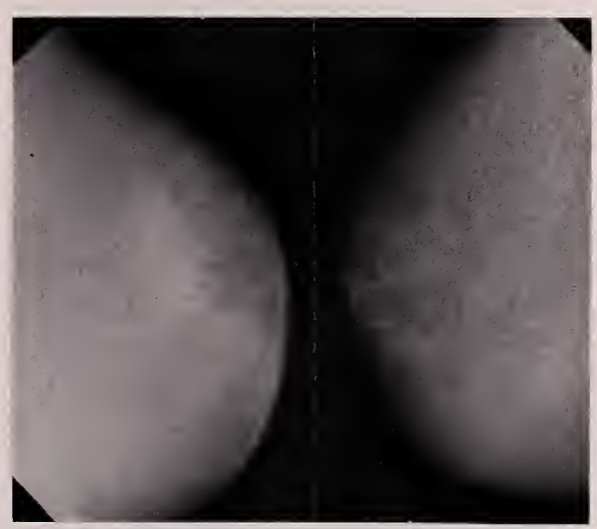

Fig. 4. Medio-lateral view of both breasts. Predominantly scirrhous carcinoma is in the left breast. Note diffuse increased density due to fibrosis, skin thickening, and calcification of carcinoma limited to the mass.

Fig. 5. Carcinoma of the right breast with coarse calcification within it. This condition must not be interpreted as benign calcification. The lesion appears loculated with little evidence of infiltration. Venous engorgement is of help in the diagnosis of carcinoma. If the coarse calcification should be considered as the benign type, this lesion may be interpreted as a benign fibroadenoma.

benign inflammatory process (Figs. 6, 7).

Venous engorgement in the region of an identified dominant nodule is a secondary sign helpful in establishing the diagnosis of carcinoma. The breasts generally are symmetrical. Increase in the size of the veins in only one breast may indicate an underlying carcinoma. The venous engorgement is proximate to the dominant nodule. Most often, the lesions which show very little infiltration, fibrotic response, or other secondary signs of a malignant condition will show increase in vascularity, the only additional change present to confirm the true identity of the visualized, dominant nodule. In the absence of other changes, the apparent increase in vascularity of one breast as compared with the other is disregarded as a variable and of no significance (Figs. 8 and 9).

Fibrotic response may be out of proportion to the primary lesion to such an extent that the underlying carcinomatous lesion is difficult to identify. This sign has been particularly helpful in evaluating lesions in the region of the tail of the breast. The value of comparing one breast with the other cannot be overemphasized. Minimal changes of increased density may be the first recognized finding of an underlying small carcinoma, particularly if the increased density is within the background of residual glandular tissue. Fibrosis appears as increased density of one region of the breast as compared with its counterpart. Also, in comparison with the opposite breast, there appears to be a loss of volume of the affected area of fibrosis (Fig. 10).

Axillary lymphadenopathy visualized on the mammogram occurs in a high percentage of patients with carcinoma. This secondary sign of carcinoma, however, has not been of great value in arriving at the correct diagnosis. Generally, carcinoma of the breast is recognized and the presence of axillary adenopathy merely is noted. Occasionally when primary and secondary changes in the

Fig. 6. Skin thickening of acute or inflammatory carcinoma. Note the diffuse increased density and absence of a visualized dominant nodule.

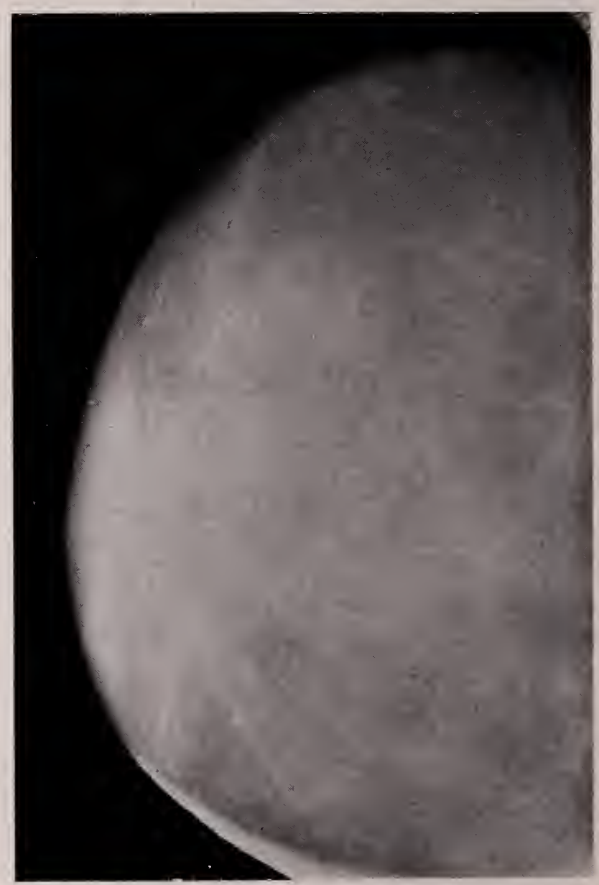

Fig. 7. Benign inflammatory process. This is infrequent, but stresses the need for biopsy prior to therapy.

breast are equivocal, the presence of significant adenopathy warrants the presumptive diagnosis of carcinoma. Both axillae are examined in order to determine that the visualized adenopathy is unilateral rather than bilateral. If bilateral, its significance would be minimized. Significant nodes generally measure over 1 cm. in greatest diameter.

Nipple retraction is considered the secondary sign of least value in evaluating the possible presence of carcinoma. When nipple retraction due to carcinoma is present, there is evidence of the mass of carcinoma and, generally, regional or diffuse skin thickening. Nipple retraction, when visualized without evidence of a dominant nodule or additional change to suggest carcinoma, cannot be considered a sign of carcinoma, and is generally present bilaterally. The appearance of the nipple should not be neglected, however, since Paget's disease should produce asymmetrical enlargement and, generally, underlying extension.

Occasionally, a mammographic examination is considered indeterminant because a dominant nodule or additional carcinomatoid change cannot be demonstrated, although a clinically palpable mass is present. Generally, when present, the absence of secondary characteristics favors the diagnosis of a benign lesion. Other indeterminate lesions may show conflicting secondary changes; in that case, the secondary sign of most importance is favored in rendering the report of benign or malignant disease. Correlating the palpable size of the lesion when it is not clear-

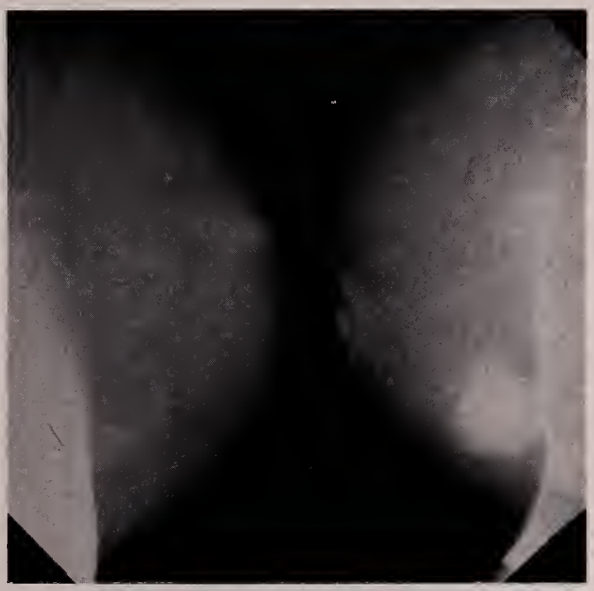

Fig. 8. Anaplastic carcinoma with venous engorgement.

Fig. 9. Malignant cystosarcoma phylloides. The lesion shows the minimal infiltration typical of sarcoma. The venous engorgement establishes the diagnosis of a malignant condition.

Fig. 10. Fibrotic response in the upper portion of the right breast. Note loss in volume, skin thickening, and increased density, as compared with the left breast.

ly visualized on the mammogram may be of help, since a large lesion which is not visualized would certainly arouse the suspicion of underlying carcinoma. In these cases, if a biopsy is not performed, a follow-up examination after an interval of time is requested. If the mammogram shows a dominant nodule with characteristics insufficient to establish a diagnosis of carcinoma, the case may correctly be diagnosed as malignant if the palpable mass measures larger than the mass visualized on the film study. The possibility of a benign lesion being situated in close proximity to a smaller malignant lesion should not be overlooked.

Discussion

Mammography has created renewed interest in the study of the natural history of carcinoma of the breast. Lymphangiography, angiography, and histopathologic correlation with mammographic information may alter and affect the patient's course in the future. Radiotherapeutic and chemotherapeutic response in the unmastectomized patient is evaluated. Preliminary information suggests that the venous engorgement associated with malignant tumors is caused by the arteriovenous shunting of malignant vasculature similar to grade 4 astrocytomas and renal cell tumors. Fibrosis probably represents a defensive response of the surrounding tissue to limit the spread of carcinoma. Direct lymphatic venous communication may occur in the breast. Can the route of metastases and grade of malignancy of a tumor be predicted?

Several types of carcinoma can be distinguished by the radiographic appearance. The scirrhous carcinoma produces the spicular and tentacled margins, is irregular in shape, and poorly circumscribed. The medullary and mucin-producing carcinomas tend to show only slight blurring of the margins; there is little evidence of infiltration or fibrotic response. Often an additional indication of the malignant character of these lesions is the evidence of adjacent venous engorgement. The acute or inflammatory carcinoma characteristically shows diffuse thickening of the skin, more apparent in the medial quadrant. Diffuse, increased density is also noted throughout the entire affected breast, as compared with the opposite breast.

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Stranding, probably indicating lymphatic engorgement, is observed in the acute carcinomatous breast.

Comedocarcinoma most often shows small, punctate, calciferous densities too numerous to count. The calcification of ductal carcinoma may be diffuse without evidence of an associated mass. When a mass is visualized the calcification may occur outside the apparent boundary of the lesion. Calcification occurring in scirrhous carcinoma is almost always within a well-defined mass.

The malignant cystosarcoma phylloides, if adequately visualized in a background of fat, will show only slight blurring of the margins. The mass appears lobulated, and evidence of adjacent venous engorgement is noted. Most often, the mass is dense and homogenous. Other varieties of sarcoma present a similar appearance. Less malignant varieties of cystosarcoma phylloides may indicate their malignant character only by evidence of fixation to the skin.

The significance of the secondary characteristics of carcinoma is now being evaluated. An anaplastic carcinoma reviewed has shown increase in vascularity but no other secondary changes (Fig. 8). Fibrotic response is probably a defense mechanism of the surrounding tissue to limit carcinoma penetration and seems to occur in slower growing neoplasms. Although calcification seems to occur predominantly in the comedocarcinomas, its presence in other varieties of carcinoma would seem to indicate that a tumor has been present for a considerable period of time, probably undergoing partial necrosis and subsequent calcification.

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Choice of Therapy for Mammary Cancer

CHARLES ECKERT, M.D.

No method of treatment for breast cancer in use today is really effective for the majority of cases. In this article, basic theories are reviewed and a protocol in selecting treatment for individual patients is given.

REVIEW ARTICLES

FOR NEARLY 50 years, radical mastectomy as independently described by Halsted and Meyer was the accepted method of treating mammary cancer. The operation was regarded so highly by the surgical leaders during this period that it served as the model for all cancer surgery. A few dissidents continued to use simple mastectomy, either routinely or in selected cases, but this procedure was condemned by most surgeons. Preoperative and particularly postoperative radiation therapy was used, but principal evidence of their value was subjective. Preoperative irradiation never gained general acceptance, but many surgeons used it and still continue to use postoperative irradiation, particularly when the axillary lymph nodes are found to be involved.

However, with the passage of time, the mortality rate from breast cancer did not decline despite more widespread use of the procedure by increased numbers of well-trained surgeons. A disturbing proportion of patients who were not candidates for the radical operation because of either locally advanced or disseminated disease were untreated. Further, the survival rate credited to radical mastectomy in the group with axillary lymph node metastases, for which the operation was designed, was disappointingly small. Thus, there was a need to find other methods of treatment.

Four general approaches have been used: first, extension of radical mastectomy to include removal of either supraclavicular lymph nodes and internal mammary lymph nodes or internal mammary lymph nodes alone; second, a return to simple mastectomy; third, reevaluation of the use of radiation therapy combined with either simple or radical mastectomy; and fourth, an attempt to define more precisely operability by conventional radical mastectomy. Unfortunately, end results of these different treatments have not shown decisive differences.

It is the purpose of this article to review the present status of the main alternative methods of treating mammary cancer to provide a rational approach to the individual patient.

Introduction

Curative therapy for breast cancer—whether it be conservative, radical, or superradical surgery, radiation therapy, or radiation therapy plus various forms of surgery—is based on the theory that there is a time in the evolution of this neoplasm in which the disease, while recognizable clinically, remains localized to the breast, or to the breast and axillary lymph nodes. Experience with patients with this disease illustrates a wide spectrum of behavior that at times is in conflict with this theory and at other times is congruent with it. For example, patients are seen who have widely distributed metastases without a palpable mass in the breast who subsequently are proven to have primary mammary cancer. In other instances, the first evidence of disease may be an enlarged axillary lymph node due to metastatic growth from a breast cancer not clinically evident. At the other end of the spectrum is the patient with a large primary tumor of long duration with no evidence of spread, either at the time of treatment or during prolonged follow-up. In none of these groups is the

time, quality, or type of therapy critical in determining the outcome. However, such cases do not rule out the possibility that in a significant part of the behavior spectrum of breast cancer, the relationship between time, growth, and metastasis is critical in determining the end result of ablative or destructive therapy. To say this is not to prove it; indeed, proof of this essential behavior pattern is most difficult to obtain. Here, once again turning to individual case experience, one sees patients who at the time of radical mastectomy have extensive spread to axillary lymph nodes but who apparently are cured by radical mastectomy. While suggestive, this type of experience does not prove that further delay by days, weeks, or months would have altered the outcome. Neither does it tell us whether removal of the primary tumor will at times influence metastases to axillary nodes, or how great is the potential of further dissemination from axillary lymph node deposits. Late recurrence or evidence of distant metastasis at long intervals after a seemingly successful operation further clouds the issue.

Data on survival from untreated breast cancer also indicate the range in lethality between the rapidly progressive and relatively benign neoplasms. Of interest is the similarity of survival curves of untreated breast cancer cases in series of disparate size reported from widely separated geographic areas.[7, 8]

Park and Lees[19] using data from the literature, as well as data of their own, studied the question of the relationship between delay in treatment and curability. They were unable to demonstrate a positive correlation between these factors. In the same study evidence was presented to show that the growth rate of any given breast cancer is relatively constant and that the likelihood of metastatic "takes" is also constant throughout the period of growth. From this study they conclude, "It has not been proved that the survival rate of cancer of the breast, using the 5-year survival rate as an index, is affected by treatment at all."

Collins' and others' data is in agreement with Park and Lees that the growth rate of an individual tumor is constant, and that for a given lethal tumor, the period between its clinical recognition and the death of the patient is a relatively brief interlude in the total natural history of the tumor.[5]

The studies relating duration of disease to the end results of treatment are probably of little value because of the inaccuracies in the basic data. The patient's lack of acuity in observation, fallacious memory, and unwillingness to confess the true full duration of the disease grossly distort the material. The correlation between tumor size, frequency of axillary lymph node metastases, and curability should be a more accurate measure of the influence of delay on prognosis. MacDonald[17] found this correlation to be relatively poor except in the very small and the very large lesions. Fifty-six percent of lesions 1 cm. or less in diameter had metastasized, while 23 percent of lesions over 5 cm. in size had not metastasized. The term "biological predeterminism" was used to describe the biological factors inherent in each tumor from its onset which play such an important role in eventual outcome. Other reports, however, show the relation between tumor size and frequency of metastases to be closer than that found by MacDonald.

Using the biological classification of mammary cancer described by Hultborn and Tornberg (Table 1),[14] Butcher[3] applied the method of probit analysis to the different biological types. As a control he also analyzed by the same technique the untyped data of Greenwood[8] on untreated breast cancer. He found that types 1 and 2 and type 3 without axillary lymph node metastases, which together comprised 37 percent of the total, affected life expectancy only slightly (92 percent age corrected survival). The type 3 cases with axillary metastases and type 4 cases with and without axillary metastases were highly lethal cancers. When the fitted probit transformations of these lethal cancers were plotted on the same graph with the probit transformations of Greenwood's

TABLE 1.—*Biological Classification of Mammary Cancer (Tornberg).*

1. Nonmetastasizing (not invasive)
Intraductal carcinoma without stromal invasion
2. Rarely metastasizing (always invasive)
a. Purely extracellular mucinous cancer
b. Medullary cancer with lymphocytic infiltration
c. Well differentiated adenocarcinoma
3. Moderately metastasizing (always invasive)
a. Adenocarcinoma
b. Intraductal carcinoma with stromal invasion
4. Highly metastasizing (always invasive)
a. Undifferentiated carcinoma
b. All types of tumors with vascular invasion

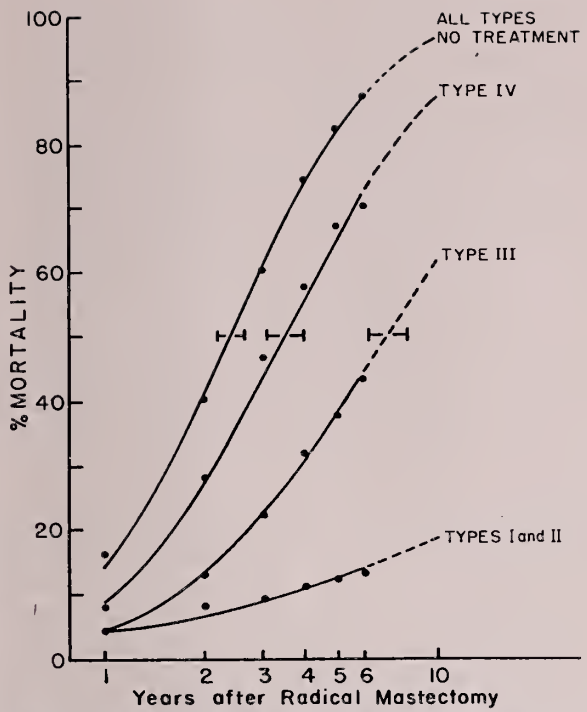

Fig. 1. Comparison of the mortality expectancy of the different types of mammary cancer with the untreated series of Greenwood (After Butcher[3]).

survival data a significant difference in survival was evident despite the inclusion in the Greenwood data of an undetermined number of the favorable biological types (Fig. 1). This work is important because it validates a method of histologic grading which defines the extremes of biological behavior. The study also demonstrates significant improvement in survival of unfavorable cases treated by radical mastectomy as compared with untreated cases. It is also of interest that on the basis of this analysis Butcher concludes that "the proportion of women with mammary cancer in a consecutive series who might survive as well after simple mastectomy as after radical mastectomy is rather high (75%)."[3]

Throughout the vast literature pertaining to mammary cancer is found the repeated warning against comparisons between series from different centers over varying periods of time. Because prognosis has been related to age, location of the primary tumor within the breast, clinical and pathologic stage of the disease, menstrual status, biological factors inherent in the tumor (possibly defined), and biological factors inherent in the host (totally undefined), all of the known factors, at least, must be reasonably similar before valid comparisons between different series can be made.

In summary, mammary cancer exhibits a wide spectrum of behavior patterns ranging from the highly malignant, rapidly progressive, and almost inevitably fatal, to the relatively benign, dilatory, and nonlethal. Likely there is a "gray" zone in which the correlation between time of onset and time of treatment is close, and where the character as well as the time of treatment may influence outcome, but certain proof of this behavior pattern is difficult to obtain. Butcher's study is the best evidence available, and it also defines the different behavior patterns in terms of reproducible histopathology. The fallacies in comparisons of data from different institutions and from different time intervals are discussed. Those factors believed to influence prognosis which must be similar in order to make valid comparisons are enumerated.

With this background, we will now proceed to a discussion of individual forms of treatment.

Radical Mastectomy

The theoretical basis for the use of this operation is the principle of wide removal of the primary tumor, in continuity with the lymphatics and first zone of lymph nodes to which it spreads. Since clinical examination of the axilla is inaccurate (Table 2), all patients with histologically proved primary breast cancer are treated by the complete operation. Proof to the contrary being lacking, metastases to axillary lymph nodes are assumed to be the potential source of further lymphatic and hematogenous metastases.

In the presence of certain signs of locally advanced disease, best enumerated by Haagensen and Stout,[11] radical mastectomy is contraindicated because it will not cure. Whether life expectancy is adversely affected by transgression against these criteria of operability has not been proved, but when the disease is locally advanced, radical mastectomy will usually not effect local control. If this be so, radical mastectomy is contraindicated. These local signs of advanced disease form the basis of Haagensen's clinical

TABLE 2.—*Error Percentage in Clinical Evaluation of Status of Axillary Lymph Nodes.*

Clinical Opinion	Haagensen	Butcher
Negative	32%	32.2%
Positive	24%	24.5%

classification[10] (Table 3) which is a precise method of clinically classifying breast cancer.

The objections to radical mastectomy raised by proponents of different forms of therapy are as follows. It fails to accomplish that which it was designed for, namely, to control cancer metastatic to axillary lymph nodes. When axillary lymph nodes are involved, the radical operation rather than controlling cancer disseminates it. When axillary lymph node metastases are present, frequent metastases to zones which are not removed (supraclavicular, internal mammary) limit the number of patients who can be potentially cured. The operation is applicable only to selected cases.

The first objection has been expressed by several, but we shall consider the statements made by McWhirter[18] since they summarized the reasons for a trial of an alternative method of treatment. He stated that the average failure rate of radical mastectomy was 65 percent within five years, and that by ten years, few in this group would be alive. To contrast with this figure, Lewison[16] reporting on the five-year period 1946-1950, observed a 41.3 percent five-year survival rate, and a 24.6 percent ten-year survival rate in patients with axillary lymph node metastases. Butcher's previously quoted study further substantiates this accomplishment of radical mastectomy.

Of great importance is the fact that recurrence in the axilla is seldom seen following radical mastectomy, providing patients are properly selected and the operation is properly done. When axillary lymph nodes are fixed to the chest wall, or to the neurovascular bundle, operation is contraindicated because under these circumstances control is then impossible and dissemination is likely.

Although it is accurate to state that radical mastectomy cannot be used for all patients with primary untreated breast cancer, it equally is accurate to state that no method of therapy can be applied to all primary untreated cases in any series. McWhirter can use simple mastectomy and irradiation therapy for patients inoperable because of locally advanced disease as well as for those who would be operable, but patients with disseminated disease equally are untreatable by any form of curative therapy. Because one method of treatment can be applied to a larger portion of a group does not necessarily indicate superiority for that method over the method applicable only to the smaller portion. Superiority will still depend upon a comparison within the group to which both methods are applicable.

TABLE 3.—*Haagensen's Clinical Classificaton of Mammary Cancer.*

Stage A	Tumor confined to the breast. No "grave" signs.
Stage B	Palpable axillary lymph nodes thought to contain cancer, not fixed or over 2.5 cm. in diameter. No "grave" signs.
Stage C	One "grave" sign present consisting of edema of limited extent, axillary node over 2.5 cm. in diameter, fixation of axillary nodes to skin or chest wall, ulceration (except Paget's disease) solid fixation to chest wall.
Stage D	Patients having any two of the "grave" local signs, distant metastases, satellite skin nodules, extensive edema, edema of the arm, or "inflammatory" carcinoma.

The final objection is based upon the long-recognized propensity for breast cancer metastatic to axillary lymph nodes to involve the supraclavicular and internal mammary lymph nodes. Halsted recognized this, in fact in one series he extended his operation to include division of the clavicle and continuity dissection of the supraclavicular chain.[12] He abandoned this extension of radical mastectomy, since morbidity was increased without improvement in control of disease. Andreassen and others[1] more recently determined the frequency of supraclavicular lymph node involvement to be 33 percent in those patients having axillary lymph node involvement.

Internal mammary lymph node metastases were recognized by both Halsted and W. Sampson Handley to be potential sites of metastases, but it remained for R. S. Handley[13] to show that in 34 percent of operable cases these nodes indeed harbored metastases. Most of these metastases occurred in the presence of axillary spread, but in a small percentage of inner hemisphere and central lesions, metastases to internal mammary lymph nodes occur in the absence of axillary lymph node metastases.

These additional lymph node zones of spread are possible explanations for certain of the failures of radical mastectomy, but whether the disease is still sufficiently limited in the face of metastases to these regional nodes to justify extended radical mastectomy or radiation therapy as an adjunct

to, or substitute for, surgery will be discussed subsequently.

The morbidity of radical mastectomy has been alluded to as a justification for its abandonment. Emotional disturbances are frequent in women following mastectomy, but are not reduced by the saving of the pectoral muscles effected by simple mastectomy. Seldom are emotional disturbances sufficiently great to be uncontrollable by sympathetic support of the surgeon and the use of a suitable prosthesis to restore contour. Decreased function of the arm on the affected side is usually minimal if the incision is properly placed and early physiotherapy is instituted. Surgical lymphedema is troublesome when cancer persists locally, but such edema is a relatively infrequent complication.

Extended Radical Mastectomy

The aforementioned studies of the frequency and significance of supraclavicular and internal mammary lymph node metastases spurred a number of efforts to improve the results of surgical treatment by the concomitant or secondary removal of supraclavicular and internal mammary lymph nodes. A sufficient period has now elapsed to evaluate some of these efforts.

In general, confirming the earlier work of Halsted, attempts to eradicate metastases to supraclavicular lymph nodes have been unrewarding, whether the operation be done by exceedingly radical technique (Wangensteen),[25] or by the more conservative method of Andreassen and others.[1] It therefore appears that proved supraclavicular spread should continue to be an absolute contraindication to surgical treatment of breast cancer.

On the contrary, a number of reports are now available which indicate that surgical removal of involved internal mammary lymph nodes may increase survival. The data of Urban,[26] Dahl-Iversen and Tobiassen,[6] and Sugarbaker,[24] while early, incomplete, and not well controlled, still suggest that this may be so. The operations used by Urban and by Sugarbaker are essentially the same; they include en bloc removal of a segment of the chest wall with the underlying internal mammary vessels and lymph nodes attached. The mortality and morbidity incident to this extension has not been great. The operation done by Dahl-Iversen and associates is considerably less extensive consisting usually of division of costal cartilages to allow removal of the underlying vessels, lymph nodes, and areolar tissue as a dissection separate from the mastectomy. Here also the mortality and morbidity has not been excessive, and the five-year results in patients with involved nodes are promising (Table 4). They conclude, "The prognostic importance of internal mammary invasion would appear to be of the same order as the importance of metastases to other regional nodes."[6]

TABLE 4.—*Survival with Internal Mammary Metastases.*[6]

	Number of Patients	Number Surviving	Percent Survival
Axilla (+) Internal mammary (+)	59	30	51
Internal mammary only	11	7	64
Total series	366	255	70

Increased Patient Selectivity For Radical Mastectomy

The energies of Haagensen and co-workers[10] at the Presbyterian and Delafield Hospitals in New York have been directed toward improving the results of radical mastectomy by increased selectivity of patients for the operation. The contributions of this group in delineating specific signs of advanced disease in which radical mastectomy always fails was a most important one. More recently the technique of "triple biopsy" has been employed by them further to define operability. Believing that extension to supraclavicular and internal mammary lymph nodes is an indication of surgical incurability, a preliminary biopsy of the tumor is done, at the same time the highest axillary lymph nodes and internal mammary lymph nodes from the first, second, and third intercostal spaces are biopsied. When spread to these lymph nodes is found, the patients are treated with intensive radiation therapy (Guttmann).[9]

The triple biopsy requires a separate operation, and if carried out routinely involves an unnecessary additional hazard to the patients whose lymph nodes are found to be

negative. Others use this procedure in patients with evidence of locally advanced disease who are of borderline operability.

The significance of metastases to the highest axillary nodes as an indication of inoperability has been challenged by several observers who find considerable salvage in patients with highest axillary lymph node involvement.

Simple Mastectomy And X-Ray Therapy

The previously alluded to Edinburgh experiment initiated in 1941 by McWhirter as a result of disenchantment with the accomplishments of radical mastectomy has great merit. All cases, without selection, were treated by simple mastectomy and carefully calculated homogeneous dosage of heavily filtered radiation therapy. Treatment included both internal mammary and supraclavicular lymph node regions missed by radical mastectomy alone, as well as the chest wall and axilla. The series is large, well followed and well documented. The five- and ten-year survival rates of the "operable" group of 58 and 39 percent respectively, compare favorably with results obtained by other methods of treatment.

Kaae and Johansen[15] using McWhirter's method of treatment report a 64 percent five-year survival in a limited series.

One factor not brought out by these statistics is the condition of many of the patients alive at the end of the five- and ten-year intervals. Bruce[2] observed many of the patients surviving for five or more years to have necrotic, ulcerating tumors in the treated areas.

In a most interesting study Watson[27] compared McWhirter's survival data with the survival of patients in Saskatchewan treated by radical mastectomy and postoperative radiation therapy. All primary cases were included in both studies and both studies included almost all cases of mammary cancer occurring in a geographical unit. The Saskatchewan series showed almost a 10 percent greater five-year survival than that obtained by McWhirter. Based upon this evidence as well as upon the aforementioned observation of persistent disease Bruce[2] states that radical mastectomy will once again be used in Edinburgh for properly selected patients.

McWhirter's contribution is not to be denigrated, however, for without question it contributed greatly to our knowledge of the influence of radiation therapy on mammary cancer. Furthermore, his method remains the treatment of choice for patients inoperable because of locally advanced disease. Using orthovoltage technique 30 and 15 percent, five- and ten-year survival rates were obtained. Guttmann,[9] using supervoltage therapy, treated 48 of Haagensen's patients inoperable because of metastases to the internal mammary lymph nodes and the highest axillary lymph nodes or both. Fifty-four percent of these patients survived for five years. This figure may be compared with Haagensen's operable cases with proven axillary lymph node metastases in which the five-year survival rate was 53 percent.

Radical Mastectomy And Radiation Therapy

Two controlled studies of the addition of postoperative radiation therapy to radical mastectomy have been reported, one from Manchester by Paterson and Russell,[20] the second from St. Louis by Butcher, and others.[4] Paterson and Russell investigated two different treatment methods; the first, called the quadrate series, consisted of treatment directed primarily to the chest wall and axilla; the second, or peripheral series, consisted of treatment directed to the supraclavicular and internal mammary lymph nodes. The technique in the St. Louis study was similar to the peripheral series of Paterson. In both of these reports survival of treated and untreated patients was almost identical. In both studies an orthovoltage source was used; the possibility therefore remains that as suggested by Guttmann's results, a similar study using supervoltage therapy may show an additional salvage by the addition of postoperative radiation therapy.

The best study of preoperative radiation therapy was that of Richards of Toronto.[21] He obtained the greatest gain from intensive radiation in patients with locally advanced disease (stages 4 and 5 by his method of classification) in whom radical mastectomy was later performed. This method of treatment had been previously tried at the Memorial Hospital without appreciable benefit.

Dr. Charles Eckert, professor and chairman, Department of Surgery, Albany Medical College of Union University, Albany, N. Y., was a 1964 Annual Session speaker.

Unfortunately Richards' death terminated his study after a relatively brief trial. New interest in this approach to therapy may occur.

Simple Mastectomy

A number of qualified surgeons have long believed simple mastectomy to be just as effective an operation for mammary cancer as radical mastectomy. Among the most widely publicized and controversial studies was that of Smith and Meyer of Rockford, Ill.[23] They reviewed the results of treatment of breast cancer in that community from two different time intervals; in the first simple mastectomy was used, in the second, radical mastectomy. The survival was essentially the same in the two groups. Their material was reviewed by a team from the National Cancer Institute[22] who found the two series to be comparable and the results previously reported to be correct. This study again prompted the demand that this issue be resolved by a rigidly controlled study of simple versus radical mastectomy, the cases selected by randomization.

Discussion

Lewison[16] has emphasized the basic similarity between the results of treatment of breast cancer by these divergent methods, particularly as seen in ten-year survival data. It is true that many apparent differences in results of treatment at the five-year level disappear in the succeeding five years. Lewison also espouses the need for a randomly selected, rigidly controlled clinical trial in an effort to resolve the differences in opinion regarding the merits of simple and radical mastectomy. However, since it seems that none of these methods of treatment are effective in treating highly malignant breast cancer that has metastasized widely by the time diagnosis is possible, and since it seems that many breast cancers will do as well with conservative as with radical surgery, such a study, I believe, can add information only for the small portion of the total sample which remains. Energy directed toward this type of study will, in my opinion, be better expended in finding an effective treatment for disseminated mammary cancer.

At present, I believe there is sufficient information available to allow one to select treatment to fit the individual case. A suggested protocol for selection is as follows:

1. For Haagensen clinical stage A, lesion in the outer hemisphere of the breast, the standard radical mastectomy is applicable.
2. For Haagensen clinical stage A, lesion in the inner hemisphere or central portion of the breast, extended radical mastectomy as described by Urban is recommended.
3. For all Haagensen clinical stage B, radical mastectomy is applicable possibly with postoperative radiation therapy using supervoltage technique.
4. For Haagensen clinical stage C, triple biopsy is indicated. If positive nodes are found supervoltage x-ray therapy may be used with or without simple mastectomy, depending upon the size of the breast. If lymph node biopsies are negative, radical mastectomy is recommended as in clinical stage B.

Summary

The theoretical basis for the various methods of treating breast cancer is reviewed. No method of treatment in present use is found to be really effective in the majority of breast cancers. A protocol for selective treatment based on clinical staging, the location of the primary tumor in the breast, and in patients with locally advanced disease by biopsy of the highest axillary and internal mammary lymph nodes is described.

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Extreme Hyperuricemia Associated with Prednisone Therapy For Acute Lymphoblastic Leukemia

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CASE REPORTS

HYPERURICEMIA and hyperuricaciduria occur frequently in leukemia and lymphoma.[2-8, 11] Lynch found that some degree of hyperuricemia occurred before therapy in 124 (67 percent) of 186 cases of reticuloendothelial malignancy. In each of ten disease categories analyzed, the incidence of hyperuricemia was 50 percent or greater.[6] Other cases have been described in which serum uric acid concentrations were normal prior to treatment, but climbed to extreme heights following therapy.[2, 5, 6, 8, 11]

Hyperuricaciduria may occur in the presence of normal uric acid concentrations or may accompany only minimal hyperuricemia.[3, 4, 7, 8, 10] When hyperuricemia appears, uric acid crystals may be deposited in the renal tubules, and uric acid stones may lodge in the ureter. When these are excessive, either may lead to oliguria or anuria.[2, 5, 6, 8, 11] Lynch reported 25 patients who developed uremia caused by uric acid nephropathy secondary to malignant diseases of the reticuloendothelial system. These included 22 cases from the medical literature and 3 cases examined at Strong Memorial Hospital.[6] Other instances of this phenomenon have been described by Firmat and associates[2] and Weintraub and associates.[11]

The following case demonstrates the problems caused by hyperuricemia in the treatment of leukemia and exemplifies the extreme degree of hyperuricemia which may occur.

Case Report

First Admission.—A 16-year-old white boy noted small, nontender cervical masses which rapidly enlarged. He was seen at another hospital on July 5, 1963; cervical lymph node biopsy was performed and lymphoblastic lymphosarcoma was diagnosed. The patient was given 25 mg. (0.4 mg. per kg. of body weight) of nitrogen mustard intravenously in two equal doses on July 6 and 7. The lymph nodes became smaller.

The boy was admitted to the University of Texas Medical Branch Hospital on July 9. His chief complaint was dyspnea; he was somewhat disoriented and afebrile. There was a large pleural effusion on the right side. Slightly enlarged nontender lymph nodes were palpated in the cervical, axillary, and inguinal regions. The liver was not palpable. The tip of the spleen could be felt.

Dyspnea was substantially relieved and the pulse rate lowered following the removal of 425 ml. of serosanguineous pleural fluid. Cytologic examination showed mesothelial cells and small mature lymphocytes. A roentgenogram of the abdomen suggested that both kidneys were enlarged. The BUN was 174 mg. per 100 ml., and the serum uric acid 9.2 mg. per 100 ml. Blood count and liver function studies were normal. Fig. 1 depicts changes in BUN, serum uric acid, and white cell count throughout the illness. Urinalysis showed one-plus proteinuria, and many amorphous granules in the sediment.

The patient was instructed to drink large quantities of fluids, and urinary volume was maintained above 2,500 ml. per 24 hours throughout the hospitalization period. Two weeks later the patient was discharged. The lymph nodes were smaller, the liver was not palpated, and the tip of the spleen was barely palpable. Blood count and urinalysis were normal. The BUN was 56 mg. per 100 ml., and serum uric acid 7.3 mg. per 100 ml.

Second Admission.—One week later, the patient was readmitted because dyspnea recurred. A large collar of nontender lymph nodes encircled the neck. Large nodes also were observed in the axillary and inguinal areas. The liver extended 6 cm. below the right costal margin, and the spleen extended 4 cm. below the left costal margin. A chest roentgenogram showed a large pleural effusion on the right side and a widened mediastinum.

Massive hyperuricemia and hyperuricaciduria may occur in acute leukemia. Possible causative mechanisms, potential danger of hyperuricemia and hyperuricaciduria, and management of the syndrome are reviewed.

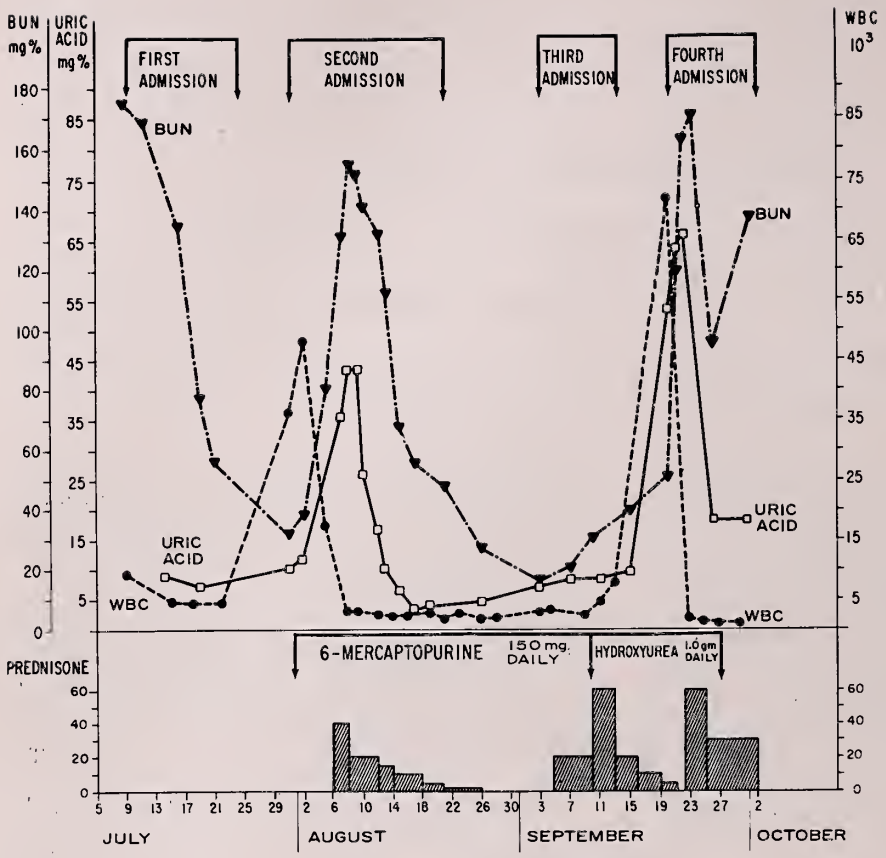

Fig. 1. Response of leukocytes, serum uric acid, and blood urea nitrogen to antileukemic therapy is shown. Dosage of prednisone is expressed as milligrams per day.

Laboratory data revealed BUN, 32 mg. per 100 ml.; serum uric acid, 10.5 mg. per 100 ml.; urinalysis, normal; hemoglobin, 12.5 gm. per 100 ml.; platelets, 174,000 per cu. mm.; and leukocytes, 36,200 per cu. mm. The leukocyte differential count showed 28 percent polymorphonuclear leukocytes, 15 percent stabs, 1 percent metamyelocytes, 2 percent myelocytes, 1 percent basophils, 2 percent eosinophils, 33 per cent lymphocytes, 4 percent monocytes, and 14 percent lymphoblasts. A bone marrow film presented an appearance typical of acute lymphoblastic leukemia.

From the right pleural cavity, 1,040 ml. of serosanguineous fluid was removed. A daily dose of 150 mg. (3 mg. per kg. of body weight) of 6-mercaptopurine (6-MP) was started (Fig. 1). Five days later, the cervical nodes had enlarged and extended into the scalp and face. There was bilateral parotid gland enlargement and a left peripheral facial nerve palsy. The liver extended 10 cm. below the right costal margin, and the spleen extended 12 cm. below the left costal margin. Petechiae were present over the extremities and trunk.

The BUN was 81 mg. per 100 ml.; serum uric acid, 20 mg. per 100 ml.; hemoglobin, 8.3 gm. per 100 ml.; platelets, 15,000 per cu. mm.; and leukocyte count, 17,700 per cu. mm. with 30 percent lymphoblasts. Four days after these laboratory data were recorded, prednisone, 40 mg. daily, was added to the therapeutic regimen (Fig. 1). A rapid reduction in the size of lymph nodes ensued. Within 36 hours, almost all of the parotid swelling and cervical lymph nodes had cleared, and the facial palsy was no longer evident. After 48 hours, only a few pea-sized lymph nodes could be felt. The tip of the spleen and the liver edge were barely palpable. A chest roentgenogram was normal. A blood count showed hemoglobin, 8.3 gm. per 100 ml.; platelets, 21,000 per cu. mm.; and leukocytes 3,300 per cu. mm. with a normal differential. Bone marrow aspirate revealed only 10 percent of the cells to be leukemic. At this point, painless hematuria occurred. The BUN was 159 mg. per 100 ml. and serum uric acid 43.6 mg. per 100 ml. After 24 hours, the BUN was 153 mg. per 100 ml. and serum uric acid 43.5 mg. per 100 ml. Microscopic examination of the uncentrifuged urine revealed many uric acid crystals.

Oral fluids were forced to tolerance, and sodium bicarbonate (12 gm.) was administered daily in six equal doses. This regimen resulted in a daily urinary output of over four liters. The urinary pH was always 7.0 or greater. Within 24 hours, the gross hematuria disappeared. Prednisone dosage was rapidly reduced and 6-MP was continued. The patient was discharged on Aug. 21; there was no significant lymphadenopathy, no evidence of neurologic disease, and no hepatosplenomegaly. Laboratory data revealed BUN, 48 mg. per 100 ml.; serum uric acid, 4 mg. per 100 ml.; hemoglobin, 9.1 gm. per 100 ml.; platelets, 181,000 per cu. mm.; and leukocytes, 2,400 per cu. mm. with a normal differential. Examination of a bone marrow aspirate revealed only a few leukemic elements. Several days after discharge, prednisone was stopped but 6-MP was continued.

Third Admission.—The patient was readmitted 13 days later with a recurrence of cervical lymph nodes

but with no acute distress. Vital signs were normal. There were no axillary or inguinal lymph nodes. The liver and spleen were not palpable. Chest roentgenogram showed a slight widening of the mediastinum, but was otherwise normal. The hemoglobin was 9.3 gm. per 100 ml., platelets were 180,500 per cu. mm., and leukocytes were 3,200 per cu. mm. with a normal differential. The bone marrow had not changed since Aug. 21. The BUN was 18 mg. per 100 ml. and serum uric acid was 7.4 mg. per 100 ml.

Management consisted of forcing oral fluids, 12 gm. sodium bicarbonate daily, and 20 mg. prednisone daily. After five days there was no decrease in the size of the lymph nodes. Prednisone was increased to 60 mg. daily, 6-MP was discontinued, and hydroxyurea was started in a dose of 1 gm. (20 mg. per kg. of body weight) daily in two equal portions (Fig. 1). After three days of therapy at the higher prednisone dose there was no decrease in lymph node size; thereafter, the daily dose was rapidly reduced. Throughout the third hospitalization, the patient was comfortable. There was no increase in the size of cervical lymph nodes, new lymph nodes did not develop, and there was no detectable hepatosplenomegaly. At the time of discharge, Sept. 15, the blood count was normal. The BUN was 31 mg. per 100 ml., and a urinalysis was normal.

Fourth Admission.—One week later the patient was readmitted. For the previous three days he had suffered nausea, vomiting, and progressively increasing dyspnea. There was massive cervical, axillary, and inguinal lymphadenopathy. The liver extended 10 cm. below the right costal margin, and the spleen was 10 cm. below the left costal margin. Petechiae were noted over the entire body. A chest roentgenogram showed a large right pleural effusion and large hilar lymph nodes. A blood count on admission revealed a hemoglobin concentration of 10.1 gm. per 100 ml., 14,400 platelets per cu. mm., and 72,400 leukocytes per cu. mm. with 70 percent lymphoblasts. Serum uric acid was 53.2 mg. per 100 ml. and the BUN was 51 mg. per 100 ml. Serosanguineous fluid, 2,000 ml., was removed from the right pleural cavity.

The patient was given 60 mg. prednisone daily. Oral fluids were pushed to six liters per day, and 12 gm. of sodium bicarbonate were administered daily in six equal doses. Hydroxyurea therapy was continued. After 48 hours of prednisone therapy, only a few small lymph nodes could be palpated. The tip of the spleen and the edge of the liver were barely palpable. During this time, a urinary output of five liters daily was maintained. Urinary pH was 7.0 or greater and remained so until death. Gross hematuria did not develop; however, microscopic examination revealed red cells in the urinary sediment. Uric acid crystals could be seen on microscopic examination of uncentrifuged urine. Serum uric acid was 66.4 mg. per 100 ml. and the BUN was 171 mg. per 100 ml. Leukocytes fell to 1,600 per cu. mm. and the white cell differential count returned to normal. They stayed unchanged until the patient died. On the third day, prednisone therapy was reduced to 30 mg. daily and hydroxyurea was stopped (Fig. 1).

On the sixth day the temperature rose to 102.6 F. Coagulase positive *Staphylococcus aureus* was cultured from the blood. Methicillin, penicillin, and gamma globulin were administered. Prednisone therapy was reduced to 20 mg. daily. Thirty-six hours after his temperature elevation, the patient had a grand mal seizure and lapsed into coma. He did not regain consciousness and died five days later. The interpretation of the grand mal seizure was that intracerebral hemorrhage had been induced by thrombocytopenia. On the day before death, the serum uric acid was 18 mg. per 100 ml., and the BUN was 138 mg. per 100 ml. Permission for autopsy was denied.

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Discussion

Several mechanisms may explain the hyperuricemia and hyperuricaciduria in lymphoma and leukemia:

1. The malignancies of the reticuloendothelial system are characterized by rapidly proliferating tissue which breaks down at a rate much greater than normal.[2, 4]

2. Antineoplastic therapy may produce massive lysis of tissue rich in nucleic acid.[2, 5, 8, 11] Both of these mechanisms may result in large quantities of purines being converted into uric acid.[1, 4]

3. There is evidence that in some cases of leukemia, the *de novo* synthesis of uric acid is accelerated.[4]

4. The kidneys are commonly infiltrated with leukemic cells. Richmond and associates observed the kidneys to be more frequently involved with lymphoma than any other organ except lymph nodes, bone marrow, liver, and spleen; these authors believe that hyperuricemia is aggravated when there is associated azotemia and lymphomatous infiltration of the kidneys.[9]

In any patient, one or more of these four mechanisms may be operative.

The principles of management include maintenance of a large urinary output and alkalinization of the urine. Patients should be encouraged to drink large quantities of water. If vomiting occurs, intravenous dextrose in water should be administered.[2, 8] It is desirable to maintain a urinary volume of three or more liters daily. Uric acid is relatively insoluble at a pH less than 6.0. In alkaline urines, uric acid exists in the form of more soluble sodium and potassium salts.[1] For this reason, the administration of large

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quantities of sodium bicarbonate is recommended.[2, 8] Some patients may be unable to tolerate the quantities of bicarbonate needed.[2]

It is sometimes possible to remove uric acid crystals from the ureter and renal pelvis by catheterization and irrigation,[5] but this procedure will not effect removal of uric acid crystals from the renal tubules. Instrumentation also adds to the danger of infection. Hemodialysis[2, 8] and peritoneal dialysis[11] have been helpful in managing the oliguric and anuric phases of the syndrome.

In the case presented, the sequence of events dramatically emphasized the excessively high levels of serum uric acid which may occur in association with leukemia. Treatment with prednisone induced massive lymph node lysis, hyperuricemia, and hyperuricaciduria on two occasions. Both times the serum uric acid level had been above normal before therapy. During the second admission, the serum uric acid concentration rose from 10.5 mg. per 100 ml. at the time of admission to 20 mg. per 100 ml. five days later, and finally to 43.6 mg. per 100 ml. after the patient had received prednisone therapy. No uric acid determinations were made during a four-day interval prior to the start of prednisone therapy (Fig. 1). During this interval, lymph nodes became progressively larger. This suggests that the uric acid level at the time prednisone was begun may have been considerably greater than 10.5 mg. per 100 ml., and that the excessive rise in serum uric acid may not have resulted entirely from corticosteroid treatment.

During the fourth admission, the serum uric acid concentration just prior to prednisone therapy was 53.2 mg. per 100 ml. After three days of corticosteroids, it rose to 66.4 mg. per 100 ml. During this admission, much of the hyperuricemia may have been an expression of the metabolic abnormalities induced by the leukemia. A roentgenogram suggested enlargement of the kidneys. Thus, leukemic infiltration of these organs may also have contributed to hyperuricemia, thereby augmenting the effect of prednisone therapy and the natural course of the disease.

The decrease in lymph node size appeared to be wholly produced by prednisone. Neither 6-MP nor hydroxyurea exerted any overt effect. Good hydration and large doses of sodium bicarbonate effectively prevented suppression of urine excretion. The urinary volume always exceeded 3.5 liters daily, and the urine pH was always 7.0 or higher. An unexpected and unexplained event was this patient's failure to respond to prednisone during the third admission, followed by good response to the same drug during the fourth admission.

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Sclerosing Angioma Following Tattoo

LEONARD MELTZER, M.D., and BERNE L. NEWTON, M.D.

In this case, a sclerosing angioma developed at a tattoo site, and the histologic picture seems to confirm the tattoo as the cause.

CASE REPORTS

SCLEROSING ANGIOMA has been described by many names including fibroma durum, dermatofibroma lenticular, histiocytoma, sclerosing hemangioma, and nodular subepidermal fibrosis. These name variations may have resulted from differing opinions not only as to the origin but also as to the very nature of the process itself—neoplastic or reactive inflammatory. Fibroblasts, histiocytes, and endothelial cells have each been implicated in the formation of this lesion which is usually described as a hard single nodule, most frequently located on the leg. It adheres firmly to the overlying epidermis. The nodule may reach a size of 3 cm. in diameter, and its color may vary from waxy yellow to dusky red. Pseudoatrophy of the overlying skin may occur due to pressure. Symptoms range from none to definite tenderness. In Rentiers' and Montgomery's series of 100 cases, a history of trauma was reported in 20 percent.

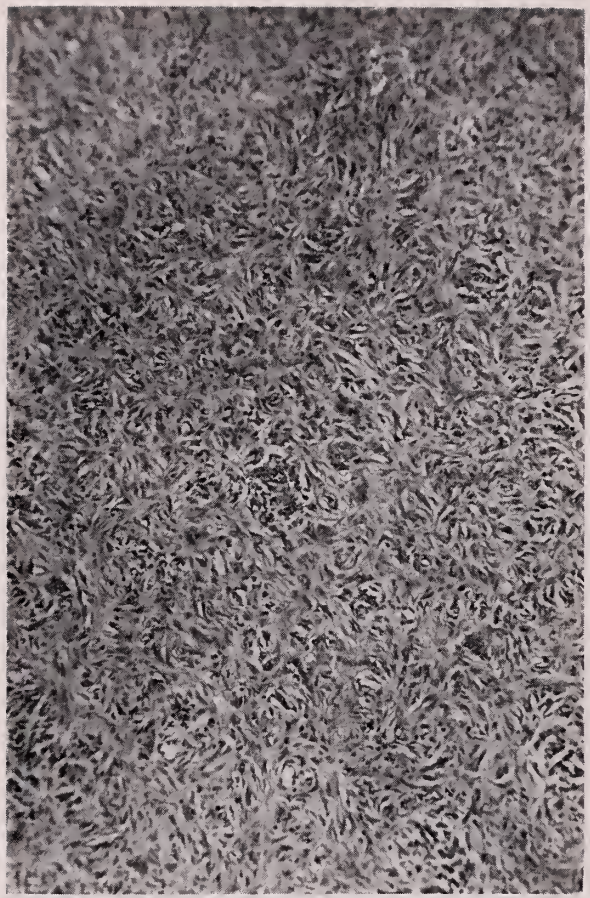
The following case supports the role of trauma in the formation of sclerosing angioma.

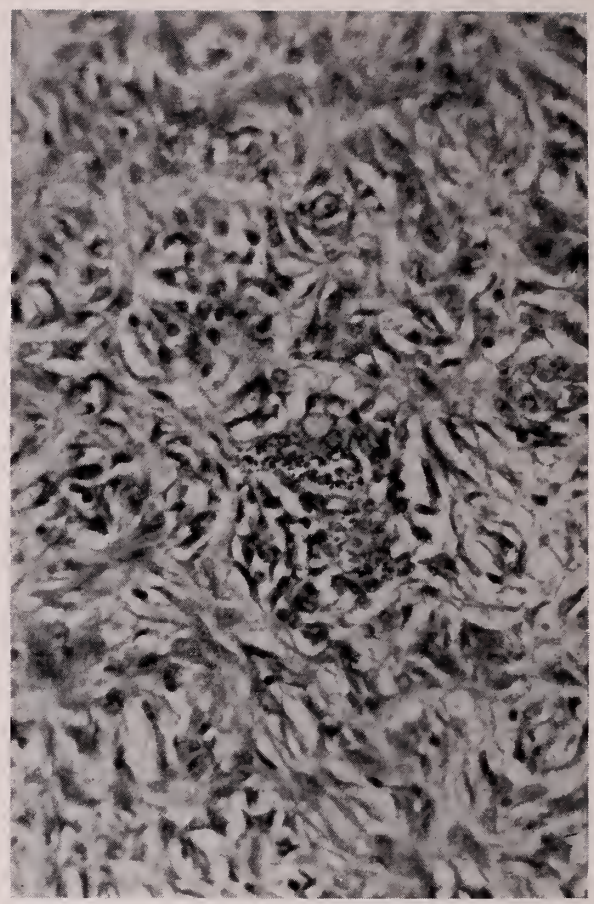
A 40-year-old white man sought treatment in May, 1963, for a tender, painful lump on the lateral aspect of the right leg. The patient stated that in 1943 a tattoo (Fig. 1) was inscribed at the site of the present lesion. Six months later a small nodule appeared in this area, growing slowly to its present size of slightly over 1.5 cm. in diameter. For the past two months there had been increasing tenderness and pain. Clinical examination showed an intimate relation between the tattoo and the nodule. The lesion was excised under local anesthesia and submitted for pathologic study.

The specimen consisted of a skin ellipse 2.5 by 1.5 by .8 cm., situated on the surface of which was a 1.7 by 1.3 cm. slightly elevated, gray-tan area. The center of this area was in turn slightly depressed and appeared blue to black with linear mottling. The cut surfaces across the short axis and at right angles to this axis revealed bright tan-yellow, homogenous, rather poorly circumscribed tissue alteration extending down to fat.

Microscopic study (hematoxylin–eosin stain) showed that the dermal structure was altered throughout its thickness except for a thin uninvolved zone apparent immediately beneath the epidermis. There was slight uniform epithelial hyperplasia in some areas on the surface and atrophy in others. The dermal change (Fig. 2) consisted of proliferating uniform-appearing fibroblasts and endothelial cells. Scattered macrophages were present (Fig. 3), some of which contained fine to moderate-sized brown pigment particles and which, by utilizing Perl's Prussian blue stain, gave a strongly posi-

Fig. 1.





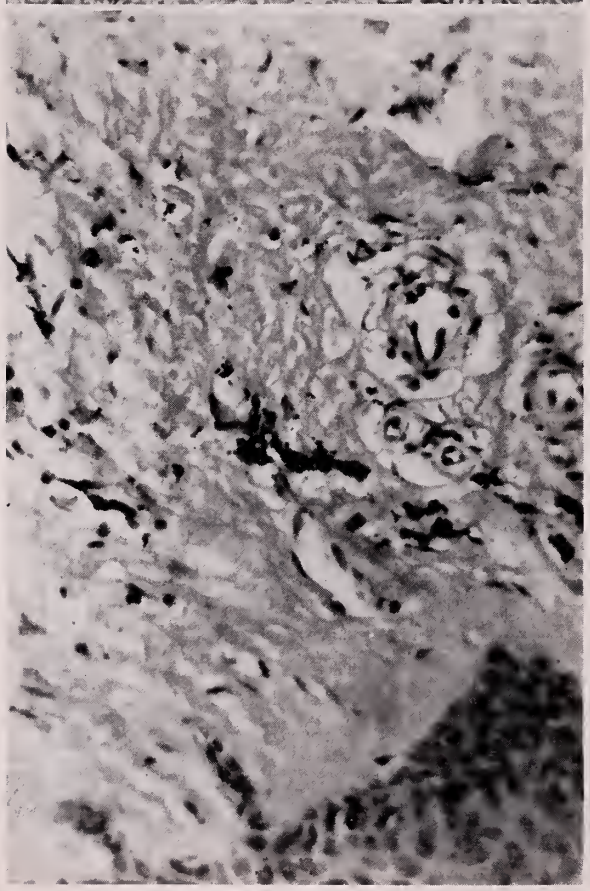


Fig. 2. Top left. X 100

Fig. 3. Top right. X 250

Fig. 4. Below left. X 250

tive reaction for iron. This pigment was considered to be hemosiderin. In addition, coal black, fine-to-coarse pigment granules which gave a negative reaction for iron were seen free in the tissues and within macrophages (Fig. 4). This pigment, which tended to be scattered in the subepidermal tissue and was uninvolved by the cellular proliferation, was interpreted as carbon pigment. Typical multinucleated giant cells were not apparent, although a few ill-defined cells of this type were seen.

The pathologic findings in this case indicate a reaction of the histiocytoma sclerosing hemangioma-nodular subepidermal fibrosis group, and not a granulomatous pattern as is described by Rostenberg and others, Sulzberger and Tolmach, and Loewenthal. Since there was evidence of phagocytic activity in the cells of the lesion (hemosiderin granules) dermatofibroma may be excluded. While more florid examples of this condition may be difficult to differentiate from the more mature appearing fibrosarcomas, especially dermatofibrosarcoma protuberans, the overall cell uniformity in this case rules out this possibility. Certain animals, particularly

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guinea pigs, upon receiving subcutaneous or intramuscular injections of certain materials (apart from recognized carcinogenic substances) may have a fibromatous or a pseudosarcomatous tissue reaction. Such fibromatous reactions occur in man, although they are apparently not truly neoplastic.

Summary

The present case illustrates the origin of a nodular subepidermal fibrosis within a tattoo. The relationship of the carbon particles to the lesion strongly suggests that the tattoo is the causative factor.

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UT Chemistry Professor Researches for Cancer Weapon

It may be possible to trick wildly multiplying cancer cells in a human patient by sowing into the cells the seeds of their own destruction.

This is the aim of research being conducted by Michael J. S. Dewar, Ph.D., Robert A. Welch Professor of Chemistry at the University of Texas.

He has been working with a newly discovered class of boron compounds. Boron compounds have been largely ignored by laboratory chemists because of their unpleasant properties.

Dr. Dewar has discovered that an atom of boron plus a nitrogen atom bears a striking chemical kinship to a pair of carbon atoms.

He hopes to replace a pair of carbon atoms in this way in one of the essential constituents of a nucleic acid, the material in the nucleus of a cell that controls its heredity.

If a cancer cell could be tricked into accepting such a compound into its nucleus, it would then spawn mutations, or new types of cells.

"Since mutant cells are almost always lethal, the cancer cells would die off rather than multiplying and spreading," Dr. Dewar explains.

The aim of Dr. Dewar's research is to find the means for boron-nitrogen atoms to be absorbed selectively into cancer cells so that healthy cells will not be harmed.

If this research is successful, physicians will have a new weapon which presumably will be effective against all types of cancer.

This facet of Dr. Dewar's research is supported by a grant from the National Institutes of Health.

Historical and Philosophical Aspects Of Iatrochemistry and Iatromechanics

Part II. Paracelsus: Alchemical Mystic

WILLIAM S. KEEZER, M.D.

Part I which appeared last month traces the influence of the alchemists to the 16th and 17th centuries. Part II brings to the scene that controversial character, Philippus Theophrastus Aureolus Bombastus von Hohenheim, better known as Paracelsus, who was convinced that the traditional art of healing was on a false path.

HISTORICAL ARTICLES

PARACELSUS (1493-1541) publicly burned the *Canon* of Avicenna and the works of Galen in front of the University of Basel. He often appeared a mountebank, his language was unbridled and abusive, and his habits were as bad as his morals. He has been described by some as a drunken quack of an alchemist and by others as a prophet and genius; but, "He stirred with impatience and vigour the weed-grown pool of the rigid authority of the schools, and advocated, in strident tones, personal opinion and independent judgment."[1] He was undoubtedly one of the most original personalities of his day, and his approach to the problems of health and disease was unique. In his efforts to form a philosophy of medicine, he attacked problems that are still unsolved today. We cannot deny that it was Paracelsus that drew attention to many questions that needed investigation.[10]

Superstition, mysticism, and false theories were the cornerstones of medicine's structure in the days of Paracelsus. In his system of medicine he placed the magnet above all other remedies in his lists of infallible cures. "A monarch of secrets," he called it. "The magnet contains mysterious healing virtues; it is impossible to dispense with it in the treatment of disease."[2] He went further on to say:

> The magnet, like the stars and other bodies of the universe, is endowed with a subtle emanation, '*fluidum*,' that has a favorable influence on the health and life of man. It assures cure for discharging sinuses of the limbs, for fistulae of the various parts of the body, for fluxes of the eye, ear, and nose, and for jaundice and dropsy. It stops hemorrhagic disturbances in women.[2]

In his *Volumen Paramirum*, as quoted by Pachter, Paracelsus states:

> The stars determine nothing, incline nothing, suggest nothing; we are as free from them as they are from us.
>
> . . . . . .
>
> The stars and all the firmament cannot affect our body, nor our color, beauty and gestures, not our virtues and vices. . . . The course of Saturnus can neither prolong nor shorten a man's life.[5]

The Paracelsian concept of the world is centered around his basic concern with man's relation to God. "His basic motif is man as the beginning and centre of all creation. In man all life culminates. He is the centre of the world; everything is seen in terms of man."[3]

Through the principle of a hierarchical order of creation, ascending from matter to God, Paracelsus writes in all the contradictions of a paganizing mysticism of nature and a pious Christian faith. "Only a few men before him and hardly any after him have conceived an anthropocentric system so lofty and at the same time so rational. In it everything follows logically from one source; it is a system that elevates profession to vocation, trade to art, and science to wisdom."[3] Paracelsus stated:

> If a man wishes to become acquainted with many diseases, he must set forth on his travels. If he travels far, he will gather much experience, and will win much knowledge.[3]

Paracelsus set forth on this voyage of dis-

covery through the world, which, with a few interruptions, was to last until his death, leading him through all the lands of western Europe. He visited the mining districts, studied the healing springs and made long series of chemical experiments. He continued always to practice as a physician, helping and healing wherever he could. He had thrown away his doctoral biretta and wore an ordinary slouch hat, and disciples, many of low degree, joined their fortunes with his. It was a motley company that moved from place to place. As he traveled, it became more and more evident to him that the traditional art of healing was on a false path. Its theories were false and its therapeutic methods were false. He began to keep a record (in his native German) of his own observations and ideas.

His great aim was to break the bonds of ancient authority and accepted dogma, and open the way for a science based on open-minded experience, experiment, and observation—or, as Paracelsus would say, on the "Light of Nature." For him, the phenomena of nature, whether seen or hidden, were the revelation of God and God's will. Though his natural philosophy was deeply rooted in the Neoplatonic philosophy of the Florentine Academy,[7]* Paracelsus was too much an original thinker to be a strict adherent to this or any other form of philosophy.

As the earth was thought to be the center of the material universe, so man was considered the center of the external universe,

PARACELSUS at the age of 47, from a wood cut by August Hirschvogel. (Reproduced with permission of Abelard-Schuman, Ltd., New York.)

man being the microcosm with the external universe the macrocosm. Through their spirits and occult properties, all things in the external universe, sun, moon, stars, planets, animals, metals, and waters, exerted definite influences upon man. The opposite was also thought to be true—that man through knowledge and wisdom would be able to exert an influence on the powers of nature in marvelous occult ways. Jacobi interprets Paracelsus:

> The created world has been given over to man in order that he may fulfill it. More than that: man's original and specific mission is to lead it to perfection; he has been placed in the world solely for this purpose.[3]

The medieval hermetic alchemy of the transmutation of metals was not only a chemical process, but in addition, a spiritual and psychic process symbolic of the pattern that was followed by everything in creation. The four main pillars on which Paracelsus founded his science were: philosophy, the science of the material and elemental aspect of creation; astronomy, the science of the sidereal aspect of creation (these two in

*This influence of medieval Neoplatonism may have reached Paracelsus in several ways. He knew Arnald of Villanova and Hildegard of Bermersheim, as well as the alchemical Lullists and John of Rupescissa.

their interrelations and their essence are the prerequisites for penetrating the structure of men); alchemy, the science of the natural phenomena and their interactions; and virtue *(proprietas)*, the fourth pillar, which gives the necessary support without which the other three pillars could never stand.

This resolves itself in the hands of Paracelsus into recognition of, and obedience to, the will of God and to his direction of the universe through the powers of nature and the teachings of Christ. No man can be a good physician unless his concept of the medical mission is based on love and ethics. He rejected the four Aristotelian elements of fire, air, earth, and water and substituted for them his three alchemical elements: mercury, the principle of liquidity and volatility; sulphur, the principle of combustibility; and salt, that which is permanent and resists the action of fire.[7, 12]

The "prime matter" of the world, in the Paracelsian view, must be distinguished from the prime matter of individual objects. When dealing with the world, it is the word *fiat*, the Logos of the Fourth Gospel, the Platonic archetype and ideal pattern of the world that is to become manifest in material creation. In this case it is spirit and is also called "ultimate matter." On the other hand, the original state of an individual object, the state in which it was created, is its prime matter and this is *seed*.[8]

Water was the matrix (womb) of the world and it became the matrix of man. God created man to give his spirit a dwelling place in the flesh. God made an animal body from the trinity of mercury, sulphur, and salt, and after it was created, it was endowed with the "astrol" spirit of life. From this it can be seen that for Paracelsus, man has an animal body and sidereal body that gives it life.[3] Man, therefore, is an image of the macrocosm or the "great creature," but man is the microcosm. Paracelsus had the Aristotelian and Platonic viewpoint about women, i.e., they are merely vessels. Paracelsus states:

> Woman is like the field in which the child is sown and planted. She is the maternal womb (matrix) of man, like a tree bearing fruit. Man is like the fruit that the tree bears.[3]

He goes further to say that good fruit comes only from good seed and that God left the planting of the seed to the decision of man and that this decision depends upon man's will. This free decision lies with both man and woman—it requires both to have the desire for the planting. Paracelsus followed fairly closely the beliefs of Duns Scotus (1265-1308), a Franciscan who was much closer to Plato than to Aristotle, and believed in Free Will.[5]

Further proof of Paracelsus' belief in Free Will and transcendence is shown in the following quotations:

> Just as the firmament with all its constellations forms a whole in itself, likewise man in himself is a free and mighty firmament. And just as the firmament rests in itself and is not ruled by any creature, the firmament of man is not ruled by other creatures, but stands for itself and is free of all bonds. For there are two kinds of created things: heaven and the earth are of one kind, man is of the other. . . .[3]

He goes further in this discussion of man's transcending the stars when he states in *Paramirum*:

> The course of Saturn disturbs no man in his life, neither lengthens nor shortens it. For if Saturn had never been in the heavens nor in the firmament, people would be born just so, and though no moon had been created still would people have just such natures. You must not believe that because Mars is cruel, therefore Nero was his child. Although they had the same nature neither obtained it from the other. You see Helen and Venus of one nature, and though Venus had never existed still would Helen have been a strumpet, and although Venus is older than Helen consider that before Helen there were also strumpets.[12]

> Man's wisdom is in no way subjugated, and is no one's slave; it has not renounced or surrendered its freedom. Therefore the stars must obey man and be subject to him, and not he to the stars. Even if he is a child of Saturn and Saturn has overshadowed his birth, he can still escape Saturn's influence, he can master Saturn and become a child of the sun.[3]

From the foregoing quotations it can be seen that Paracelsus did not agree wholeheartedly with the almost universal belief in the influence of the stars. This customary mythical interpretation concerning the nature of the influence exerted by the heavenly bodies on man was popular until the middle of the 1700's, Paracelsus and his writings to the contrary.

H. Ranzau (1676) makes the statement that the first cause of disease was the fall of the first man into sin and that the second cause was the influence of the stars (astrology). These statements may be accepted as

Dr. Keezer, the medical director of the Oklahoma State War Veterans Home Facility at the Central State Griffin Memorial Hospital in Norman, has acknowledged indebtedness to Drs. Carlton W. Berenda and William Horosz of the Department of Philosophy, University of Oklahoma, for their constructive criticism of this article.

representing the conventional and purely mystical view of the matter.[12] The main interest of Paracelsus lay in medicine, and since he rejected the ancient authorities on both theory and practice of medicine, it is necessary to discuss his own theories on that subject. He made great effort to harmonize this with his philosophy of nature and the results of his experience and observations. On this question as to the causes of disease, Paracelsus distinguished five causes of disease or five principles. They might be called powers or realms. They were (1) *Ens Astri* (the influence of the stars, astrology), (2) *Ens Veneni* (the influence of poisons), (3) *Ens Naturale* (the influence which exists in the nature of the individual, the microcosm), (4) *Ens Spirituale* (the influences acting not directly upon the body, but through the spirit), and (5) *Ens Dei* (the influence of the will of God acting directly to produce illness by way of warning or punishment).

With respect to the first principle, we have already seen that he recognizes the influence of the stars without admitting their control on the destinies of man. In his discussion of the second influence, he points out that the body was given to us without poison, but that we must give the body food, and this food contains poison. In the body, food and poison must be separated. This separation is effected by the archeus (a directing force or spirit) which is situated in the stomach. As long as the archeus performs properly the body thrives; but should it become ill or incapacitated, the separation is incomplete and the body suffers.

The third influence or principle *(Ens Naturale)* is somewhat more complicated. Man, the microcosm, was for Paracelsus the epitome of the macrocosm, and in man the counterparts of all the external universe were to be found. Just as the external universe has its sun, moon, and planets, so the microcosm has its sun, moon, and planets. For example, the brain corresponds to be the moon, the lungs correspond to Mercury, the liver to Jupiter, the kidneys to Venus, the gall to Mars, and the heart to the sun. Each of these, like the heavenly bodies, has its predestined and predetermined course.

In regard to the fourth influence, present day thinking would consider it fantastic and fanciful, but remember that in Paracelsus' day witches and sorcerers were tortured and burned. To Paracelsus, however, this *Ens Spirituale* was neither the soul nor the devil nor any of the devil's effects. In short, these were the diseases of mental disarrangements. In the *Ens Dei* he recognized the influence of a will of God upon the health of men, but rather than accept the theology of the time, he emphasized the idea that God preferred to work through nature, rather than miraculously by a direct interference.[12]

There are five causes of disease, so there must be, according to Paracelsus, five kinds of doctors. However, Paracelsus did not state his five categories of doctors were supposed to fit the five kinds of diseases. The following is a list of the types of doctors as described by Paracelsus: (1) *Naturales*, who treated contraries with contraries; (2) *Specifici*, who empirically used specific remedies; (3) *Characterales*, who treated "like with like"; (4) *Spiritales*, who used chemical drugs in connection with mental disturbances; and (5) "Christ and the Apostles," who intervened between man and God.[5] To quote Paracelsus: "The physician is a servant of nature, and God is the master of nature."[12]

According to Paracelsus' viewpoint, man is subject not only in part to nature but also to the products of his own creation. Man has a rational soul (intelligence), and he has that noble range of emotions which is called his soul proper. These higher functions enable man to be free and to aspire to divinity. One who lives by his powers of reason (wisdom acquired by the study of nature) is virtually free. He is then not subject to the stars, since he orders his own destiny. It is through these powers of reason that man is able to have thoughts. To quote Paracelsus:

> Thoughts are free and are subject to no rule. On them rests the freedom of man, and they tower above the light of nature. For thoughts give birth to a creative force that is neither elemental nor sidereal. . . . Thoughts create a new heaven, a new firmament, a new source of energy, from which new arts flow. . . . When a man undertakes to create something, he es-

> tablishes a new heaven, as it were, and from it the work that he desires to create flows into him. . . . For such is the immensity of man that he is greater than heaven and earth.[3]

It now becomes clear why Paracelsus placed so much emphasis on "virtue." The higher the doctor's rational soul is elevated above his animal soul, the greater his power over it. It is the virtue of a pious man that breaks the magic circle of predestination. A different and overpowering "white magic" resides in this sphere and is immortal. Hence, Christ and the Apostles are the physicians that are able to transcend and intervene between man and God in the fifth power or realm of disease, *Ens Dei.*

> Where else can Heaven be rediscovered if not in man? Since it acts from us, it must also be in us. Therefore it knows our prayer even before we have uttered it, for it is closer to our hearts than to our words. . . . God made His Heaven in man beautiful and great, noble and good; for God is in His Heaven, i.e., in man. For He Himself says that He is in us, and that we are His temple.[3]

Paracelsus strove to establish the freedom of the individual soul through reason and creativity, but to do this, he had to postulate a final and surmounting soul which he supposed through faith to be completely free. He ended, however, by admitting that faith and science are incompatible. He had sought God in nature, but had found Christ in man, ending finally by admitting the split between science and theology which is characteristically modern.

Paracelsus, with his humanistic outlook on man, felt man capable of transcending the astrological influence of the stars by wisdom. This wisdom was acquired by the direct study of nature, wherever, and whenever possible. Above all, he did not study nature in the classroom or by consulting the ancient lore, but rather by going to nature itself.

Paracelsus was a Utopian. He overestimated the possibilities of his science. He strove to discover truths for which the science of his age had neither definition nor method of verification. He tried to grasp the whole before knowing the details. His concepts are poorly defined, and his language shows a struggle with words.

In 16th century England, Paracelsian ideas helped bring medical men back to nature. Galenic medicine at this period failed to keep in touch with fact by personal verification and experiment. However, it had its liabilities, for it tended to import supernaturalism and mystical allegory more intimately into the science of medicine.[4]

Paracelsus will always be remembered for the introduction of many chemical remedies, but he did infinitely more in that he attacked the basic problems of the healing art, asking for the how and why. He was a scientist in search of a philosophy of medicine. He used the experiences of medicine and science as materials in order to create a synthesis. He wanted to understand the world in which he was living and man's part in it in health and disease. His approach to the problems was that of a vitalist and spiritualist.[11]

Paracelsus worked long at rationalizing the alchemical tradition, in the interests of his medical art. The details are bizarre, and he had no conception other than a qualitative one and above all, no appreciation of the power of mathematics,[9] such as was to emerge in the philosophy of Descartes.

[Next month: Part III. René Descartes: Dualistic Mechanist.]

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Comments on the Connection Between Paracelsus and Faust

The legend of Dr. Faustus or Faust, now famous in literature, was enlarged by details drawn from the life of Paracelsus. Goethe in particular drew heavily from his study of Paracelsus in developing the main character of his master drama. The earlier work of Christopher Marlowe, *Tragical History of Doctor Faustus*, also describes a central figure much like Paracelsus.

During the Renaissance and Reformation, there sprang up in Europe a tale of a magician and charlatan who was ready to forego salvation and risk damnation to gain understanding and power over nature. Dr. Faustus, the story went, had traveled extensively and studied all forms of books but had found inadequate the learning of his age. Desiring to know the mysteries heretofore concealed from human eyes, he bargained with the Devil. He was given power over space and matter and created the homunculus (little man). A black poodle, really a devil, was at his side constantly. When Dr. Faustus died, the dog ran away with his soul.

In *Index sanitatis*, Philipp Begardi places Faust with Paracelsus, "wicked, cheating, useless and unlearned doctors." In *Ignatius*, John Donne places Paracelsus among four pretenders to the "principal place right next to Lucifer's own throne," beside Copernicus and Machiavelli.

Henry M. Pachter has claimed that Goethe's interpretation of the Devil is Paracelsian as is the idea of the two souls in Faust's heart. Goethe introduces a new thought to the Faust tale. Unable to believe that knowledge leads to damnation, he absolves the hero. When the Devil appears to take Faust to Hell, the angels are there, too, and say:

> Him who unwearied still strives on
> We have the power to save.

Goethe's *Faust* departs widely from the older versions of the legend. Faust does not repent but simply emerges by the inborn force of his character into a higher state:

> Man errs so long as he is striving,
> A good man through the obscurest aspiration
> Is ever conscious of the one true way.

This idea inspired a similar theme revealed by Robert Browning in *Paracelsus*.

Rumors of his day held that Paracelsus may have been the damnable Doctor Faustus; however, there were others who were credited with being Faustus. Paracelsus himself became a legend, and as recently as 100 years ago, during an epidemic of cholera, people made a pilgrimage to his grave in Salzburg, searching for magic healing.

So the stories of Faust and Paracelsus have become linked and are at times the same story.

Clinicopathologic Conference

Ray Remmers, Jr., M.D., Robert O. Morgen, M.D., Gilbert Ross, Jr., M.D., Richard P. Lyon, M.D., Albert J. Paquin, Jr., M.D., Kenneth J. MacKinnon, M.D., and V. A. Stembridge, M.D.

Case Presentation

DR. REMMERS: A 19-year-old white male was admitted to the University of Texas Medical Branch Hospitals, Galveston, Texas, on May 25, 1963.

He was said to have been in good health until four weeks before this admission at which time he developed a dull constant left flank pain with radiation into the left lower quadrant and the left testicle. He was hospitalized in another city where x-rays were said to have revealed multiple bilateral large radiopaque renal stones and a large left ureteral stone. An excretory urogram revealed a nonfunctioning right kidney with minimal function on the left. Two serum calcium and phosphorus levels were within normal limits. X-rays of the teeth revealed a normal lamina dura. A left ureterolithotomy was performed during the initial hospitalization. One week later stones passed down the left ureter causing obstruction and a left nephrostomy was then performed. Several blood urea nitrogen (BUN) determinations during this hospitalization ranged from 20 to 30 mg. per 100 ml. He did well postoperatively despite some bleeding from the nephrostomy site, and was discharged May 13, 1963. However, on May 17 he again noted severe left flank pain followed by significant bleeding from nephrostomy site. He was readmitted to the hospital where his hemoglobin was noted to be 6.0 gm. per 100 ml. He received several transfusions of whole blood. The bleeding stopped spontaneously, but recurred on May 20 and required repeated transfusions. He was transferred to the Medical Branch Hospitals on May 25 for further evaluation and treatment.

Past and family histories and systems review were nonrevealing. There was no history of previous flank pain, passage of urinary stones or pyuria. At age 10 he experienced a two-day episode of painless hematuria after falling on his back. There was no hematuria prior to or after that episode. His parents and siblings were all living and well.

Physical examination on admission revealed a pale 19-year-old white male in no distress. He appeared to have lost considerable weight, but otherwise appeared healthy. There was active bleeding from the left nephrostomy site. The nephrostomy tube could not be irrigated. His temperature was 101 F. and his blood pressure was 170/90. Some of the laboratory findings are shown in Table 1.

Additional laboratory studies not recorded in the table included many urinalyses which

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This is the ninth in a series of clinicopathologic conferences from the University of Texas Southwestern Medical School. Dr. C. T. Ashworth, professor and chairman of the Department of Pathology, serves as guest editor for the series.

This month, the Conference comes from the 43rd Annual Meeting of the South Central Section of the American Urological Association, Inc. The president of the group, Dr. Charles A. Hooks of Galveston, and the Executive Committee made the conference available for this series. Participants included Dr. Ray Remmers, Jr., of the Department of Medicine, University of Texas Medical Branch, Galveston, moderator, with clinical discussions by Dr. Robert O. Morgen, Department of Medicine, Baylor University College of Medicine, Houston; Dr. Gilbert Ross, Jr., Department of Urology, University of Missouri School of Medicine, Columbia, Mo.; Dr. Richard P. Lyon, Department of Urology, University of California School of Medicine, San Francisco; Dr. Albert J. Paquin, Jr., Department of Urology, University of Virginia School of Medicine, Charlottesville, Va.; and Dr. Kenneth J. MacKinnon, Department of Urology, Royal Victoria Hospital, Montreal, Canada. Pathological findings and discussion are by Dr. V. A. Stembridge, Department of Pathology, the University of Texas Southwestern Medical School, Dallas.

Presented is a case of a 19-year-old male with nephrolithiasis and advanced renal disease.

showed from one to two plus proteinuria, occasional red cells, and too many white cells to count. Total serum protein, albumin-globulin ratio, fasting blood sugar, blood sugar two hours after a meal, and serum cholesterol were all within normal limits. No stone analyses were recorded.

TABLE 1.—*Some Laboratory Findings During Hospitalization.*

	Hospital Days						
	1	3	6	7	8	9	12
Hgb.	10.0	10.8	10.2				8.1
WBC	12,100		18,700	18,700			23,000
Na	126	118	131	136	141	134	134
K	4.5	4.8	4.7	5.4	4.1	4.7	4.5
CO_2	14.5		15.1	10.2	21	14.5	14
Cl	96		88	85	103	97	91
BUN	77	106	147	183	71	117	134
Ca		7.9	6.8	Hemodialysis			
PO_4		5.0	8.0				

Bleeding time, clotting time, prothrombin time, platelet count, serum fibrinogen, and clot reaction were all within normal limits. A persistent shift of the neutrophils was noted with some toxic granulations. Chest x-ray on admission revealed evidence of pulmonary congestion with borderline cardiomegaly and minimal bilateral pleural effusions. The electrocardiogram was normal. Urine culture revealed *Escherichia coli* in the right kidney and *Pseudomonas* in the left kidney.

Course: The patient was mildly febrile during the initial hospital stay. He was admitted to the Urology Service and shortly thereafter taken to surgery where a left flank exploration was performed because of persistent bleeding through the nephrostomy site. The kidney pelvis was opened and 12 to 16 small stones and numerous clots were removed. The previous operative area was explored for sites of bleeding; however, none were found. The kidneys were thought to be both normal in size but hydronephrotic. A kidney biopsy was attempted, but no renal tissue was obtained. In the entire area there was considerable oozing which was finally controlled by packing with gauze. A Pezzar head catheter was placed in the previous nephrostomy site and the wound was closed in the usual manner. The patient was treated with tetracycline, streptomycin, and numerous units of whole blood. Over the next three days the bleeding from the left nephrostomy gradually subsided. During this period his urinary output varied from 1,100 to 1,500 ml. per day, about one-third of this coming from the left nephrostomy tube and the remainder from an indwelling Foley catheter in the bladder. His temperature which had been elevated for the first 48 hours gradually fell to normal. On his fourth hospital day, increasing dyspnea, tachycardia, and a ventricular gallop were noted and were thought to be due to overhydration. These symptoms responded well to fluid restriction in 24 hours. At this time the previous antibiotics were discontinued and penicillin and chloromycetin therapy were instituted.

Because of the progressive renal failure, on the fifth hospital day the right kidney was explored in an attempt to establish urine flow on that side. Several stones were removed, including a large staghorn calculus. A nephrostomy was performed in the usual manner. The patient tolerated the procedure well. His output increased transiently following surgery, then progressively fell. On the seventh hospital day, an uneventful hemodialysis was carried out employing the Twin Coil Artificial Kidney and regional heparinization. There was vast clinical im-

provement in the patient after hemodialysis, however clinical and laboratory signs of renal failure recurred over the last five hospital days. During this time his urinary output also gradually fell to approximately 500 cc. per day. On the 12th hospital day the bleeding recurred from the site of the left nephrostomy and became severe on the 13th day. Whole blood and intravenous fluids were administered. On the evening of the 13th day, he became extremely hypotensive and died shortly thereafter despite the administration of further whole blood and intravenous fluids.

Discussion

DR. REMMER: This is a case of a young man who presented with a multiple renal calculi and severe chronic renal failure with a rapid downhill course. Because we have a large panel, and especially because this case does not particularly lend itself to the classical CPC type of discussion. I have asked the panel to comment critically on various aspects of this disease, especially the diagnostic workup and management. I feel that this type of constructive criticism is extremely valuable.

Dr. Morgen, would you like to proceed?

DR. MORGEN: This young man, allegedly asymptomatic until six or seven weeks prior to death, presented with striking bilateral nephrolithiasis. The calculous disease was associated with bilateral, obstructive nephropathy, certainly, at the least, bilateral, partial intrapelvic obstruction. The presence of secondary, acute or subacute active pyelonephritis is a virtual certainty as evidenced by persistent proteinuria, pyuria, and bacilluria accompanied by a febrile course and leukocytosis. Since we cannot determine the duration of the calculous disease, and because it may have been present for many months or even years, it seems reasonable to raise the distinct possibility of longstanding, chronic pyelonephritis as a factor underlying this patient's renal insufficiency. While only mildly azotemic at the time of his first admission, this taken together with the excretory urogram suggests considerable reduction of renal functional capacity at that time. Subsequently, as outlined in the protocol, the patient ran a progressively downhill course characterized by rapidly increasing hyperazotemia, acidosis, and, finally, a severe uremic syndrome.

At this point we might review the available x-rays (Fig. 1). On the right side there are multiple, large, peculiar calculi that are rounded with smooth, regular outlines, and densely radiopaque, lying within the pelvocalyceal system. On the left side there are multiple, smaller calculi presumably lying in the calyces and pelvis and, perhaps, within the papillae. I do not recall previously having seen renal calculi with just this appearance. However, it is apparent that the stones are calcigerous. Diffuse nephrocalcinosis is not visible in these films, but this does not exclude considerable focal calcification within the renal parenchyma.

Patients with single or recurrent calcigerous urolithiasis frequently have no readily demonstrable underlying cause or predisposing factor. However, in young patients with severe, bilateral calculous disease or frequently recurring stones, one or more etiologic and predisposing factors or both are found in a high proportion of cases. These include a gross increase of some precipitable urinary crystalloid (such as calcium, oxalate, cystine, uric acid), an abnormality of urine acidification, an obstructive factor, or pre-

Fig. 1. Photograph of anteroposterior x-ray of abdomen showing renal calculi.

existing urinary tract infection. Since this patient's stones are densely calcigerous it is reasonable to suspect a hypercalciuric state (with or without hypercalcemia).

The major known causes of hypercalciuria fall into three groups. First, there is excessive dissolution of bone with liberation of the calcium of bone salts, and the important causes are primary hyperparathyroidism, metastatic-osteolytic bone disease, and acute immobilization. The second group is characterized by excessive absorption of calcium from the gut; the important causes include excessive ingestion of milk, the "milk-alkali" syndrome, excessive vitamin D intake, and sarcoidosis. The third type is renal tubular acidosis in which cases hypercalciuria may or may not be present. The final type is idiopathic. Of these causes of hypercalciuria the only condition that can be excluded with certainty is acute immobilization. However, most of the remainder seem highly unlikely in this case. Repeatedly normal serum calcium concentrations, apparently intact lamina dura, and absence of nephrocalcinosis weigh heavily against primary hyperparathyroidism. The patient's age, the absence of signs of a primary lesion, of bone pain, and of osteolytic lesions in the available x-rays, combined with normocalcemia make metastatic bone disease unlikely. There is no history of excessive intake of milk, soluble alkali, or vitamin D; while the history is not always conclusive we can accept it as a first approximation. I would exclude sarcoidosis because of a lack of constitutional symptoms, absence of skin lesions, lymphadenopathy, and pulmonary infiltration, combined with normocalcemia and normoglobulinemia. The absence of episodic weakness or paralysis, the normokalemia on admission, and the absence of apparent nephrocalcinosis speak against a diagnosis of renal tubular acidosis. The only clue in favor of this possibility is that at the time of transfer to the Medical Branch Hospitals the acidosis (serum CO_2 content of 14.5 mM. per liter) seems a little more severe than one would expect with the degree of hyperazotemia (BUN of 77 mg. per 100 ml.). However, such a finding might well be explained by an element of acquired renal hyperchloremic acidosis secondary to chronic pyelonephritis, inappropriate infusion of saline in the presence of severe renal impairment, or a minor laboratory error. This leaves idiopathic hypercalciuria, a condition characterized by normocalcemia, normal or reduced serum phosphate concentrations, and recurrent calcigerous urolithiasis. While there is no evidence in direct support of this diagnosis, I cannot exclude the possibility. I can only speculate that this may well have been the underlying cause of the problem.

Several other causes of nephrolithiasis might be considered briefly for the sake of completeness. Uric acid calculi may result from primary hyperuricemia, excessive ingestion of dietary purines (uric acid precursors), or persistently acid urine. Since these calculi are usually small and radiolucent I must conclude that this possibility is highly improbable. Cystinuria may be excluded because of the atypical history and the densely radiopaque character of the stones. While cystine stones may be large and are usually round, smooth, and regular in outline, as were the calculi in this patient, they are radiolucent with rare exceptions. Calcium oxalate stones are typically densely radiopaque, but they are usually small, often irregular in shape, have a rough or spiculated surface, and may cast a mottled or "snowflake" shadow by x-ray—characteristics that are not apparent in this case. A rare cause of multiple and recurrent calcium oxalate nephrolithiasis is "primary hyperoxaluria," an inborn error of metabolism resulting in excessive production and excretion of oxalic acid. The disease usually becomes apparent in infancy or early childhood; nephrocalcinosis and an early death are common. Because of the radiographic character of this patient's stones and a relatively late onset of clinical manifestations, this possibility seems unlikely to me. Finally, a remote possibility exists that this young man had primary urinary tract infection and pyelonephritis with the formation of "fibrin stones" with secondary precipitation of calcium phosphate salts and magnesium-ammonium phosphate encrustation.

With regard to the bleeding that occurred from the nephrostomy sites, I suspect that local factors were responsible. The coagulation studies that were made exclude most of the common abnormalities of coagulation. While a first stage coagulation defect could have existed, the patient had no history of hemophilia or Christmas disease. Much of the bleeding may have resulted from small blood vessel erosion in the renal pelvis by stones or the nephrostomy tube, although

this would not explain the generalized wound oozing that was described.

The protocol suggests that this unfortunate young man's terminal event was one of sudden and profound circulatory failure. While there was active bleeding from the nephrostomy site, he was given ordinarily adequate blood volume replacement therapy in the form of transfusions and intravenous fluids. The serum electrolyte studies exclude hyperkalemic cardio-intoxication, but the severe acidosis may have been a contributory factor. Yet another speculative possibility is bacteremic shock, especially in view of the persistent renal infection and multiple surgical manipulations.

One comment regarding management seems pertinent. Over the past two years we have had a great deal of experience with permanently indwelling arteriovenous cannula systems as well as with earlier and more frequent hemodialyses in the management of various types of severe acute and chronic renal failure in the MacDonald Renal Failure Unit of St. Luke's Hospital. As a result of this experience I can suggest that early cannulation and earlier and more frequent hemodialyses probably would have resulted in improvement of the patient's overall condition, would have made him a far better surgical risk, and would have allowed more time for investigation and treatment. This is pertinent in this patient because of his youth, normal-sized kidneys, and some question regarding life-sustaining recoverability of renal function.

In summary, I propose that this patient's primary problem was bilateral, calcigerous nephrolithiasis, that the stones most likely are composed of calcium phosphate predominantly, that there was an underlying metabolic abnormality leading to hypercalciuria, and that this well may be idiopathic. I suspect that microscopic examination will reveal extensive nephrocalcinosis as well as severe pyelonephritis.

DR. ROSS: I wouldn't think that the apparent discrepancy between the degree of acidosis and a BUN of 77 is necessarily suggestive of renal tubular acidosis in a patient who is septic, not eating, exposed to recent operative trauma, and who acidifies his urine to a pH of 6.0. There is no information in the protocol that definitely supports a diagnosis of any metabolic disorder but such must obviously be considered. I think that in spite of the densely radiopaque stones that cystinuria or other metabolic abnormalities are definite possibilities. Unfortunately, there was not enough information on the architectural status of the conduit system to rule out some form of occult obstruction. The only history suggesting prior urologic difficulty was a brief episode of hematuria at the age of ten; but I think we have all seen patients who first come in with advanced obstruction at the age of sixteen or seventeen. I would be inclined to suspect that the basic etiology of the stones relates to some form of obstructive uropathy, possibly at the ureterovesical junction. The bleeding would suggest a local lesion, although it could be related to the trauma of surgery and nephrostomy. Since the kidney and urine are rich in fibrinolytic enzymes, a trial of epsilon amino-caproic acid might have been in order. I am sure that the bleeding and multiple transfusions made all aspects of the management of his renal insufficiency more complex. The type of local lesion aside from the stones is speculative. He might have had a small hemangioma, or perhaps a traumatic A-V fistula. There is not too much evidence to support this, but the patient was in congestive failure before becoming oliguric, and was hypertensive. Postoperative deterioration might be related to changes in renal hemodynamics from operation, an acute exacerbation of pyelonephritis, and possibly necrotizing papillitis. The final cause of death would appear due to a combination of factors including hypovolemia, possible septic shock, or likely a pulmonary embolus. My last comment regards the use of tetracycline. We have had the experience of seeing tetracycline produce acceleration of azotemia and I think it is well accepted that tetracycline does induce protein catabolism and accelerate the elevation in BUN, creatinine, and phosphorus.

DR. LYON: What were the urine densities running throughout this? Were they 1.010 or were they less?

ANSWER: They were all around 1.010.

DR. LYON: Such densities would weigh in favor of primary renal-vascular disease as against a primary obstructive process where densities tend to reach maximum values of 1.006, even in the presence of dehydration.

Dr. Ross has already given a comprehen-

sive summary of the stone problem, and my contribution to this discussion should be additive. Therefore, I would like to run quickly through some ideas relative to the kidney problem itself. First, any chance of salvaging this patient seems remote, regardless of how the case might have been handled; however, we must continue to learn from such experiences. It seems to me that with the data at hand in this case, a correct diagnosis will have to be achieved by reaching into the grab-bag.

As to management, I would defer to my teachers, men of great experiences in eras where such staghorn stones were common problems, who, when they see a staghorn stone resting easily in place, are most likely to do as Dr. Chute suggested earlier: watch and think before deciding to attack the kidney itself surgically. Here, we do not know whether infection was present before the original surgery was undertaken. Such infection could only affect the final renal lesion leading to renal failure. If urine sediments in this case suggested inflammation with obvious bacterial invasion, one would be influenced toward not "rocking the boat," beyond removing the active obstruction at hand. This was initially done by removing the ureteral stone which was the first obstruction. When the second stone or stones reobstructed the same ureter, I would have been sorely tempted to carry out a repeat procedure, attempting to remain outside the renal parenchyma, hoping in this way to get in and out without introducing infection or upsetting the existing fine line of renal balance.

Secondly, what happened to the patient on the third day is important, for something had changed. Although his urine output remained close to 1,500 cc., which is enough urine of 1.010 specific gravity to excrete the usual 24-hour protein breakdown products, uremia and acidosis progressed rapidly. Something more serious had happened to the kidneys themselves, and this did not seem of surgical significance. Therefore, a medical approach at that time should have taken over. I would agree with Dr. Morgen that, at this point, nearness of the patient to a dialysis unit takes first importance in the hope that dialysis would provide time for effective antibacterial therapy and the enlistment of body defense mechanisms as aided by control of the existing severe metabolic and ion imbalances. If it is not possible to fly or drive the patient to this facility, we should go ahead with peritoneal dialysis as can be done in the most remote of hospitals. In the case under discussion, the opposite kidney was held suspect and yet we are not informed of any studies to confirm a site of obstruction on that side. It would seem unlikely that the opposite staghorn had coincidentally become obstructive.

A third point that should be mentioned is that close attention to water balance naturally is of the utmost importance with any serious renal problem. We have easily available a critical method of such control in the form of body weights. The existing hypotonicity was recognized by a serum sodium of 118: correction was effectively carried out, and I feel that this was done promptly enough to prevent it from materially affecting the outcome. The hope that renal function would improve with a return to normal tonicity, however, was not realized.

Finally, as to the cause of his fatal renal failure, hypoxia of renal tissue must have been a major contributor, whatever the agent. Advanced renal-vascular disease as part of the chronic pyelonephritic process tends to make any compromise in renal perfusion serious. The terminal oliguria also brings to mind the possibility of multiple cortical abscesses as the final straw.

DR. PAQUIN: I think 19 is an unlikely age for a urinary tract tumor. I tend to think of this patient as really a child with a stone. One of the significant features of his history is that at the age of 10 he had hematuria which has not been explained. Hematuria at this age in this type of patient suggests one of two things. First, that he did have congenital obstructive uropathy, which, combined with a slight blow, produced bleeding. A second explanation is that he had stones even before the age of 10, although he was asymptomatic and his only sign was hematuria. Thus, I believe his disease really began before 1963. Indeed, it had been a progressive disorder for more than nine years leading to azotemia and stones. Another interesting feature of this case is that the disease is bilateral, suggesting that there may be a metabolic disorder associated with an acid urine such as cystinuria, oxalosis, and certainly the most common stone in childhood, uric acid stone. Although some of these stones, particularly cystine stones and uric acid stones, are radiolucent, we

have no proof that over a 10-year period he did not have mixed stones with the superimposition of calcium phosphate rendering them radiopaque. The patients with oxalosis I have seen have died in their late teens. In summary, we should consider two major types of congenital disorders. First, that of obstructive nephropathy with secondary deposition of stones, but I do not think that this is the case. I think that the operative findings of normal-sized kidneys associated with minimal hydronephrosis and an acid urine places the diagnosis in the second category, namely that of a calcific disease of a congenital metabolic nature. Of these the likely disease is cystinuria, oxalosis, or uric acid stones.

DR. MacKINNON: Mr. Chairman, one by one my choices were shot down, and it is always an advantage to be fourth man on a panel. I really have little to add to the points already clearly made. Tending to exclude a coagulation defect, I think, is the fact that the man has had two nephrostomies and he bled from one side only. I think it does bring up the possibility of a lesion to account for the bleeding to which attention has not been drawn. Dr. Paquin has mentioned the possibility of neoplasm, and the fact that it is most unlikely in this age group. The possibility of an A-V fistula has been mentioned by Dr. Ross. At this meeting we have discussed A-V fistulas from needles, from trauma, and from a knife, and I suppose the last possibility is from a tube. The unilateral bleeding perhaps demands a certain amount of attention, but I think it is unlikely to be anything as exotic as an uncommon neoplasm. I think it can be well explained as occurring in association with operative trauma plus the foreign bodies embraced by both the calculi and the tube.

DR. MORGEN: I would only reiterate that which the other members of the panel have already pointed out—there is inadequate information to arrive at a firm diagnosis in this case. With regard to the acidity of this patient's urine, I would consider it "alkaline." A urine pH of 6 in a patient with severe metabolic acidosis suggests a severe impairment of renal acidification mechanisms. With regard to the daily urine volume of 1 to 1½ liters, this may be regarded as relative oliguria in severely hyperazotemic patients; personally, I would not have expected such urine flow rates to have even maintained the status quo in a patient such as this. I agree that cystinuria must be considered in a patient such as this, but I would have expected clinical manifestations at an earlier age. However, I admit that this possibility cannot be excluded with certainty even though the patient's stones were heavily calcified. The radiographic appearance of the stones and the patient's clinical course do not favor a clinical diagnosis of primary hyperoxaluria as I understand this rare condition. However, again I must admit that there is inadequate data to exclude it with certainty.

Pathological Findings*

DR. STEMBRIDGE: The major pathologic changes were in the kidneys. The right was hydronephrotic and measured 12 by 12 cm. A thin shell of parenchyma measuring 3 mm. in thickness remained, and there were numerous calculi in the distended pelvis. These smooth, multifaceted calculi varied from 1 cm. to 3.5 cm. and were either white or dark brown (Fig. 2). The parenchyma of the slightly enlarged left kidney bulged

*Dr. A. J. Strano of the Department of Pathology, University of Texas Medical Branch, Galveston, performed the autopsy in this case, and made available his findings, gross photographs, and histologic preparations.

Fig. 2. Gross photograph of bisected right kidney showing hydronephrosis and thin shell of parenchyma. Numerous smooth multifaceted calcium oxalate calculi are present, some of which are white, while others are dark brown. The dark brown color is due to incorporation of blood in the stone matrix.

Fig. 3. Gross photograph of bisected left kidney. The surface bulges and presents a granular sandpaper-like grittiness. Hemorrhagic striae and indistinct cortico-medullary markings are also present.

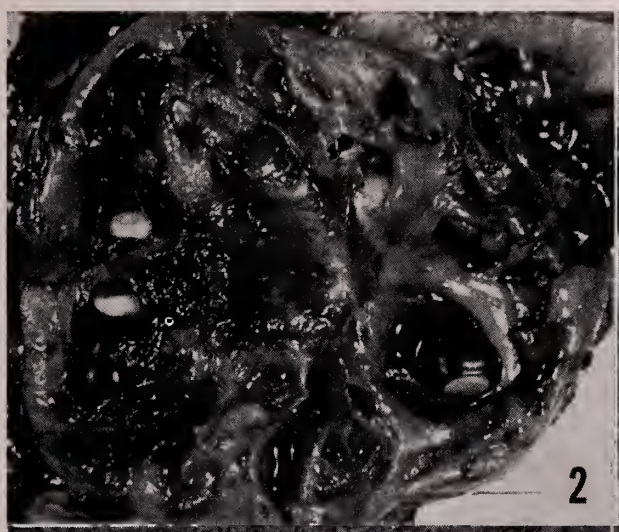
Fig. 4. Photomicrograph of kidney illustrating calcium oxalate deposits within tubules and interstices. Hematoxylin and eosin stain. (96 X)

Fig. 5. Photomicrograph of identical microscopic field seen in Fig. 4 using polarized light to demonstrate the birefringent crystals of calcium oxalate. Hematoxylin and eosin stain. (96 X)

Fig. 6. Photomicrograph of histochemical preparation of kidney for calcium. The calcium deposits are dark deposits. von Kossa stain. (34 X)

Fig. 7. Photomicrograph of renal tubule distended by radially arranged clear crystals of calcium oxalate. Hematoxylin and eosin. (415 X)

and contained numerous hemorrhagic striae which rendered the corticomedullary markings indistinct (Fig. 3). A sandpaper-like, fine grittiness could be palpated on the cut surface of both kidneys, and the sectioned surfaces presented a finely granular appearance, particularly in the left kidney cortex. Microscopically, there were found extensive deposits of crystalline material in the renal tubules and throughout the interstitial tissue (Fig. 4). These clear deposits were birefringent under polarized light (Fig. 5), and a von Kossa stain for calcium was positive (Fig. 6), indicating that the crystals were com-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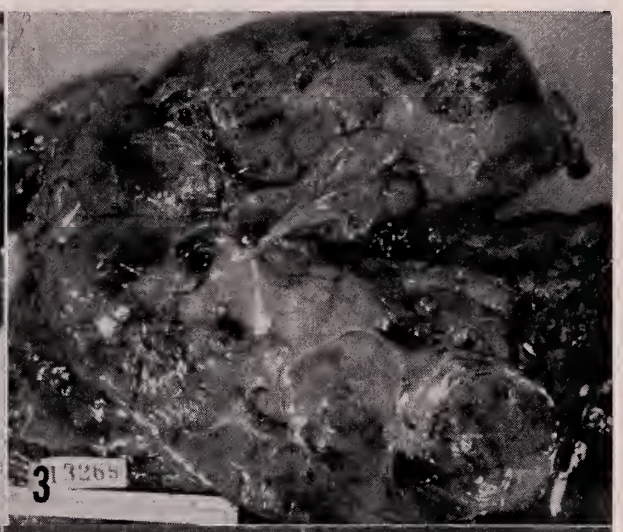
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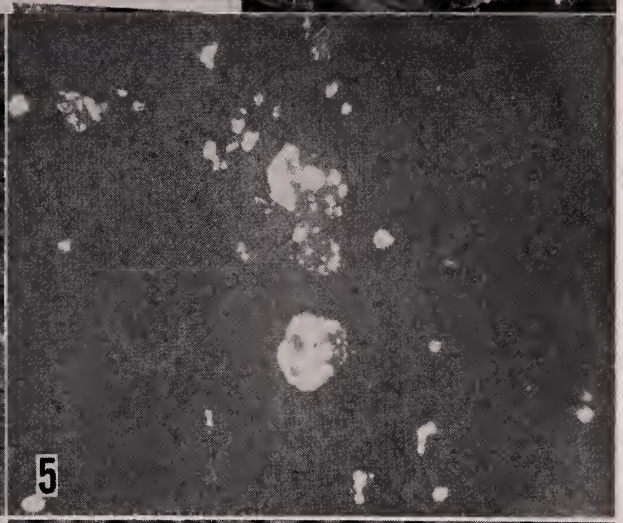
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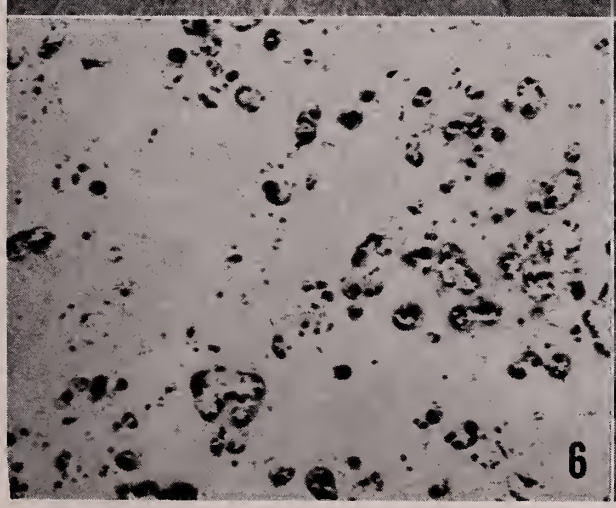
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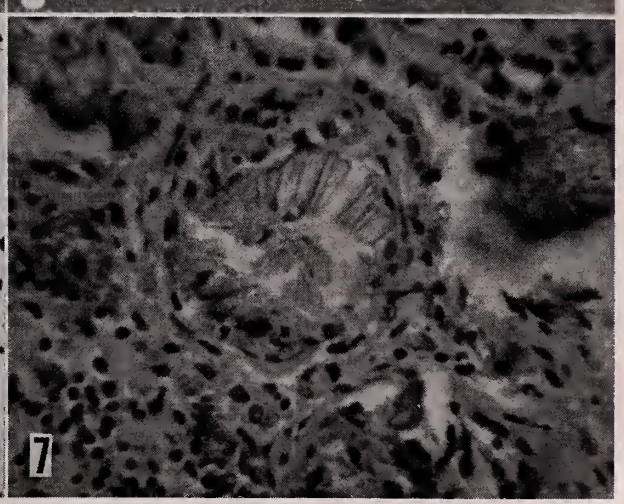
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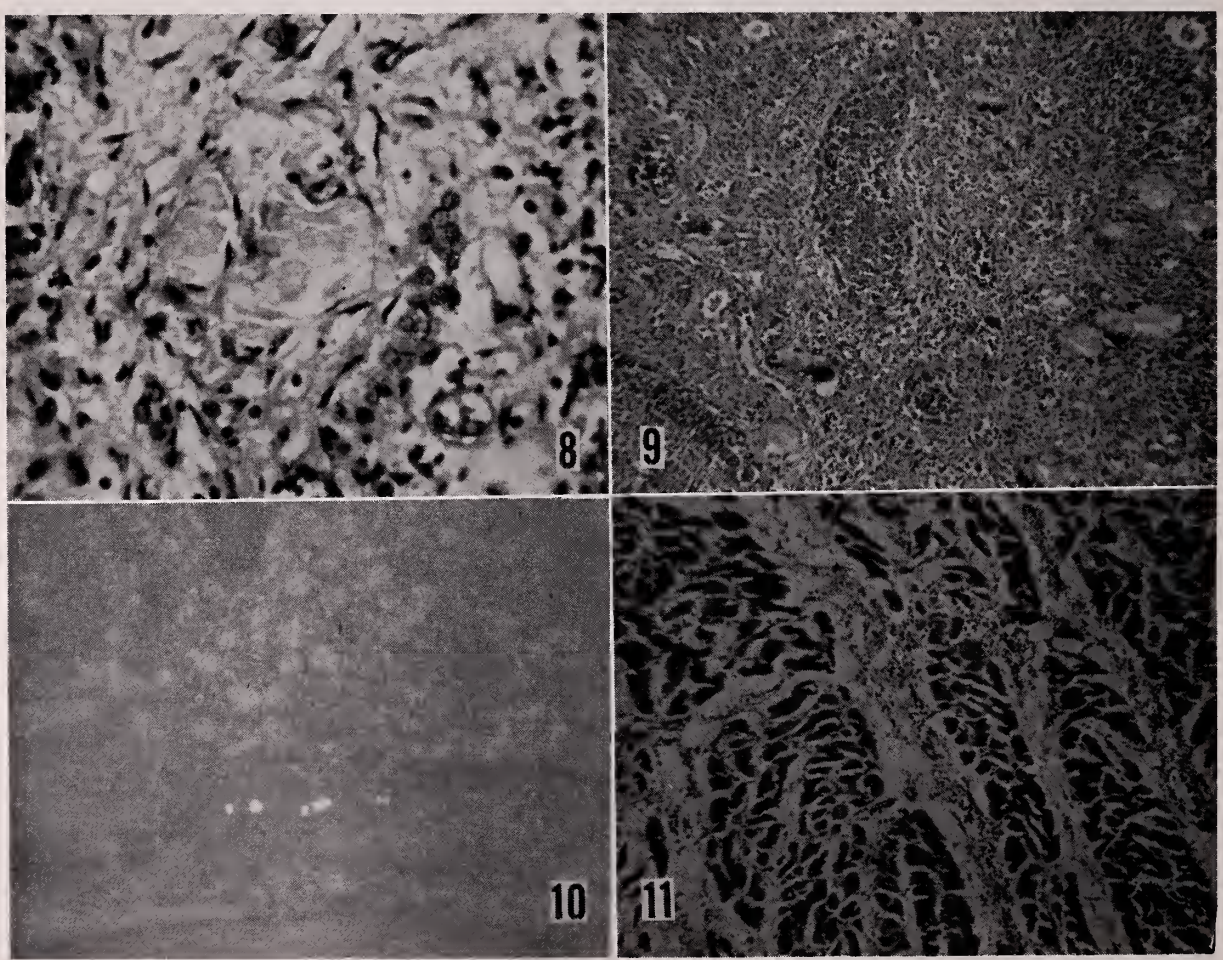
posed of calcium oxalate. The crystals presented a yellowish hue and were arranged either in sheaves, or as radially disposed prisms or rhomboids. The crystals were about 50 microns in length and 5 microns in width (Fig. 7). Aggregates of the crystals had produced tubular obstruction which was followed by epithelial cell degeneration and ultimate destruction of the tubule. The crystals frequently extruded from the lumen into the interstices where they had provoked an intense fibrous tissue proliferation and a moderate chronic inflammatory response without foreign body giant cells (Fig. 8). Most of the renal parenchyma was scarred by this mechanism. An occasional glomerulus contained calcium oxalate crystals, but none was found in the existing old pyelonephritic scars.

No calculi were present in the left renal pelvis; although within the thickened pelves of both kidneys there were numerous deposits of calcium oxalate, surrounded by fibrosis and chronic inflammation. The left kidney was diffusely involved by superimposed acute pyelonephritis (Fig. 9). Bilateral perirenal hematomas of 50-75 cc. each were present, and were undergoing organization which had entrapped some of the oxalate crystals. An anomalous renal artery was found supplying the right kidney. Severance of this vessel was responsible for the clinical blood loss. Bacteriologic culture of this right perirenal tissue yielded a growth of *Pseudomonas*.

Extrarenal deposits of calcium oxalate crystals were also demonstrated. In both the cortex and trabeculae of bone (Fig. 10) numerous anisotropic deposits had produced mild distortion of the bone architecture, but there were no marrow abnormalities. Less conspicuous crystals were present in numerous single myocardial fibers, and in the spleen and lymph nodes. No birefringent material was found in liver, thyroid, testes, lungs, adrenals, thymus, pancreas, prostate, or parathyroids. No primary or secondary parathyroid gland disturbance was observed.

The clinical uremic state was manifested



by acute fibrinous pericarditis. An intense acute myocarditis (Fig. 11) with edema was also present. Pancreatic acinar ectasia with inspissation of secretory material within the ducts was prominent. There was also evidence of congestive heart failure. The heart showed dilatation of all chambers; and there was anasarca, including moderate effusion in all serous cavities. Chronic passive congestion was present in the lungs, liver, and spleen.

Hemosiderin deposits were found in the spleen, and there was a moderate degree of fatty metamorphosis of the liver. The lungs had a moderate degree of pulmonary edema, and there were occasional hemosiderin-containing macrophages in the alveoli. A small triangular shaped scar was found in the periphery of one lung which was thought to be an area of organized pneumonia or possibly a healed pulmonary infarct. In support of the latter possibility, several organized thrombi were found in the periprostatic venous plexus.

Final pathologic diagnoses:

1. Oxalosis with deposits in kidneys, bone, heart, spleen, and lymph nodes.
2. Severe nephrolithiasis.
3. Severe hydronephrosis and acute and chronic pyelonephritis (positive culture for *Pseudomonas*).
4. Acute and chronic ureteritis and cystitis.
5. Bilateral nephrostomy wounds.
6. Bilateral perirenal hematomas.
7. Transection of anomalous right renal artery with hemorrhage.
8. Uremia with pericarditis and pancreatic acinar ectasia.
9. Acute interstitial myocarditis.
10. Congestive heart failure with cardiac dilatation, anasarca, and generalized chronic passive congestion.
11. Moderate fatty metamorphosis of liver.
12. Emaciation.
13. Hemosiderin deposits in spleen.
14. Thrombosis of periprostatic venous plexus.
15. Healed pulmonary infarct.

Although extensive renal deposits of calcium oxalate are seen in cases of acute ethylene glycol poisoning, the deposits are limited to the kidneys. The widespread distribution of oxalate deposits in our case indicates a metabolic disorder, namely oxalosis or primary hyperoxaluria. This is a genetic disorder, an inborn error of metabolism, in which the calcium oxalate crystals occur not only in the kidneys, but systemically as well. The exact site of the metabolic defect is thought to be in the metabolism of glyoxylic acid with secondary accumulation of oxalic acid.[4]

In the approximately 30 cases of oxalosis recorded in the literature, young males are principally involved, a strong familial history is characteristic, and only a few cases have been diagnosed before death. The oxalate is excreted as a saturated solution of calcium oxalate, whereupon it precipitates within the tubules as either a monohydrate or dihydrate form. The resulting calcium oxalate crystals are birefringent, clear or yellowish (in contrast to deposits of calcium carbonate or calcium phosphate which are blue in hematoxylin-eosin sections), and they stain histochemically for calcium.[2] In addition to chemical analysis,[7] x-ray diffraction studies have proved these to be calcium oxalate.[6] Patients with oxalosis have a high oxalate excretion in the urine (over 150 mg. per 24 hrs.) as compared with the normal (10-40 mg. per 24 hrs.).[1] For a while, the kidneys may successfully compensate by excreting the excessively produced oxalate, but decompensation and a vicious cycle of renal deterioration may be precipitated by acute pyelonephritis or other incidents.

Although calcium oxalate is commonly found in urine, it is not deposited in the kidneys in other forms of renal failure. The extrarenal deposits certainly support the concept of a systemic disease and mitigate against a simple pH alteration as the reason for the deposits.[4] The more usual extrarenal sites involved are bone, heart, liver, spleen, lymph nodes, thyroid, testes, adrenals, thy-

Fig. 8. Photomicrograph of interstitial renal scar in which are present extruded crystals of calcium oxalate, degenerated epithelial cells, and chronic inflammatory exudate. Hematoxylin and eosin stain. (415 X)

Fig. 9. Photomicrograph of left kidney demonstrating intense acute pyelonephritis. Tubules are distended with inflammatory exudate, and some contain clear crystals of calcium oxalate. Hematoxylin and eosin. (96 X)

Fig. 10. Photomicrograph of bone using polarized light to accentuate the birefringent deposits of calcium oxalate, here shown against a faintly visible tissue background. Hematoxylin and eosin. (96 X)

Fig. 11. Photomicrograph of heart showing separation of myocardial fibers by acute inflammatory exudate and edema, presumably related to uremia. Hematoxylin and eosin. (96 X)

mus, pancreas, prostate, and parathyroid glands.[3] An ante mortem diagnosis of primary oxalosis may be suspected or made in a young patient with nephrocalcinosis if there is a high urinary oxalate excretion. The diagnosis may be confirmed by demonstrating deposits of calcium oxalate either by kidney biopsy or bone marrow tissue.[8]

DR. MORGEN: One practical point that might become useful in the future is the recent demonstration that calcium oxalate urolithiasis can be produced in cats and rats fed a pyridoxine deficient diet and that high urinary oxalate excretion in humans can be reduced by pyridoxine. It is interesting to speculate what the role of pyridoxine might be in the management of recurrent oxalate stone formers without oxalosis. This certainly deserves careful consideration.

DR. REMMERS: Systemic oxalosis or primary oxaluria is a rare disease, there being less than 100 cases reported. You certainly will not see one every day in your office. The disease has been fairly thoroughly studied from the metabolic aspect using C_{14}-labeled amino acids.[5] The results of these studies point to an inborn error of metabolism resulting in a defect in the conversion of glyoxalate to glycine. Normally, only a small portion of glyoxalate is converted to oxalate, the major portion going to glycine. In this disease, however, a metabolic defect exists impeding the transfer of glyoxalate to glycine, therefore resulting in the excess production of oxalate. Oxalate is not metabolized in the body and because of the overproduction is excreted in the urine in excessive amounts. This ultimately results in many large calcium oxalate stones. The diagnosis can be made prior to death by demonstrating large amounts of oxalate and glyoxalate in the urine of these patients. At present there is no specific treatment for the disease.

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See mailing addresses of authors on page 1014.

Texans Have Greatest Choice

A resident of Texas has a choice of 479 insuring organizations from which to choose his health insurance protection, more than persons living in any other state in the union.

According to the Health Insurance Institute, there are over 1,800 health insuring organizations in the United States. These include 903 insurance companies, 77 Blue Cross plans, 72 Blue Shield and medical society plans, and nearly 800 other types of organization.

Second, third, fourth, and fifth-ranking states in the number of licensed health insuring organizations are Illinois with 441, New York with 418, Pennsylvania with 402, and Florida with 399.

NEWS

Legislation of Medical Interest To Be Reviewed at January Conference

MATTERS of medical interest to come before the Congress and the State Legislature will be outlined and discussed at the Texas Medical Association 1965 Conference on Legislation. To be held at the Headquarters Building in Austin Jan. 16, the Conference is in conjunction with an Orientation Program for new and transfer members.

Dr. Max E. Johnson, San Antonio, President of TMA, will open the all-day meeting with an address entitled "The Challenge Ahead in 1965." Guest speakers at the meeting will be the Rev. Walter R. Courtenay, pastor of the First Presbyterian Church, Nashville; Dr. Ever Curtis, Gloucester, Mass., AMA Committee on Medical Practice; Dr. Jack Schreiber, Canfield, Ohio, president of the Mahoning County, Ohio, Medical Society; and Joseph E. Burger, Pasadena, Calif., director of public relations, H. V. Nootbaar Company.

Both those attending the Legislative Conference and the Orientation Program will have opportunity to hear the guest speakers.

Meetings of committees and councils have been scheduled during the day, and the Executive Board will convene Sunday morning, Jan. 17.

Three panel discussions have been scheduled for the Legislative Conference. These are symposiums for county medical society officials to be held during the morning and a discussion, "State Legislation of Medical Importance," scheduled in the afternoon.

Physicians, their wives, and guests are invited to be luncheon guests of the Association. The noon meal will be served in the basement of the Headquarters Building. The Medical Protective Company has arranged a hospitality hour in the Crystal Ballroom of the Driskill Hotel at the close of the meeting.

Physicians may now make room reservations at the Austin hotel or motel of their choice. Rooms for members of the TMA have been set aside at the following facilities: Stephen F. Austin Hotel, 701 Congress Avenue; Commodore Perry Hotel, 800 Brazos; Driskill Hotel, 117 East 7th; Gondolier Hotel, 1000 South Interregional Hwy.; Terrace Motor Hotel, 1201 South Congress Avenue; Villa Capri Motor Hotel, 2360 Interregional Hwy.; and Chariot Inn, 7300 Interregional Hwy.

"A Man in a Rowboat" is the title of the address to be given by the Rev. Courtenay, Canadian-born, Georgia-reared pastor of one of Nashville's leading churches. An author and regular contributor to religious and secular publications, he is in demand as a speaker on campuses, before business associations, service clubs, and religious groups. Rev. Courtenay's sermons have been widely acclaimed. Nine have been read into the *Congressional Record*. In 1957, *Life*, in a feature about him, called one of his sermons one of the six best Easter sermons delivered in 1956. His sermon, "The Problem of Equality," was carried in full in the Nov. 18, 1963, issue of *U.S. News and World Report*.

Dr. Curtis, who when asked what her hobbies were answered, "My five children," is a Gloucester, Mass., general practitioner. She is a member of the AMA Speakers Bureau, has traveled around the world, attended Johns Hopkins, Sophie Newcomb, and Tulane, and is active in her county and state medical societies. She will speak on "The Last Mile."

"Let's All Get into the Act" is title of Dr. Schreiber's talk. The president of the Mahoning County, Ohio, Medical Society, Dr. Schreiber has been in general practice in Canfield, Ohio, since 1957. A member of the International Brotherhood of Magicians, he uses "magic" to illustrate his talks. He is a radio and TV personality, the originator and moderator of a weekly radio and TV show called "Consultation" and another radio show that gives 30-second health hints.

He also is author of several articles for *Medical Economics*.

Rep. Tunnell, who served as Speaker of the Texas House of Representatives for the 58th Session and who is seeking a second term as Speaker for the 59th Session, will address physicians on "Issues Facing the 59th State Legislature." He is a native of Tyler and represents that district. An attorney by profession, he has served as a member of the Legislative Audit Committee, vice chairman of the Legislative Budget Board, vice chairman of Texas Legislative Council, chairman of Legislative Internship Advisory Committee, and has been elected Executive

Committeeman of the National Legislative Conference.

Final speaker of the day will be Mr. Burger, a public relations executive for H. V. Nootbaar Company, Pasadena, Calif., manufacturers representatives in the feed milling industry, doing business in California and Hawaii. He is recognized as an outstanding popular inspirational speaker and has appeared before business, professional, and agricultural groups. He makes an average of 100 speeches a year before audiences in every state, Mexico, and Canada. His Austin address will be entitled "How To Get Better."

The Symposium for County Medical Society Officials—Presidents and Other Officers will be moderated by Dr. Russell Holt of El Paso. Panelists will include Dr. Tom M. Oliver, Waco, "The President—The Man in the Driver's Seat"; Dr. Walter Walthall, San Antonio, "Serious Questions Confronting Medicine"; and Dr. Schreiber, "Effective Techniques in Medical Society Leadership."

The Symposium for County Medical Society Secretaries will be moderated by Dr. R. Harvey Bell, Palestine. "The Significance of the Secretary's Job" will be presented by Dr. Walter C. Barnes, Texarkana; Dr. Dick K. Cason, Hillsboro, will tell about "TMA Forms, Reports, Membership Classification, Dues, and Deadlines"; and Dr. James H. Sammons, Highlands, will talk on "By-laws—How To Use Them."

The panel discussion, "State Legislation of Medical Importance," will be moderated by Dr. John M. Smith, Jr., of San Antonio. Panelists and their topics will include Dr. Charles H. Brown, Wichita Falls, "Mental Health"; Dr. Charles A. LeMaistre, Dallas, "Tuberculosis"; Dr. Joe T. Nelson, Weatherford, "Second Phase—MAA—Kerr-Mills Act"; Dr. Floyd A. Norman, Dallas, "Licensing of Clinical Laboratories"; Philip R. Overton, LL.B., Austin, "Licensing and Certification of Health Groups"; and Dr. C. W. Daeschner, Galveston, "Thallium Poisoning and Child Abuse Legislation."

Dr. Copeland Named to AMA Board; Houston Chosen Site of 1967 Meet

DR. J. B. COPELAND, Austin, has been elected to the Board of Trustees of the American Medical Association. Meeting in Miami Nov. 29-Dec. 2 for the 1964 Clinical Meeting, the AMA also voted to award its 1967 Clinical Meeting to Houston.

Dr. Copeland was chosen by the AMA House of Delegates to fill the unexpired term of Dr. James Z. Appel, Lancaster, Pa., who was named President-Elect.

In awarding to Houston the Clinical Meeting to be held in late November, 1967, the House of Delegates acted favorably upon the recommendation of the Board of Trustees. The Houston meeting will be the second which the AMA has held in Texas. Dallas was the site of the 1959 session.

The invitation to meet in Houston in 1967 was sponsored by the Harris County Medical Society and the Texas Medical Association. The AMA chose Houston after considering other invitations to meet in Boston, Cincinnati, Kansas City, St. Louis, and Washington, D. C.

Dr. Copeland will serve as one of 15 Trustees which have responsibility for directing the affairs of 200,000-member American Medical Association.

He is the third Texan to be selected in AMA history for membership on the Board of Trustees. Texas physicians who previously served were Dr. Edward H. Cary, Dallas, and Dr. F. J. L. Blasingame, Wharton, who now serves as Executive Vice President of the American Medical Association.

Dr. Copeland's election will provide Texas with a second position of leadership in the AMA's policy-making bodies. Dr. M. O. Rouse, Dallas, has served for the past year and a half as Speaker of the 228-member House of Delegates.

Dr. Copeland, who is Deputy Commissioner of the Texas State Department of Health, devoted 33 years to the general practice of medicine in San Antonio. He is a past president of the Bexar County Medical Society. He was a founder and first president of the Texas Academy of General Practice. He served for nine years as Chairman of the Committee on Legislation of the Texas Medical Association, and presently is in his eighth year of service on the TMA Board of Trustees. His extensive policy level experience also includes 12 years on the Texas State Board of Health, including nine years as chairman of the Board. Dr. Copeland accepted the position of Deputy Commissioner in September, 1963, with the objective of further strengthening the relationship between practicing physicians and public health services.

Dr. Copeland has served for 15 years as a Delegate to the AMA, and has been Chairman of the Texas Delegation since 1959. He has been a member of the AMA Committee on Medical Military Affairs and the Committee on Communications. He presently holds two important responsibilities as a member of the AMA Council on National Security and the Committee to Study the Organization and Function of the House of Delegates.

Chiropody Now Called Podiatry

A release from the association headquarters states that "in keeping with the national trend, the Texas organization of foot doctors, formerly known as Chiropody Society of Texas, Inc., has recently changed its name to the Texas Podiatry Association, and the practitioners now refer to themselves as podiatrists. However, it is the same organization as before, and the podiatrist is still the doctor licensed by the State of Texas who specializes in the treatment of the foot."

1965 Conference on Legislation

Texas Medical Association Headquarters Building

1801 N. Lamar Austin Texas

8:30—REGISTRATION AND COFFEE

OPENING SESSION

Sam Thompson Room

9:00—CALL TO ORDER AND WELCOME—Travis Smith, M.D., Abilene, Vice President, Texas Medical Association

9:10—THE CHALLENGE AHEAD IN 1965—Max E. Johnson, M.D., San Antonio, President, Texas Medical Association

9:30—A MAN IN A ROWBOAT—Walter R. Courtenay, D.D., S.T.D., Nashville, Tenn., Pastor, First Presbyterian Church

10:00—THE LAST MILE—Ever Curtis, M.D., Gloucester, Mass., AMA Committee on Medical Practice

10:30—LET'S ALL GET INTO THE ACT—Jack Schreiber, M.D., Canfield, Ohio, President, Mahoning County (Ohio) Medical Society

11:00—COFFEE BREAK

SECOND MORNING SESSION

Library Stack Area

11:15—SYMPOSIUM FOR COUNTY MEDICAL SOCIETY OFFICIALS—Presidents and Other Officers

Russell Holt, M.D., El Paso, Moderator, Councilor, First District, Texas Medical Association

1. THE PRESIDENT—THE MAN IN THE DRIVER'S SEAT—Tom M. Oliver, M.D., Waco, Councilor, Twelfth District, Texas Medical Association
2. SERIOUS QUESTIONS CONFRONTING MEDICINE—Walter Walthall, M.D., San Antonio, Chairman, Board of Councilors, Texas Medical Association
3. EFFECTIVE TECHNIQUES IN MEDICAL SOCIETY LEADERSHIP—Jack Schreiber, M.D., Canfield, Ohio, President, Mahoning (Ohio) County Medical Society

DISCUSSION, QUESTIONS, AND ANSWERS

ALSO

Room 205

11:15—SYMPOSIUM FOR COUNTY MEDICAL SOCIETY SECRETARIES

R. Harvey Bell, M.D., Palestine, Moderator, Councilor, Eleventh District, Texas Medical Association

1. THE SIGNIFICANCE OF THE SECRETARY'S JOB—Walter C. Barnes, M.D., Texarkana, Councilor, Fifteenth District, Texas Medical Association

Saturday the 16th of January

2. TMA FORMS, REPORTS, MEMBERSHIP CLASSIFICATION, DUES, AND DEADLINES—Dick K. Cason, M.D., Hillsboro, Vice Councilar, Twelfth District, Texas Medical Association
3. BY-LAWS—HOW TO USE THEM—James H. Sammons, M.D., Highlands, Secretary, Board af Councilors, Texas Medical Association

DISCUSSION, QUESTIONS, AND ANSWERS

12:30—LUNCHEON—Basement, Compliments af the Texas Medical Association, Catering by Walter Jetton, Fort Worth

AFTERNOON SESSION

Sam Thompson Room

1:30—PANEL DISCUSSION: STATE LEGISLATION OF MEDICAL IMPORTANCE—Jahn M. Smith, Jr., M.D., San Antonio, Moderator, Chairman, Council on Medical Jurisprudence, Texas Medical Association

1. MENTAL HEALTH—Charles H. Brown, M.D., Wichita Falls, Member, Committee on Mental Health, Texas Medical Association
2. TUBERCULOSIS—Charles A. Le Maistre, M.D., Dallas, Chairman, Governor's Cammittee for the Eradication of Tuberculosis in Texas
3. SECOND PHASE—MAA—KERR-MILLS ACT—Joe T. Nelson, M.D., Weatherford, Member, Council on Medical Jurisprudence, Texas Medical Association
4. LICENSING OF CLINICAL LABORATORIES—Floyd A. Norman, M.D., Dallas, Chairman, Special Committee to Study the "Vacuum" Created by Recent Rulings Related to Lay-Owned Labatories, Texas Medical Association
5. LICENSING AND CERTIFICATION OF HEALTH GROUPS—Philip R. Overton, LL.B., Austin, General Counsel, Texas Medical Association
6. THALLIUM POISONING AND CHILD ABUSE LEGISLATION—C. W. Daeschner, M.D., Galveston, Professor and Chairman, Department of Pediatrics, University af Texas Medical Branch

GROUP DISCUSSION

QUESTIONS AND ANSWERS

3:30—COFFEE BREAK

CLOSING SESSION

Sam Thompsan Raom

David Wade, M.D., Austin, President-Elect, Texas Medical Association, presiding

3:45—ISSUES FACING THE 59TH STATE LEGISLATURE—Honorable Byron Tunnell, Tyler, Speaker, House of Representatives, The State of Texas

4:15—HOW TO GET BETTER—Joseph E. Burger, Pasadena, Calif., Director of Public Relations, H. V. Nootbaar Company

4:45—ADJOURNMENT

EVENING FELLOWSHIP AND HOSPITALITY

6:30—HOSPITALITY HOUR—Crystal Ballroom, Driskill Hatel, Compliments of The Medical Protective Company

Orientation Program

Texas Medical Association Headquarters Building

1801 N. Lamar Austin Texas

8:30—REGISTRATION AND COFFEE

OPENING SESSION

Sam Thampson Raam

9:00—CALL TO ORDER AND WELCOME—Travis Smith, M.D., Abilene, Vice President, Texas Medical Association

9:10—THE CHALLENGE AHEAD IN 1965—Max E. Johnsan, M.D., San Antania, President, Texas Medical Association

9:30—A MAN IN A ROWBOAT—Walter R. Courtenay, D.D., S.T.D., Nashville, Tenn., Pastor, First Presbyterian Church

10:00—THE LAST MILE—Ever Curtis, M.D., Glaucester, Mass., AMA Cammittee an Medical Practice

10:30—LET'S ALL GET INTO THE ACT—Jack Schreiber, M.D., Canfield, Ohio, President, Mahaning Caunty (Ohia) Medical Society

11:00—COFFEE BREAK

SECOND MORNING SESSION

Sam Thampson Raam

James D. Murphy, M.D., Fart Worth, Speaker, Hause of Delegates, Texas Medical Assaciatian, presiding

11:15—AN ACCOUNTING OF STEWARDSHIP; WHAT HAPPENS TO YOUR DUES—Byron P. Yark, M.D., Haustan, Chairman, Baard of Trustees, Texas Medical Assaciatian

11:35—THE BEST MEDICAL CARE FOR ALL TEXANS—C. Lincaln Williston, Austin, Executive Secretary, Texas Medical Assaciatian

12:00—LUNCHEON—Basement, Campliments of Texas Medical Associatian, Catering by Walter Jetton, Fort Warth

Saturday the 16th of January

AFTERNOON SESSION

Library Stack Area

Charles Max Cole, M.D., Dallas, Vice Speaker, House of Delegates, Texas Medical Association, presiding

1:00—MEDICAL ETHICS—OBLIGATIONS OF THE PHYSICIAN TO HIS COLLEAGUES AND TO THE PROFESSION—James H. Sammons, M.D., Highlands, Secretary, Board of Councilors, Texas Medical Association

1:20—THE PHYSICIAN'S RESPONSIBILITY AS A CITIZEN—Robert Mayo Tenery, M.D., Waxahachie, Immediate Past President, Texas Medical Association

1:40—DOCTOR-PATIENT RELATIONS—Walter Walthall, M.D., San Antonio, Chairman, Board of Councilors, Texas Medical Association

2:00—SERVING THE DOCTORS OF TEXAS—Donald M. Anderson, Austin, Assistant Executive Secretary, Texas Medical Association

2:20—THE PHYSICIAN'S RESPONSIBILITY IN PRESERVING VOLUNTARY HEALTH INSURANCE—A. Rex Kirkley, M.D., Dallas, Chairman, Committee on Health Insurance, Texas Medical Association

2:35—THE VETERANS ADMINISTRATION HOSPITAL PROGRAM; ITS EFFECT UPON THE PRIVATE PRACTICE OF MEDICINE—Milton V. Davis, M.D., Dallas, Chairman, Committee on Military and Veterans Affairs, Texas Medical Association

2:50—LEGAL ASPECTS OF MEDICINE; MALPRACTICE—HOW TO AVOID IT—C. Dean Davis, LL.B., Austin, Assistant General Counsel, Texas Medical Association

3:30—COFFEE BREAK

CLOSING SESSION

Sam Thompson Room

David Wade, M.D., Austin, President-Elect, Texas Medical Association, presiding

3:45—ISSUES FACING THE 59TH STATE LEGISLATURE—Honorable Byron Tunnell, Tyler, Speaker, House of Representatives, The State of Texas

4:15—HOW TO GET BETTER—Joseph E. Burger, Pasadena, Calif., Director of Public Relations, H. V. Nootbaar Company

4:45—ADJOURNMENT

EVENING FELLOWSHIP AND HOSPITALITY

6:30—HOSPITALITY HOUR—Crystal Ballroom, Driskill Hotel, Compliments of The Medical Protective Company

Quackery Meet Well Attended

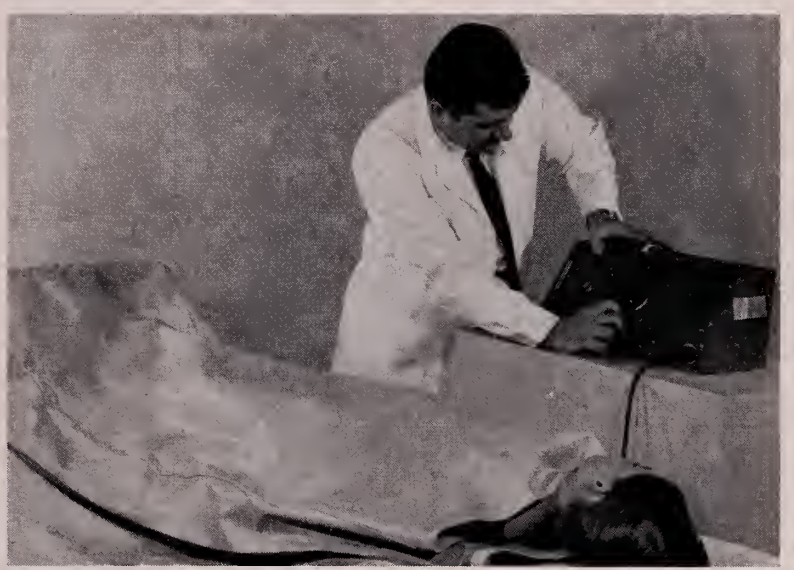
The Central Texas Conference on Health Quackery on Nov. 19 in Austin was well attended, indicating real interest in learning about medical and nutritional quackery and ways to stamp them out.

Held at the Villa Capri Restaurant, the meeting was sponsored by five organizations—Austin Dietary Council, Capital City Dietetic Association, Food and Drug Administration (Dallas District), Texas Nurses Association (District V), Texas State Department of Health, and Travis County Medical Society.

Speakers included Sam D. Fine, director, Dallas District, Food and Drug Administration; Oliver Field, director of the Department of Investigation of the American Medical Association; Elizabeth Neige Todhunter, Ph.D., dean of the School of Home Economics, University of Alabama; and Jerry J. Walsh, special education consultant with the Arthritis and Rheumatism Foundation; and Dr. M.

"Not even good pot holders," says Jerry J. Walsh of this item modeled by pretty girl in picture which quacks have found to be o good seller. Persons with arthritis prove to be best prospects for these "therapeutic" infrared mitts. More than 100,000 have been sold at $65 a pair, according to Walsh, special educationol consultant with the Arthritis and Rheumatism Foundation, New York. (Photogroph courtesy *Today's Health.)*

This device may look funny, but its use was serious business for those who ascribed to the "orgone energy" theory. Invisible "orgone" energy, according to the inventor, permeoted the atmosphere. Cure for the sick person came by inhaling through an "orgone" accumulator. (Photograph courtesy of *Today's Health.)*



Heolth practitioners were the dupes sold more than 1,000 of these devices. They in turn charged their patients for treatments which "directly heated the blood" to get possible benefits of fever therapy. Heating units were placed next to the patient's legs. The patient was wrapped in blonkets and then placed inside the rubber blanket. The heat was turned on for a half-hour to an hour. Practitioner occasionally refreshed patient with cool drinks of water. (Photograph courtesy of *Today's Health.)*

The man with the shiny pate is a frequent target for the quack. This so-called hair restoring gadget, on model above, uses a Christmas tree light bulb to produce infrared energy. (Photograph courtesy *Today's Health.*) Dr. M. H. Crabb, secretary of the Texas State Board of Medical Examiners, once paid a quack hair restorer an exorbitant price for several bottles of lotion and a stiff brush when he was acting as his own investigator. Dr. Crabb deplores the fact that practicing medicine without a license is only a misdemeanor in Texas and the penalty imposed on conviction is not stiff enough to deter a prosperous violator for long.

H. Crabb, secretary, Texas State Board of Medical Examiners.

Displayed during the meeting were various machines and gadgets used in quack practices. Some of these are pictured.

San Antonio To Host International Meeting

Dates of the 29th annual meeting of the International Medical Assembly of Southwest Texas are Jan. 28-30. It will take place in San Antonio at the Granada Hotel.

The program names 16 guest speakers. These are Dr. Joseph Beninson, Detroit, dermatology; Dr. Charles W. Crumpton, Madison, Wis., internal medicine; Dr. John M. Weller, Ann Arbor, Mich., internal medicine; Dr. Francis C. Wood, Philadelphia, internal medicine; Dr. Roger B. Scott, Cleveland, Ohio, obstetrics and gynecology; Dr. Harold F. Falls, Ann Arbor, Mich., ophthalmology;

Dr. P. R. Lipscomb, Rochester, Minn., orthopedic surgery; Col. Frank M. Townsend, Brooks Air Force Base, pathology; Dr. James H. Wall, White Plains, N. Y., psychiatry; Dr. Lewis A. Barness, Philadelphia, pediatrics; Dr. C. Everett Koop, Philadelphia, pediatric surgery; Dr. John M. Converse, New York, plastic surgery; Dr. Harley C. Carlson, Rochester, Minn., radiology; Dr. Carlos Walther, Mexico City, surgery; Dr. Bentley P. Colcock, Boston, surgery; and Dr. John J. Murphy, Philadelphia, urology.

There will be a number of social events during the meeting including a party at the San Antonio Country Club Jan. 28, a brunch and style show the morning of Jan. 29 and a dinner dance that evening. On Jan. 30 there will be breakfast given by the San Antonio Chapter of the Christian Medical Society, a golf tournament, and cocktail buffet.

Additional information may be obtained by writing to Dr. M. A. Childers, president, or S. E. Cockrell, Jr., executive secretary, 202 West French Place, San Antonio.

Injury Reports Requested By Athletics Committee

Serious injuries to participants in junior high, high school, and college athletics are of interest to the Committee on Athletics. Dr. Robert G. McCorkle, chairman, has asked members of the TMA to report such injuries to him.

Reports, which should include the name and address of the injured, sport, date and type of injury, and circumstances causing the injury, should be sent to Dr. McCorkle in care of Texas Medical Association, 1801 N. Lamar, Austin.

Texas Children Part of Survey

The Public Health Service's Health Examination Survey will be conducted in Brownsville and Houston during the coming months. Examinations will be given in Brownsville from Jan. 14 through Feb. 3. Tentative dates set for the Houston visit are Feb. 8-March 1.

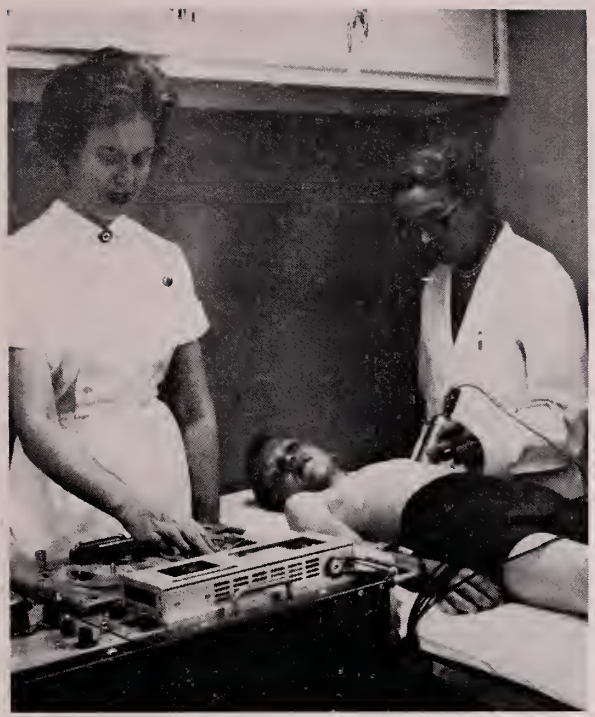
The survey is part of a nationwide sampling study of children, authorized by Congress in 1956, in which children ages 6 through 11 are examined in 40 areas of the country. About 8,000 children are selected by a scientifically designed sampling process in cooperation with the Bureau of Census.

The examinations will be given in a mobile examination center.

The purpose of the survey is to collect on a uniform basis statistical information on various aspects of children's health and to obtain data on certain physical and physiological measurements of these children, relating to growth and development. (See pictures, p. 1033.)

The process will include a specially designed limited examination by a pediatrician to detect neuromuscular and joint conditions, and heart disease; a dental examination by a dentist; a test of visual acuity and color vision; an audiometric test performed in a soundproof room; an X-ray of the wrist for bone age; an X-ray of the chest for cardiovascular and pulmonary abnormalities; a ten-lead electrocardiogram; a test of vital capacity, using a spirometer; an exercise tolerance test under a measured workload, using a bicycle ergometer; a grip-strength test; and recordings of height, weight, skinfold thickness and various other anthropometric measurements; and a series of psychometric tests administered by a psychologist.

The Health Examination Survey is designed to collect data primarily on growth and development. The survey is not intended as a screening procedure; referral for diagnosis is not made. The fact that the examination is not complete and is not a substitute for a visit to the child's own physician and dentist is stressed with the parents of each child examined. A report of findings is to be sent to the child's physician and dentist when the parent requests that this be done.



Measuring Heart Sounds

Measuring Weight

Testing the Hearing

Testing the Eyes

The examining physicians will be fellows or senior residents in pediatrics working temporarily with the Public Health Service. Other members of the team will include nurses, a dentist, two psychologists, and X-ray and other technicians regularly on the PHS staff.

Eradication of TUBERCULOSIS in Texas

JOHN W. MIDDLETON

TUBERCULOSIS continues to be a problem in Texas. The number of new cases each year slowly declined for many years but for the past four years has been essentially unchanged. In 1963, 2,455 Texans developed active tuberculosis.

In September, 1963, Gov. John Connally appointed a committee to study the tuberculosis problem and make recommendations for a program of control in Texas that would lead to ultimate eradication of the disease. The committee was composed of physicians and other persons representing a wide spectrum of experience.

In its report to the Governor last October the Committee emphasized the present fragmentation of efforts at tuberculosis control in the state. Case finding is the responsibility of the State Department of Health, hospitalization that of the Board for Hospitals and Special Schools, and other services are provided by the State Department of Public Welfare and the Texas Education Agency. Tuberculosis control effort at local levels is spotty, depending upon the interest of and funds available to the local health departments. Many patients are hospitalized a long distance from home. Posthospital treatment and follow-up varies widely and in many cases is nonexistent. The State Department of Public Welfare plays an important, though limited, role in support of the family of the incapacited tuberculosis patient, primarily through aid to dependent children. The Texas Education Agency through its Division of Vocational Rehabilitation trains ambulatory patients for gainful occupations in keeping with residual medical disabilities. It is small wonder that the Committee reports, "Fragmentation of services, division of authority, confused and diffused responsibility have resulted."

Further, the Committee feels that the individual physicians in the state have not been sufficiently involved in the tuberculosis control program.

Treatment of active cases and care of those most likely to develop the disease is thought to be the keystone in prevention of further spread of infection. The effort must be statewide, with clearly defined authority and responsibility, but to be effective the program must be functional at the local level.

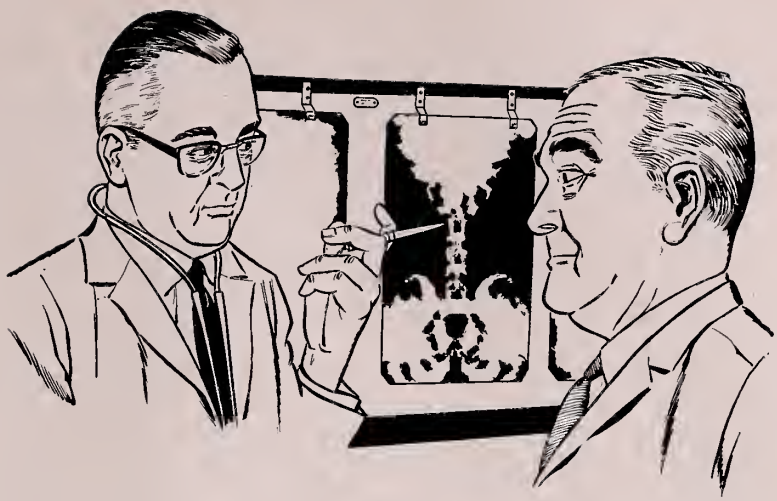
The Committee has proposed consolidation of all forces concerned with the tuberculosis control program under the State Department of Health. A Division of Tuberculosis Services would have entire responsibilities to find cases, provide treatment, and supervise the patient following his release from hospitalization.

Under the Division of Tuberculosis Services, 11 regional areas are recommended, based on population and estimates of the tuberculosis problem in each region. Each region would be responsible for the program within its area, including complete treatment of the infectious patient, identification of all patients with active disease, conducting screening examinations to protect high risk groups, and maintaining regional laboratory and x-ray facilities.

The Committee believes that the majority of patients can be treated successfully in approved community hospitals by qualified practicing physicians without increased cost to the State. The recommendations provide for such care on a contract basis, with the physician chosen by each patient from a list of those approved as qualified for such care. Posthospitalization treatment and supervision would also be available on a contract basis.

The Committee took note of special problems existing along the Texas-Mexico border. One of the regions to be established includes the entire border, in order that an effective control program may be established for that area. It is recommended that the efforts be fully coordinated with the Government of Mexico and particularly those of the bordering Mexican states, since a control program for the Texas side alone would not only be ineffective but would ignore the obvious mobility of the population.

Because of problems of cost, availability of personnel, and administrative reorganization, it was recommended that the program initially be put into effect in five regions with the highest prevalence of tuberculosis. The suggested regions would be headquartered at Houston, Harlingen, San Antonio, Dallas, and Corpus Christi. It is hoped that this can be done at an early date, with the remaining six regions to be developed during the period 1967 to 1973. The conclusions and recommendations of the Committee are as follows:



1. Tuberculosis remains the most expensive specific infectious disease in Texas and is the infectious disease of greatest importance from the public health standpoint.

2. Present state and local efforts to control tuberculosis in Texas are inadequate and are unlikely to alter the serious tuberculosis problem. Fragmentation of services, division of authority, and ill-defined responsibility characterize the present program.

3. Perpetuation of tuberculosis in Texas is unnecessary. Chemotherapeutic measures and modern public health methods, when applied to their fullest potential, can interrupt the spread of tuberculosis and prevent new infection.

4. Prevention of new infection is the hallmark of modern tuberculosis control and the gateway to tuberculosis eradication. Attainment of this goal will require an organized and coordinated effort in case finding and treatment supervision, forged at local levels and directed on a statewide scope by a new Division of Tuberculosis Services of the Texas State Department of Health.

5. A regional tuberculosis control plan appears to be the most feasible and desirable for a state as large as Texas.

6. Important new trends in the management of tuberculosis are recognized and accepted. Such elements as a shortened period of infectiousness and reduced hospitalization requirements make possible the transfer of the place of treatment to the patient's own community for all but those with advanced or protracted illness.

7. Financial support of the medically indigent patient's family during the period of incapacitation is a sound investment in the community's and the patient's interest.

8. The Texas-Mexico border area presents a unique problem in tuberculosis control that requires separate and special attention.

9. The continued need for the authority to effectively and quickly quarantine the recalcitrant patient is recognized.

10. To be effective at the community level, a broad-spectrum tuberculosis control program must actively involve the practicing physician.

The "Report of the Governor's Committee" outlines the following recommendations:

Recommendation No. 1.—The plan for the eventual eradication of tuberculosis in Texas, as described in this report, is recommended for adoption by the State of Texas.

Recommendation No. 2.—It is recommended that the State of Texas finance, initiate, and operate the plan.

Recommendation No. 3.—The full responsibility and authority for all elements of the tuberculosis control effort should be assigned to a new Division of Tuberculosis Services in the Texas State Department of Health.

Recommendation No. 4.—It is recommended that the authority and responsibility for operation of the four State tuberculosis hospitals be transferred to the Division of Tuberculosis Services of the Texas State Department of Health and that all services for the tuberculous, originating at the State level, be unified in, or correlated through, the Division of Tuberculosis Services. It is further recommended that McKnight State Tuberculosis Hospital and East Texas Tuberculosis Hospital be granted legal authority to operate outpatient clinics and to administer to patients on an outpatient basis. Such authority is currently granted only to San Antonio State Tuberculosis Hospital and Harlingen State Tuberculosis Hospital.

Recommendation No. 5.—At the inception of the program, the Governor should appoint an Advisory Committee to formulate preliminary administrative details and recommend a Director. After the appointment of a Director, the Committee will be advisory only but will meet periodically and will provide the Governor, the Legislature, and the Board of Health with an annual written program evaluation.

A Contract Care Credentials Committee shall be appointed by the Director with the approval of the Advisory Committee.

The structure and function of each of these committees is described in the report.

Recommendation No. 6.—It is recommended that a Director of Tuberculosis Services be appointed and delegated full authority and responsibility for development and execution of the outlined plan, co-

ordination of all services to the tuberculous and supervision of all field operations. The Director must be an outstanding physician qualified and able to effect a dynamic program of tuberculosis control throughout the state. It is further recommended that the Director of Tuberculosis Services be allocated sufficient staff that he may effectively discharge his program responsibilities.

Recommendation No. 7.—The adoption of a regional plan, as described in the report, is specifically recommended for adoption. Eleven regions, each headed by a Regional Tuberculosis Control Officer, and provided with the necessary personnel and resources to fulfill the requirements of tuberculosis control, are recommended for eventual development. It is recommended that five regions, selected because of highest prevalence of tuberculosis, be developed to full potential immediately.

Recommendation No. 8.—Specific recommendations are made with regard to Region 2, the border region. It is recommended that legislation be enacted which would prohibit both licensed labor contractors and the Texas Employment Commission from assigning any prospective employee until he has been annually certified as being free from tuberculosis. The costs of such certification are to be borne by the prospective employer.

It is further recommended that an annual certificate of freedom from tuberculosis be required for each alien crossing the Texas-Mexico border. Realizing the emergency nature of this problem and its serious ramifications to the people of Texas, the State should initiate, finance, and operate its own program in this area until the federal government can improve the extent and intensity of its tuberculosis screening program.

Recommendation No. 9.—It is further recommended that a joint United States-Mexico Tuberculosis Control Council be formed at a top governmental level to initiate and expedite a joint program for eradication of tuberculosis in an area 26 kilometers from the border in either direction.

Recommendation No. 10.—It is recommended that the practice of providing Vocational Rehabilitation Counselors to the State tuberculosis hospitals be continued and expanded under inter-agency agreements. Provision of such counselors in each of the regions for services to the patient hospitalized in non-State owned hospitals is recommended. Where necessary, counselors should also work with those patients who are being treated on an outpatient basis.

Recommendation No. 11.—It is recommended that financial aid to the family of the patient incapacitated by tuberculosis be made available during the period of medical treatment upon certification of need.

Recommendation No. 12.—It is recommended that the Texas Tuberculosis Code be amended to require the immediate reporting to the State Department of Health, or its designated tuberculosis divisions, by all laboratories in Texas, including privately owned laboratories, of identification of tubercle bacilli in the excreta or tissues of a human being.

Recommendation No. 13.—It is recommended that all patients or persons legally liable for their support who are able to pay costs of treatment should be required to defray such costs or as much thereof as a review of their financial circumstances indicates they are able to pay. All patients should be thoroughly screened as to financial responsibility and should be required to pay according to financial ability. It is further recommended, however, that a realistic approach to the economic pressures of chronic illness be taken and that a classification of "Medical Indigency for Tuberculosis" be established and used when circumstances justify in order that a lack of personal financial resources will not interfere with or prevent proper and successful treatment.

Recommendation No. 14.—The development of cooperative relationships between medical schools and the State tuberculosis hospitals is to be encouraged in areas of mutual concern. It is recommended that a special study committee composed of representatives from the key agencies involved and from the medical schools be directed to provide a specific series of recommendations to develop mutually advantageous programs for teaching and research which will result in attraction of the best qualified physicians for staff positions in the State tuberculosis hospitals.

Medical Ethics Series Announced

THE BOARD of Councilors of the Texas Medical Association has been asked to prepare a series of articles on medical ethics for the *Texas State Journal of Medicine*. The Board has accepted this as a challenge, for it feels that physicians too frequently know too little about the changing Principles of Medical Ethics.

Today's graduates of medical schools and of postgraduate hospital training are the best-prepared physicians in the history of mankind, but during their years of training, little if any attention is given to the subject of medical ethics. Yet lack of knowledge of how to deal with patients, colleagues, and the public can render useless the product of years of professional training. It has been shown conclusively that the physician who knows well the Principles of Medical Ethics is much less likely to be hauled into court on a malpractice suit or before the board of censors or grievance committee of his medical society.

The Board urges you, if you do not have an up-to-date issue of the *Opinions and Reports of the Judicial Council of the American Medical Association*, to order one from Mr. William J. McAuliffe, Jr., Secretary, Judicial Council, American Medical Association, 535 North Dearborn Street, Chicago, Illinois 60610. With this, you may follow this series of short articles on ethics and, when indicated, keep record of the changes of the Principles, changes in their interpretation, or new interpretations of the various sections. Your comments, questions, or suggestions will be welcomed.

—WALTER WALTHALL, M.D.,
Chairman, Board of Councilors.

Arrangements Committee Lays Plans For Enjoyable, Productive Annual Session

Dr. A. Fletcher Clark, Jr., of San Antonio, Chairman of the Committee on General Arrangements, and his committee of San Antonio physicians are making preparations to assure Texas Medical Association of an enjoyable as well as productive visit to the Alamo City during April 29-May 2, 1965.

The Pecan Valley Country Club has been reserved by Dr. David T. McMahon, sports chairman, for all day Thursday, April 29, for the golf tournament. Dr. Bernard T. Fein will serve as tennis tournament chairman if there are enough doctors interested in the sport. Six courts at the Pecan Valley Country Club are available.

Physicians and their families who are free Friday evening, April 30, will be interested in plans made by Dr. Arthur C. Ressmann, entertainment chairman. The "Fiesta Noche Del Rio" show has been reserved exclusively for TMA. The show—music, dances and songs of Old Mexico—is staged at the Arneson River Theater, an open-air theater which spans the San Antonio River (stage is on the north bank; grass-tiered seats on the south bank). It is a fast-paced show reported to leave audiences spellbound. Three restaurants are also located along the picturesque river, and registrants may have their choice of Italian, Chinese, or Mexican cuisine.

Saturday evening social events will include alumni banquets and fraternity parties. Dr. P. T. Williams, Jr., alumni and fraternity events chairman, has contacted various alumni and fraternity groups regarding their meeting during the 1965 Annual Session. Definite plans have been made by the University of Texas Alumni for cocktails and buffet in the Anacacho Room of the St. Anthony Hotel beginning at 7 p.m. on May 1. The Tulane Alumni Reception and Banquet is scheduled for 7:45 p.m. in the Walnut Room of the Granada Hotel.

Members of the TMA official family are urged to complete their registration forms and send them to the TMA Housing Bureau in care of the Chamber of Commerce office in San Antonio. The last date that preferred space will be available to them is Jan. 18.

Refresher courses which have been announced include the following lecturers and their topics:

Dr. Marvin A. Block, assistant clinical professor of medicine, University of Buffalo Medical School, Buffalo (Texas Commission on Alcoholism and the University of Texas), "The Medical Treatment of Alcoholism"; Dr. Frederick C. Blodi, associate professor of ophthalmology, State University of Iowa, University Hospital, Iowa City (Section on Ophthalmology), "Complications After Cataract Extractions"; Dr. David S. Carroll, clinical professor of radiology, Department of Radiology, University of Tennessee College of Medicine, Memphis, Tenn., President, American College of Radiology (Section on Radiology), "Osseous Manifestations of Systemic Disease";

Dr. Samuel B. Johnson, professor and head, Division of Ophthalmology, University of Mississippi School of Medicine, Jackson, Miss. (Texas Ophthalmological Association), "Diagnosis of Red and Injured Eyes"; Dr. Robert R. Kierland, head of Section of Dermatology, Mayo Clinic, Rochester, Minn. (Texas Dermatological Society), "Cutaneous Malignancies"; Dr. Joseph H. Pratt, chief, Section of Surgical Gynecology, Mayo Clinic, Rochester, Minn. (Texas Division, American Cancer Society), "Squamous Cell Carcinoma of the Cervix in Situ";

"Just one more check before I call it a night."

Dr. Hyrum R. Reichman, assistant clinical professor of surgery, University of Utah College of Medicine, Salt Lake City (Texas Proctologic Society), "Choice of Operation in Cancer of the Rectum"; Dr. William H. Remine, assistant professor of surgery, Mayo Foundation, Rochester, Minn. (Section on Surgery), "Current Trends in Management and Results of Cancer of the Stomach"; Dr. John J. Shea, Memphis Otologic Clinic, Memphis, Tenn. (Texas Otolaryngological Association), "Advancements in Ear Surgery—Importance to Other Physicians"; Dr. William H. Beierwaltes, Department of Internal Medicine, University of Michigan School of Medicine, Ann Arbor, Mich. (Committee on Nuclear Medicine), "Use of Radioisotopes in Obstetrics and Gynecology"; and Dr. John D. French, Director, Brain Research Institute, University of California Medical Center, Los Angeles (Texas Neuropsychiatric Association), "Visceral Reflex Mechanisms in Health and Disease."

Eight general meeting presentations have been announced. The speakers and their subjects are:

Dr. David S. Carroll, clinical professor of Radiology, Department of Radiology, University of Tennessee College of Medicine, Memphis, president, American College of Radiology (Section on Radiology), "Roentgen Findings in the Acute Surgical Abdomen"; Dr. Israel Davidsohn, director, Department of Pathology, Mount Sinai Hospital, Chicago (Section on Pathology), "Immunological Aspects of Cancer and Its Clinical Significance"; Dr. Thomas W. Parkin, consultant in cardiovascular diseases, Mayo Clinic, Rochester, Minn. (Texas Heart Association), "The Clinical Diagnosis of the More Common Mild Congenital Heart Defects in the Adult"; Dr. Hyrum R. Reichman, assistant clinical professor of surgery, Univer-

sity of Utah College of Medicine, Salt Lake City (Texas Proctologic Society), "Diagnosis and Treatment of Surgical Infections of the Ano-Rectum"; Dr. William H. Remine, assistant professor of surgery, Mayo Foundation, Rochester, Minn. (Section on Surgery), "Current Trends in the Management of Duodenal Ulcer"; Dr. Harriet E. Gillette, Department of Physical Medicine and Rehabilitation, Cleveland Clinic (United Cerebral Palsy of Texas, Inc.), "Recovery of Movement Following Brain Damage"; Dr. Max M. Novich, Attending Orthopaedist, Doctors Hospital, Newark, New Jersey (Texas Society of Athletic Team Physicians), "Exercises and Sports for the Pre-School Child"; and Dr. Rafael Camerini-Davalos, Joslin Clinic, Boston, "Chemical Diabetes —Diagnosis and Treatment."

Nine additional speakers have been secured recently, making a total of 36 out-of-state guests for the 1965 Annual Session. They are:

Dr. Rafael Camerini-Davalos, Joslin Clinic, Boston, guest of the Texas Diabetes Association; Dr. John D. French, director, Brain Research Institute, University of California Medical Center, Los Angeles, guest of the Texas Neuropsychiatric Association who replaces Dr. Christian Hermann of Los Angeles; Dr. Harriet E. Gillette, Department of Physical Medicine and Rehabilitation, Cleveland Clinic, Cleveland, guest of United Cerebral Palsy of Texas, Inc.; Dr. Harry D. Morris, professor, Clinical Orthopedics Tulane University School of Medicine, New Orleans, guest of the Texas Orthopaedic Association;

Dr. Max M. Novich, attending orthopaedist, Doctors Hospital, Newark, N. J., guest of Texas Society of Athletic Team Physicians; Dr. Raymond R. Rembolt, professor of pediatrics and director, Hospital School of Severely Handicapped Children, State University of Iowa College of Medicine, Iowa City, guest of United Cerebral Palsy of Texas, Inc.; Dr. V. N. Shirodkar, Patel Chambers, French Bridge, Bombay 7, India, guest of the Section on Obstetrics and Gynecology; Dr. Jackson A. Smith, chairman, Department of Psychiatry, Stritch School of Medicine of Loyola University, Chicago, guest of the Texas Neuropsychiatric Association who replaces Dr. Thomas H. Holmes of Seattle; and Dr. L. O. Underdahl, assistant professor of medicine, Mayo Foundation-University of Minnesota, Rochester, Minn., guest of the Texas Diabetes Association.

Personals

Dr. Fitzhugh Carter Pannill, presently assistant dean of the University of Texas Southwestern Medical School, Dallas, will assume the interim deanship of UT's South Texas Medical School, San Antonio, on Jan. 1. He has been granted leave of absence from the Dallas school to succeed *Dr. Robert C. Berson* at San Antonio. Dean Berson becomes executive director of the Association of American Medical Colleges on Jan. 1.

Dr. Rita Don has been selected El Paso's Outstanding Career Woman of 1964 from 12 entries submitted by local women's organizations.

Dr. Simeon Hulsey of Fort Worth has received Tarrant County Medical Society's Goldheaded Cane award for 1964.

Dr. Robert B. Burns has retired from private practice in Giddings and Lee County after serving for 32 years.

Dr. R. Lee Clark, director and surgeon-in-chief of the University of Texas M. D. Anderson Hospital and Tumor Institute in Houston, has received the National Award of the American Cancer Society, the highest honor given by the ACS, and the Albert Lasker Medical Research Award.

Dr. Courtney M. Townsend, Paris, has been elected president of the University of Texas Dads' Association.

Dr. Edgar J. Poth, chief of the division of general surgery at the University of Texas Medical Branch, has been named the school's first Ashbel Smith Professor.

Dr. William O. Russell of Houston is president of the American Society of Clinical Pathologists.

New directors of the Assembly of the Association of American Physicians and Surgeons include *Dr. Mal Rumph* of Fort Worth. He is also chairman of the Committee on the Education of Medical Students, Interns and Residents. *Dr. Carlos Fuste* of Alvin is continuing as chairman of the organization's Committee on Public Relations.

Dr. P. C. Talkington of Dallas is the incoming president of the National Association of Private Psychiatric Hospitals.

Dr. David Dow of Waco spoke on "Treatment of Acute Eye Conditions" at the November meeting of the Hill County Medical Society.

Dr. J. S. McCelvey, 94, was the subject of an article in the October Alumni Bulletin of the Jefferson Medical College.

Dr. Russell J. Blattner, physician-in-chief of Texas Children's Hospital, Houston, was the recipient of a special award of appreciation from the Houston Pediatric Society on Nov. 12.

Dr. Dan Foster of Dallas spoke on "Cholesterol and Atherosclerosis" at the October meeting of the Tarrant County Medical Society.

Dr. Robert D. Moreton of Fort Worth is the new president of the Radiological Society of North America.

Dr. Edwin L. Rippy, chairman of the Committee on School Health, will take part in a discussion on children's health at a meeting Jan. 7 in Austin sponsored by the Texas Congress of Parents and Teachers, Texas Association of School Administrators, Texas Education Agency, and Texas Medical Association.

Dr. B. L. Aronoff of Dallas is president of the Texas Division, American Cancer Society.

Dr. William T. Anderson was honored as Rotarian of the Month for October by the La Marque Rotary Club.

Dr. Floyd A. Norman of Dallas has received the Dallas Hospital Council's annual award for his contributions to the community's health.

Pathologists Plan January Meet

The Texas Society of Pathologists, Inc., will meet Jan. 29-31 at the Hotel America in Houston.

The meeting is for members only. Papers to be presented include "Incidence of Homologous Serum Jaundice in Blood Transfusions," Dr. Jack P. Abbott, Houston; "Determination of Steroids by Gas-Liquid Chromatography," Bill Creech;

"Protein-Hemoglobin Electrophoresis," Dr. Wilson Brown, Houston; "Clinical Pathology," Dr. John Shiveley, Houston; "Renal Homotransplantation," Dr. R. O. Morgan, Houston; "Patch Grafting of Small Arteries with Particular Reference to Coronary Arteries," Dr. Ed Garrett, Houston; and "Fate of Dacron Arterial Prostheses, Long-Term Follow-Up," Dr. Robert O'Neal, Houston.

Saddle Block Anesthesia Termed "Foreign Object"

MALPRACTICE actions which usually command the most newspaper space are those in which a "foreign object" or "foreign body" has been left in the patient after surgery or other medical procedures.

The definition of "foreign object" or "foreign substance" now has been enlarged to include "saddle block anesthesia," a drastic departure from what commonly was thought of as foreign objects or substances, items such as needles, clamps, sponges, etc.

This unusual definition of "foreign object" comes out of a New Jersey court. The question before the court was the determination of whether anesthesia used to establish a "saddle block" was a foreign object of sufficient dimensions to allow a malpractice action to be brought which would otherwise have been precluded by the statute of limitations in New Jersey. The result of the court's decision would determine the starting of the statute of limitations either from that time when the injection of the anesthesia was given or from the time when the discovery of the cause of injury was made.

The facts briefly indicate that the "saddle block" was administered in March, 1960, and the patient afterwards experienced complaints involving headaches, pain in her back and legs, etc. She had visited numerous doctors complaining of these symptoms and in November, 1960, was told that her complaints were the result of the administration of "saddle block anesthesia." A little less than two years after learning the reason of her injury, a malpractice action was filed by the woman. The defending physician asked that the case be dismissed because such a suit was barred by the statute of limitations in New Jersey.

The court, in reviewing the New Jersey statute of limitations, observed that ordinarily the statute of limitations runs from the time the act of negligence was performed and not from the time of the consequential injury, but in the event a "foreign object" was left in the body of the patient, the statute of limitations would not commence until the patient knew or had reason to know that the foreign object was there and that a cause of action existed.

In its opinion, the court defined "foreign object" as follows:

> not organically connected or naturally related, i.e., a substance occurring in any part of the body or organism where it is not normally found, usually introduced from without.

And, relying on authority from other jurisdictions, the New Jersey court quoted from a California case which had earlier found "the nature or character or the foreign substance or matter introduced into the body of the patient is immaterial," and held that "a foreign substance includes drugs and medicines which are introduced into the body and which are not organically connected or naturally related."

In defense of these allegations the doctor attempted to show that the drug dissolves into the blood stream and may disappear completely from the body; however, the court was not impressed with this defense and considered it immaterial.

Authorities in the various states, it seems, are in hopeless conflict as to when the statute of limitations begins. The Texas case of *Denton v. Carrell* in 1940 established that at least in Texas, a cause of action for malpractice accrues at the time the alleged negligent act is committed, and not at the time the injury from the alleged negligent act is discovered. For now, it appears that the Texas rule on the statute of limitations will not be affected adversely by the New Jersey court ruling.

The decision in the New Jersey case raises questions to whether other drugs and medicines would be considered "foreign substances" when injected or taken orally into the body for the relief of various human ailments.

—PHILIP R. OVERTON, LL.B.,
Austin.

1965 Nursing Workshops Announced

The Texas League for Nursing has announced places and dates for workshops to be held during 1965.

Group B workshops will be held in El Paso Jan. 20-22, Galveston Feb. 10-12, Fort Worth Feb. 10-12, Houston Feb. 17-19, Amarillo Feb. 24-26, Dallas Feb. 23-25, San Antonio March 2-4, and Austin March 9-11.

Group C workshops will be held in El Paso March 2-4, Dallas March 16-18, Galveston March 31-April 2, Houston April 7-9, Fort Worth April 6-8, Austin April 21-23, Amarillo April 28-30, and San Antonio April 28-30.

The workshop program was started in 1960 to help head nurses become aware of their responsibilities in planning and evaluating nursing care, in developing personnel assigned to their units, and in improving their own skills in assessing abilities of personnel and making assignments for nursing care which would utilize their skills most effectively. The program has now been broadened to include supervisors and directors of nursing services.

A registration fee of $15 is charged for each three-day session.

Additional information may be had by writing to Miss Lucy Harris, president, Texas League for Nursing, Inc., 6225 U. S. Highway 290 East, Austin.

Lincomycin, Cephalothin, Erythromycin

Lincomycin

A group of researchers reported the structural elucidation of lincomycin (Lincocin, Upjohn) at the 148th American Chemical Society Meeting (Chicago, Aug. 31-Sept. 4, 1964).

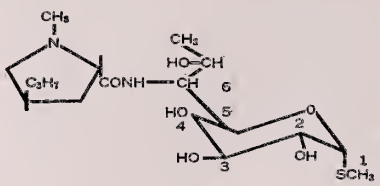
Lincomycin is a new medium-spectrum antibiotic and resembles erythromycin on the basis of its antibacterial activity. It was patented in May, 1963, and is now commercially available in England and several Latin American countries; however, it is still undergoing clinical trials in the U. S. and awaits clearance by the Food and Drug Administration.

The structural elucidation of this antibiotic has provided the basis for future scientific developments; e.g., the synthesis of analogs, which may possess an even broader range of antibacterial activity: the total synthesis of lincomycin which would be of great scientific significance.

The Upjohn report was made in a series of six papers. The characterization and gross structure were described by Drs. Ross R. Herr and George Slomp; the cleavage and stereochemistry of the carbohydrate moiety by Dr. William Schroeder; the configuration and structure of the side-chain by Drs. Brian Bannister and Herman Hoeksema; the structure and stereochemistry of the amino acid fragment by Drs. B. J. Magerlein, Fred Kagan and Mr. R. D. Birkenmeyer; and the nuclear magnetic resonance studies by Dr. Slomp and Mr. F. A. MacKellar.[1] These studies reveal that the carbohydrate portion of the molecule is an amino octose with C-2 to C-5 possessing galactosic stereochemistry and bearing an α-methylthio group at C-1. This thioglycoside is bound to n-propyl hygric acid via an amide linkage.

It is noteworthy that these studies facilitated the unraveling of the structure of celesticetin. Lincomycin, produced by *Streptomyces lincolnensis* is structurally very similar to celesticetin, an antibiotic produced by *Streptomyces caelestis*. Although Upjohn scientists had determined part of the structure of celesticetin in 1956, lincomycin studies have now led to the complete structure of celesticetin. Celesticetin has three known differences from lincomycin. Its amino acid moiety is L-hygric acid, it has a methyoxyl in the side chain of the carbohydrate portion, and it has an alkyl group on the thioglycoside part of the molecule.

Although the total synthesis of lincomycin would not be considered commercially practical, its complete structural elucidation provides the basis for the preparation of semisynthetic congeners analogous to the penicillins. Among approaches studied are modifications of the S-glycoside, substitutions of the amide by other acyl groups, and alterations in the hygric acid moiety of the molecule.



LINCOMYCIN

Fig. 1.

Cephalothin

This new broad-spectrum bactericidal antibiotic is being introduced by Eli Lilly and Co. under the name Keflin.[5] It is an antibiotic of an entirely new chemical class. It was developed in the Lilly Research Laboratories by the reaction of thiophene-2-acetic acid with 7-aminoscephalosporanic acid. The cephalosporanic acid nucleus is obtained from cephalosporin C, which is produced by the fungus *Cephalosporium*.

Cephalothin is bactericidal against both gram-positive and gram-negative organisms in concentrations that are only slightly greater than those required for bacteriostatic action. It is an injectable medicinal agent which will be used primarily in hospitals. Since this antibiotic is not absorbed following oral administration, it cannot be administered via the oral route in the treatment of systemic infections.

Clinical studies reveal that cephalothin has been used successfully in treating the following conditions:

Respiratory tract infections—follicular tonsillitis or septic sore throat, bronchopneumonia, empyema, lung abscess caused by group A beta-hemolytic *streptococci, pneumococci, Klebsiella, Hemophilus influenzae*, or coagulase-positive *staphylococci;*

Urinary Tract Infections—Acute and chronic pyelonephritis, cystitis, and asymptomatic bacteriuria caused by pathogenic strains of *Escherichia coli*, paracolon bacilli, *Aerobacter*, or *Proteus;*

Soft Tissue and Skin Infections—Furunculosis, abscesses, and wound infections with cellulitis, symbiotic gangrene, and pyodermas caused by pathogenic strains of coagulase-positive *staphylococci* and group A beta-hemolytic *streptococci;*

Gastrointestinal Infections—Bacterial gastroenteritis due to *Salmonella* and strains of *Shigella;*

Other infections—Osteomyelitis and septicemia caused by coagulase-positive *staphylococci* and gram-negative bacteremias due to the colon bacillus and *Proteus*.[2]

Erythromycin

This antibiotic which has been pharmaceutically available since 1952 continues to receive the attention of many researchers. It has been investigated from the bi-

opharmaceutics viewpoint as exemplified by a recent review in the *Journal*.[4] It was found in 1953 that erythromycin base is readily absorbed from the upper intestinal tract; however, the absorption of the antibiotic is materially lessened or the drug is destroyed quickly by the action of the gastric secretions. In order to improve the stability of erythromycin in the gastrointestinal tract, and thus improve its absorption, various esters of the drug have been prepared. The dry powder form of propionyl erythromycin appears to be an effective agent chiefly because of its very low "wettability" in the gastric fluid. Propionyl erythromycin lauryl sulfate is another example of how the stability of a drug can be improved in acidic media. This derivative is a salt of a strong acid (lauryl sulfonic acid) with a low water solubility. When the salt comes in contact with the gastric fluids, the drug retains its activity since the gastric acid is not strong enough to displace the lauryl sulfonic acid moiety. In addition the absorption of these derivatives should be greater than that for the free base because the pKa of propionyl erythromycin is 6.9 while the pKa of the erythromycin base is 8.6.

Recently, several workers have endeavored to interpret its mode of action in biochemical terms. Among these the work of Drs. A. D. Wolfe and F. E. Hahn[7] of the Department of Molecular Biology, Walter Reed Army Institute of Research, should be noted. These researchers report that erythromycin, a specific inhibitor of protein biosynthesis, inhibits the incorporation of phenylalanine by cell-free ribosomal system prepared from *Escherichia coli*.

Drs. Wolfe and Hahn state that although its chemical structure offers no ready clue to its molecular mechanism of action, two observations may offer leads in this direction. First, erythromycin has been found by Vasquez[6] to inhibit the interaction between radioactive chloramphenicol and sedimented ribosomal fractions of bacteria; secondly, the capability of erythromycin of precipitating RNA and certain polyribonucleotides has been studied by Wolfe and Hahn.

Hence, Wolfe and Hahn infer that the mechanism of action of erythromycin may be related to that of chloramphenicol and may involve an interaction with one or several categories of RNA that mediate protein biosynthesis.

Jaime N. Delgado, Ph.D.
Frank P. Cosgrove, Ph.D.
Eugene I. Isaacson, Ph.D.,
University of Texas
College of Pharmacy.

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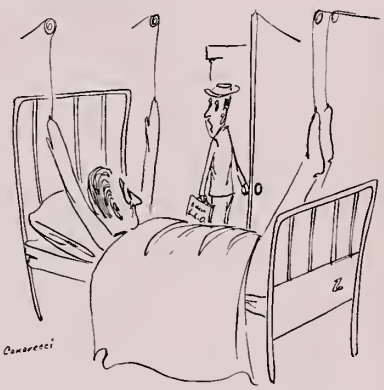
Smallpox Vaccination Certificate Essential

Every day hundreds of world travelers run into delays in quarantine because they fail to present a valid international certificate of vaccination against smallpox.

In the United States the certificate is published as Public Health Service form 731, "International Certificates of Vaccination," revised June, 1961. It is given out with the passport application. The certificate may also be obtained from local and state health departments or from offices of the U. S. Public Health Service. In addition, it may be purchased from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402, at 10 cents a copy. Travel agencies and transportation companies wanting to provide the certificate as a service to their clients may purchase copies at $5.00 per hundred.

To be valid for international travel, the certificate must be completed in every detail, including the following items: name of the person vaccinated or revaccinated; his signature (a parent may write in a child's signature); sex; date of birth; date of vaccination or revaccination; record of either primary vaccination read as successful or revaccination; written signature of the vaccinating physician, even though his nurse or technician may have given the vaccination; and a stamp approved by the health administration of the country in which the vaccination was performed. In the United States, this may be the stamp of the health department of the area in which the vaccinating physician practices.

Additional information on required and recommended immunizations and on health precautions for international travel is available in leaflets issued by the Division of Foreign Quarantine for travel in Asia, in Europe, in Africa, and in Latin America; in a leaflet entitled "So You're Going Abroad"; and in a booklet entitled "Immunization Information for International Travel." Copies can be purchased from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C. 20402. The leaflets sell for five cents apiece or $3.75 per hundred, and the booklet sells for 35 cents.



"And another thing. Don't try to leave town."

Public Health Group Sponsors Essay Contest

Texas high school students this fall will have an opportunity to learn something of the varied and interesting careers awaiting the public health worker while at the same time competing for a $600.00 scholarship award.

"Careers in Public Health" is the title selected by the Texas Public Health Association for their annual essay writing contest. Students of senior high school level, grades 10, 11, and 12, may enter by submitting an original essay of 500 words or less on this subject. Complete contest information is available through local health departments or from the Texas Public Health Association, P. O. Box 5192, Austin 31.

New Books In TMA Library

Aegerter, E., and Kirkpatrick, J. A., Jr.: *Orthopedic Diseases*, 2nd ed., Philadelphia, W. B. Saunders, 1963.

Armstrong, J. R., and Tucker, W. E.: *Injury in Sport*, Springfield, Charles C Thomas, 1964.

Bahama International Conference on Burns: *Report of the Conference on Burns Sponsored by the Colonial Research Institute at West End, Grand Bahama, March 23-25, 1963*, Philadelphia, Dorrance & Co., 1964.

Beeson, P. B., et al.: *Year Book of Medicine*, 1964-5 edition, Chicago, Year Book Medical Publishers, Inc., 1964.

Bellet, S.: *Clinical Disorders of the Heart Beat*, 2nd ed., Philadelphia, Lea & Febiger, 1963.

Bennett, C. W.: *Clinical Serology*, Springfield, Charles C Thomas, 1964.

Blodi, F. C.: *Stereoscopic Manual of the Ocular Fundus in Local and Systemic Disease*, St. Louis, C. V. Mosby Co., 1964.

Burch, G. E.: *A Primer of Cardiology*, 3rd ed., Philadelphia, Lea & Febiger, 1963.

Christensen, H. N.: *Body Fluids and the Acid-Base Balance*, Philadelphia, W. B. Saunders Co., 1964.

Ciba Foundation Study Group No. 18, M. P. Cameron and M. O'Connor, eds.: *Brain-Thyroid Relationships with Special Reference to Thyroid Disorders*, Boston, Little, Brown & Co., 1964.

Diamond, B.: *Obstructing Acromion*, Springfield, Charles C Thomas, 1964.

Dixon, A. St. J., et al.: *Salicylates, An International Symposium Sponsored by the Empire Rheumatism Council*, Boston, Little, Brown & Co., 1963.

Duke-Elder, S.: *System of Ophthalmology*, vol. 3, "Normal and Abnormal Development, Pt. 2, Congenital Deformities," St. Louis, C. V. Mosby, 1963.

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Falk, H. C.: *Urologic Injuries in Gynecology*, Philadelphia, F. A. Davis Co., 1964.

Friedman, H. H.: *Outline of Electrocardiography*, New York, McGraw-Hill Book Co., Inc., 1963.

Gray, L. A.: *Dysplasia, Carcinoma in Situ and Microinvasive Carcinoma of the Cervix Uteri*, Springfield, Charles C Thomas, 1964.

Greenhill, J. P.: *Surgical Gynecology*, 3rd ed., Chicago, Year Book Medical Publishers, 1963.

Holmes, D. J.: *Adolescent in Psychotherapy*, Boston, Little, Brown & Co., 1964.

Jokl, E.: *Medical Sociology and Cultural Anthropology of Sport and Physical Education*, Springfield, Charles C Thomas, 1964.

Jokl, E.: *Nutrition, Exercise and Body Composition*, Springfield, Charles C Thomas, 1964.

Jokl, E.: *Physiology of Exercise*, Springfield, Charles C Thomas, 1963.

Lanzl, L. H., and Carpender, J. W. J.: *Moving Field Radiation Therapy*, Chicago, University of Chicago Press, 1962.

Licht, S.: *Medical Climatology*, vol. 8 of Physical Medicine Library, Maryland, Waverly Press, Inc., 1964.

Lorr, M., et al.: *Syndromes of Psychosis*, New York, Macmillan Co., 1963.

Middlemiss, H.: *Tropical Radiology*, Great Britain, Intercontinental Medical Book Corp., 1961.

Morrell, R. M.: *Thrombophlebitis*, New York, Grune & Stratton, 1964.

Talbott, J. H.: *Style Book and Editorial Manual*, 2nd ed., Chicago, American Medical Association, 1963.

U. S. Army Medical Service, J. B. Coates, Jr., ed.: *Preventive Medicine in World War II, Communicable Diseases*, vol. 7, *Arthropodborne Diseases Other Than Malaria*, Washington, D. C., Office of the Surgeon General, Dept. of the Army, 1964.

Whittenberger, J. L.: *Artificial Respiration, Theory and Applications*, New York, Hoeber Medical Division, Harper & Row Publishers, 1962.

Wortis, J., ed.: *Recent Advances in Biological Psychiatry*, vol. 5, Proceedings of the 17th Annual Convention and Scientific Program of the Society of Biological Psychiatry, New York, Plenum Press, 1963.

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Biochemical Clinics, The Liver, I. N. Kugelmass, ed., vol. 3, New York, Reuben H. Donnelley Corp., 1964.

Clinica Chimica Acta, vol. 10, no. 1, Amsterdam, Netherlands, Elsevier Publishing Co., 1964.

International Anesthesiology Clinics, Peridural Anesthesia, P. C. Lund, ed.; *The Airway and Larynx*, Leroy W. Krumperman, ed., vol. 2, no. 3, Boston, Little, Brown & Co., 1964.

International Anesthesiology Clinics, Intravenous Anesthesia, John W. Dundee, ed., "Hypothermia," A. R. Hunter, ed., vol. 2, no. 4, Boston, Little, Brown & Co., 1964.

International Review of Tropical Medicine, D. R. Lincicome, vol. 2, New York, Academic Press, 1963.

Modern Treatment, Treatment of Epilepsy, R. N. DeJong, ed., "Treatment of Arthritis," C. M. Pearson, ed., vol. 1, no. 5, New York, Hoeber Medical Division of Harper & Row, Publishers, 1964.

Progress in Hematology, L. M. Tocantins, ed., vol. 3, New York, Grune & Stratton, 1962.

Progress in Liver Diseases, H. Popper and F. Schaffner, eds., vol. 1, New York, Grune & Stratton, 1961.

Progress in Radiation Therapy, F. Buschke, ed., vol. 1, vol. 2, New York, Grune and Stratton, 1958, 1962.

Radiologic Clinics of North America, Radiology and the Skeletal System, G. S. Lodwick, ed., vol. 2, no. 2, Philadelphia, W. B. Saunders, 1964.

Book Notes

Books reviewed in this column are available from the TMA Library.

Handbook of Orthopaedic Surgery. A. R. Shands, Jr., R. B. Raney, and H. R. Brashear, 591 pp. St. Louis, C. V. Mosby Co., 1963. $11.50.

THIS is a significantly new version of the venerable "Shands," authoritative primer of nontraumatic orthopaedics for a quarter of a century. Like previous editions, this sixth edition hammers away at *fundamentals* in the business of diagnosing and treating bone and joint diseases. It emphasizes the "normal" and deviation therefrom with alarming clarity; so much so that the orthopaedist will find himself "learning" things he thought he already knew.

Clearly simple enough for reading by nursing students, yet meaningful enough for the trained specialist, this is a notebook that does exactly what its preface promises: "to present the fundamental facts and principles of orthopaedics in brief, readily available form." No pretense is made to present any original ideas, to discuss controversial orthopaedic diagnoses or techniques, or to dwell unnecessarily long on any one subject.

The book includes a satisfactory index, a well-organized and functional table of contents, 277 illustrations, and a 70-page bibliography that is worth the price of the book. Table of contents contains description of 22 chapters, beginning with definitions, basic science principles, orthopaedic examination techniques, congenital deformities, bone, infections, tumors, arthritis, neuromuscular disorders, amputations and prostheses, and anatomical affections (spine, low back, hip, knee, ankle and foot, neck, shoulder, elbow, hand). Basic fracture principles are briefly discussed, in general terms.

Here is a primer textbook true to its stated purpose, contemporary in scope, yet old-fashioned enough to warm my heart by sticking to the *proper* spelling of the word *orthopaedic.*

—Joseph M. Abell, Jr., M.D.,
Austin.

Fundamentals of Otolaryngology. By L. R. Boies, J. A. Hilger, R. E. Priest, 4th ed. Philadelphia, W. B. Saunders, 1964.

ABOVE all other considerations, this textbook is concise and to the point. The authors' teaching experience as well as clinical experience has enabled them to make every paragraph valuable and to resist the temptation to make the book too lengthy and unwieldy for the undergraduate by adding unessential material. The text is very well illustrated with photographs and diagrams, and color plates are included where indicated.

At the end of each chapter is a reference list for those who have special interest in the field of otolaryngology. The book is well indexed and the text is freely sprinkled with subheadings in outlined forms so that specific material is easily and quickly found when the book is used as a manual or reference. This book will find wide use as a concise textbook in otolaryngology for undergraduates as well as a quick reference manual and reference list to the classic literature for residents and practicing otolaryngologists. It should be a part of the library of the generalists and the specialists, outside the field of otolaryngology who require quick concise reference source of basic otolaryngological information.

—W. P. Anthony, M.D.,
Fort Worth.

Ocular and Adnexal Tumors. M. Boniuk, ed. St. Louis, C. V. Mosby Co., 1964.

THIS book is published in an excellent size which is easy to hold and read, and it fits nicely on an ordinary bookshelf. It represents the papers presented, and panel discussions held, at the International Symposium on Ocular Tumors at the Texas Medical Center in 1962 under the sponsorship of the Department of Ophthalmology, Baylor University College of Medicine. The papers and discussions have been edited into a very readable, comprehensive, and informative book, yet one of manageable size.

"Mom! I think Dad's in the bathroom doing a physical on Santa Claus."

This may be partly due to the use of only four-color plate pages, although the book is profusely illustrated with small, but adequate, black and white illustrations. Small print on the front says, "New and Controversial Aspects." New aspects of clinical and histological diagnosis, and surgical and radiational therapy are presented. Points on which even the experts are in disagreement are discussed.

—James L. Mims, Jr., M.D.,
San Antonio.

New Films In TMA Library

Pulse of Life—1962. Lay professional, sound, color, 27 minutes.

THIS FILM gives a brief history of resuscitation methods by following laboratory operating room and physician-supervised demonstrations, mouth-to-mouth resuscitation and external cardiac compression. Audiovisual aids and animation are utilized to emphasize the problems of diagnosis and treatment. The film then dramatically reveals real life situations where these methods are used to save life.

Rx for Maryanne—1964. Lay, sound, color, 15 minutes.

THIS FILM is designed to give an insight into the industry which plays such a vital role in saving lives and relieving human misery. It tells the pharmaceutical story against the background of a mother's purchase of a prescription for her sick daughter. Nine-year-old Maryanne Farley's prescription is traced from the point it is written to the time it is actually administered.

A Question of Birth Defects—1961. Lay, sound, black and white, 23 minutes.

THIS FILM presents an interview with two doctors who discuss what is known about the causes of birth defects; factors in pregnancy such as climate, altitude, drugs, and in-

fection. The doctors also discuss whether or not psychological factors in pregnancy can cause birth defects.

Diagnosis and Management of Eye Injuries—1962. Professional, sound, color, 30 minutes.

THIS FILM deals with the diagnosis and treatment of several typical or representative eye injuries. Particular stress is placed on the general approach to the injured eye. It shows how to obtain maximum information about the eye without adding to the damage by improper handling of the eyelids. The methods of treatment shown include some surgical procedures of the emergency type.

The Making of a Doctor—1964. Sound, black and white, 30 minutes each, 3 reels.

THESE three half-hour films star real doctors and students under actual conditions. Each film is designed to serve independently and may be reserved separately.

Chapter 1 "Had I But World Enough—And Time"

Produced under actual teaching situations at the University of Missouri School of Medicine at Columbia, Mo., the film follows the academic experiences of student John Silver as he makes his way through the liberal arts and sciences of college and the preclinical and clinical curriculum of medical school to earn his M. D. degree.

Chapter 2 "Within These Walls"

How much and how little the young physician knows when he finishes medical school is dramatized in the second program. It shows an 11-year-old girl, badly jaundiced and with an acute pain in her abdomen, under the care of an entire medical team in a big city university hospital. The continuing education of the physician and his dependence upon the supporting professional personnel are effectively presented as an intern and two resident physicians participate in a baffling case which requires the combined effort of the most eminent specialists in the institution.

Chapter 3 "A Wider World"

Three of the many faces of medicine—private practice, public health, and medical research—are developed in the final program. Set in the framework of a family physician, the film depicts the broad human spectrum of illness and disease he is exposed to on a day-to-day basis. It is dramatically impressionistic in its technique, moving also into scenes of leukemic children and doctors in the research world of the Roswell Park Memorial Institute in Buffalo, New York. Life in public health work is depicted in a suspense-filled sequence of medical detection, filmed at the Erie County Health Department. Step by step, a public health doctor is followed as he isolates and identifies the cause of an outbreak of food poisoning that menaces an entire city.

Recent Deaths

DR. R. ETTER

Dr. Roscoe Etter, 76, of Tyler and an honorary member of Texas Medical Association, died in a local hospital Sept. 27. He had retired from his practice in Arp and had moved to Tyler in May, 1964.

He was born in Warren County, Tenn., Jan. 18, 1888, and was a graduate of Vanderbilt University.

He had spent 52 years in the practice of medicine. He had practiced privately in Waco and was the first physician there to limit his practice to pediatrics. He practiced with the Veterans Administration, and he had smalltown practices in Alto and Arp. During World Wars I and II he had served in the medical corps and was discharged with the rank of lieutenant colonel in 1946.

He was a Baptist and a Mason.

A member of the American Medical Association, he had been affiliated with Texas Medical Association through the Smith County Medical Society.

Survivors include his wife, Mrs. Frances Etter of Tyler; one son, Eugene T. Etter of Tyler; one brother, E. B. Etter of Chattanooga, Tenn.; one sister, Mrs. Gaither Humphreys of Currituck, N. C., and one grandchild, Stephanie Etter.

DR. R. ETTER

DR. E. BECK

Dr. Emma Beck of Fredericksburg, who was elected to inactive membership in TMA in 1953, died Sept. 25 in a Kerrville hospital. Death resulted from heart failure and arteriosclerosis.

Dr. Beck had started her career in public health work at the Torbett Clinic in Marlin and later was employed by the San Antonio State Hospital in San Antonio and the Public Health Department of the City of San Antonio. Her specialty was pathology and bacteriology.

She was born Feb. 14, 1888, in Williamson County. She attended Southwest Texas State College in San Marcos and received her M.D. degree from the University of Texas Medical Branch in Galveston in 1920. She interned at New York Infirmary for Women and Children.

DR. E. BECK

Dr. Beck was a member of the American Medical Association and TMA through the Bexar County Medical Society.

She is survived by her husband, Donaldson Rector of Fredericksburg, whom she married in October of 1928.

DR. R. H. JOHNSTON

DR. R. H. JOHNSTON

Dr. Robert H. Johnston, a Mercedes physician who had traveled throughout the world, died Sept. 17 in a Houston hospital. A resident of Mercedes for 18 years, he had been a Presbyterian medical missionary in China from 1930 to 1935.

He was born Nov. 25, 1905, in McKenzie, Tenn. He was the seventh and youngest child of Walter E. and Betty May Johnston. After attending Maryville College in Tennessee he went on to the University of Florida where he was graduated at the age of 19. He received his medical degree from Vanderbilt University in 1929 and interned at Fitzsimmons General Hospital in Denver.

The Presbyterian Mission Board sent him to head mission hospitals in Changteh and Chenchow, Hunan Province. While he was attending language school in Peiping, he and Mrs. Johnston, the former Anne Vanderslice, were married.

When he returned to the United States, Dr. Johnston set up medical practice at Clarksville, Ark. In World War II he commanded the first field hospital to be flown "over the hump" into West China.

He came to Mercedes after the war. There he was active in church work, the Rotary Club, the Masonic Lodge, the International Order of Odd Fellows, the Elks Club, the Shrine, the American Legion, and with music. He was proficient at both organ and piano and often played publicly.

He was a member of the American Academy of General Practice, the American Medical Association, the Texas Medical Association, and the Hidalgo-Starr Counties Medical Society.

He and his wife had traveled extensively in Europe and Russia. His travels were often the topic of speeches he made at various Valley functions.

He is survived by his wife; two daughters, Mrs. Eloise Galvan of Austin and Mrs. Camille Jones of Dallas; two sons, Walter Johnston of Bryan and Robert Johnston of Nashville, Tenn., who is a medical student; and four grandchildren.

DR. G. L. NESRSTA

DR. G. L. NESRSTA

Dr. George Louis Nesrsta, 58, died in a San Angelo hospital on Sept. 10. For the past year he had been chief of medical service at Shannon West Texas Memorial Hospital and in 1958 he was president of the medical staff of Baptist Memorials Geriatric Hospital, both in San Angelo.

He was born Jan. 29, 1906, in Flatonia. He was a football player for Brackenridge High School and St. Mary's University in San Antonio. He received his B.A. degree from St. Mary's in 1929 and his M.D. from Creighton University in Omaha, Neb., in 1933. He interned in Omaha at St. Joseph's.

He had practiced four years in Ballinger before coming to San Angelo where he practiced internal medicine until his death.

He married the former Lena Grace Sanderson of San Saba on June 14, 1938.

He was a member of the Tom Green-Eight Counties Medical Society, Texas Medical Association, and the American Medical Association.

A Roman Catholic, he was interested in hunting and fishing.

He is survived by his wife; three daughters, Annice Nesrsta of Amarillo, and Nancy and Evelyn Nesrsta of San Angelo; a son, George L. Nesrsta, Jr., of San Angelo; three brothers, Ben Nesrsta, Jim Nesrsta, and Charles Nesrsta, all of San Antonio; six sisters, Ann Nesrsta, Maria Nesrsta, Mrs. Frank Marci, Mrs. Jack Wargo, all of San Antonio, Mrs. George Lyon of Houston, and Mrs. Ed Etier of Fort Worth.

DR. R. D. McCONCHIE

Dr. Richard Duane McConchie of Aransas Pass died there Oct. 11 of a heart attack. He had prac-

DR. R. D. McCONCHIE

ticed in Aransas Pass for 11 years, and before that, 13 years in Wichita Falls.

He was born June 18, 1912, in Marshall, Ill., the son of B. T. Leslie McConchie and Merle Daly. He was educated in Illinois, including three years at the University of Illinois. He came to Texas for his medical training and received his M.D. degree in 1938 from Baylor University College of Medicine. He served his internship at Baylor University Hospital in Dallas and his residency at Wichita Falls Clinic-Hospital in Wichita Falls.

He was a major in the Army Medical Corps and served in the southwest Pacific and in Japan from 1942 to 1946.

He belonged to the San Patricio-Aransas-Refugio Counties Medical Society, Texas Medical Association, and American Medical Association. He was a Phi Beta Pi.

Dr. McConchie was a member of the Episcopal church. He was a past member of the Aransas Pass school board.

He married Bernice Oleta Watts of Wichita Falls on March 9, 1953.

He is survived by his wife; four sons, Richard, Jr., David, Daniel, and John Watts McConchie, all of Aransas Pass; and his mother and brother, K. L. McConchie, of Taylor.

DR. T. F. HARRIS

Dr. T. Fred Harris of Houston died there Oct. 3. He had been an

DR. T. F. HARRIS

inactive member of TMA since 1962.

A native of Marietta, Ga., he had lived in Houston for 48 years. He was born to Cicero C. and Alice Queen Harris on March 13, 1883. He attended Peacock Military Academy in San Antonio and received his M.D. degree from the University of Tennessee in 1908. He had practiced briefly in New Willard, Tex., and in El Campo before coming to Houston. He served as an Army first lieutenant during World War I.

He was a member of South Main Baptist Church in Houston, the Masonic Lodge, Scottish Rite, Arabia Temple Shrine, and the Doctors' Club. He was affiliated with TMA and AMA through Harris County Medical Society.

Survivors include his wife, Mrs. Sadie Lee Harris; a daughter, Annie Lee Harris; a brother, Claude Harris; and a sister, Mrs. Bess Guinn, all of Houston.

DR. J. G. CIGARROA, SR.

Dr. Joaquin Gonzalez Cigarroa, Sr., Laredo physician and surgeon, died May 27 on a hunting trip near Kodiak Island, Alaska. Death resulted from a heart attack immediately after he shot and killed a large Kodiak bear.

Dr. Cigarroa was born in Mapimi, Durango, Mexico, Feb. 19, 1895. His parents died during his early youth and his brother, Don Leonides, became his guardian. The entire wealth of the family was lost during the Mexican revolution which also caused the emigration of Don Leonides to San Antonio. Destitute and with only 15 pesos in his pocket, young Joaquin left for Mexico City determined to pursue a career in medicine. He worked his way through the preparatory school, for a time joined the Revolutionary forces, and finally in 1921 he received his medical degree from the National University.

In 1922 Dr. Cigarroa married the former Josefina Gonzalez de la Vega in Mexico City. She is a native of Durango and one of her brothers is a member of the Presidential Cabinet and also a former Governor of the State of Durango.

In 1924 Dr. Cigarroa and family emigrated to San Antonio, Texas. He quickly established a fine practice. During his period of residence in San Antonio he became a founder member of the International Medical Assembly and for many years he directed the Beneficiencia Mexicana. He was on the staff of the Santa Rosa Hospital where he in 1934 made medical history in San Antonio as he was one of the first surgeons to successfully suture a stab wound of the heart.

In 1937 he moved to Laredo. At the time of his death he was serving as the City Health Officer. He was past president of the Webb County Medical Society, former chief of staff at Mercy Hospital, Laredo, honorary member of the Mexican medical military corps, during his life in Mexico he was a Counselor of the Educational Ministry, and was Father of the Year for the City of Laredo.

He was a Roman Catholic.

Dr. Cigarroa was an enthusiastic big game hunter and prior to his death had been selected by International Sport as one of the 75 most noted big game hunters on the North American continent.

Survivors include the widow; two sons, Dr. Leonides Gonzalez Cigarroa and Dr. Joaquin Gonzalez Cigarroa, Jr.; a daughter, Mrs. Marco Uribe, a sister, Miss Carlota Gonzalez Cigarroa, all of Laredo; his brother, Leonides, of San Antonio; and 21 grandchildren.

COMING MEETINGS

American Medical Association, Clinical meeting, Philadelphia, Nov. 28-Dec. 1, 1965. Annual Meeting, New York City, June 20-24, 1965. Dr. F. J. L. Blasingame, 535 Dearborn, Chicago 10, Exec. Vice Pres.

Texas Medical Association, San Antonio, Annual Session, April 29-May 2, 1965; Austin, Public Relations Conference, Sept. 24-26, 1965; Austin, Conference for County Medical Society Officials, Jan. 15-17, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Sec.

DECEMBER

American Academy of Dermatology, Inc., Chicago, Dec. 5-10, 1964. Dr. R. R. Kierland, Mayo Clinic, Rochester, Sec.

Medical Society of United States & Mexico, Phoenix, Ariz., Dec. 9-12, 1964. Dr. James Nauman, 1603 N. Tucson Blvd., Tucson, Ariz., Sec.

Southern Surgical Association, Boca Raton, Fla., Dec. 8-10, 1964. Dr. J. D. Martin, Jr., Emory U., Atlanta 22, Ga., Sec.

Texas Academy of Internal Medicine, Dallas, Dec. 12, 1964. Dr. Warren W. Moorman, 901 W. Leuda, Fort Worth, Sec.

Texas Rheumatism Association, Dallas, Dec. 11, 1964. Dr. J. Kemper, 6655 Travis, Houston, Sec.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1964. Dr. Jack B. Lee, 409 Camden St., San Antonio, Sec.

Texas State Board of Medical Examiners, Fort Worth, Dec. 7-9, 1964. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Sec.

Private Clinics and Hospitals, Houston, Dec. 4-5, 1964. Harry H. Miller, Gulf Coast Hospital, Baytown, Sec.

JANUARY

Texas Association for Mental Health, Austin, Jan. 8-9, 1965. Frank B. Self, 4004 N. Lamar Blvd., Austin, Exec. Sec.

Texas Radiological Society, Houston, Jan. 29-30, 1965. Dr. R. P. O'Bannon, 1217 West Cannon, Fort Worth, Sec.

Texas Society of Pathologists, Inc., Houston, Jan. 29-31, 1965. Dr. V. A. Stembridge, Southwestern Medical School, Dallas, Sec.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 28-30, 1965. S. E. Cockrell, Jr., 202 W. French Place, San Antonio, Exec. Sec.

National and Regional

American Academy of Allergy, Bal Harbour, Fla., Feb. 13-17, 1965. Mr. J. O. Kelley, 756 N. Milwaukee St., Milwaukee 2, Exec. Sec.

American Academy of General Practice, San Francisco, April 12-15, 1965. Mr. M. F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Exec. Sec.

American Association of Obstetricians and Gynecologists. Dr. C. L. Randall, 100 Meadow Road, Buffalo 16, Sec.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 14-19, 1965. Dr. W. L. Benedict, 15 2nd St., S.W., Rochester, Minn., Sec.

American Academy of Pediatrics. Dr. E. H. Christopherson, 1801 Hinman, Evanston, Ill., Exec. Dir.

American Asssociation of Plastic Surgeons, Boca Raton, Fla., May 2-5, 1965. Dr. R. M. McCormack, 260 Crittendon Blvd., Rochester, N. Y., Sec.

American Association for Thoracic Surgery, New Orleans, March 29-31, 1965. Dr. H. T. Bahnson, Johns Hopkins Hosp., Baltimore 5, Sec.

American Cancer Society, Inc., New York, Oct. 25-29, 1965. Mr. G. Whittlesey, 219 E. 42nd St., New York, Sec.

American College of Allergists, Las Vegas., Nev., April 4-9, 1965. Dr. John D. Gillaspie, 2141 14th St., Boulder, Colo., Treas.

American College of Chest Physicians, New York City, June 17-21, 1965. Mr. M. Kornfeld, 112 E. Chestnut, Chicago 11, Exec. Dir.

American College of Gastroenterology. Mr. D. Weiss, 33 W. 60th, New York 23, Exec. Dir.

American College of Nutrition. Dr. Robert A. Peterman, 3 Craig Court, Totowa Boro, N. J., Sec.

American College of Obstetricians and Gynecologists, San Francisco, April 4-7, 1965. Mr. D. F. Richardson, 79 W. Monroe, Chicago 3, Exec. Sec.

American College of Physicians. Dr. E. C. Rosenow, Jr., 4200 Pine, Philadelphia 4, Exec. Dir.

American College of Radiology. Mr. W. C. Stronach, 20 N. Wacker, Chicago 6, Exec. Dir.

American College of Surgeons, Atlantic City, Oct. 18-22, 1965. Dr. Samuel P. Harbison, Pittsburgh, Sec.

American Congress of Physical Medicine and Rehabilitation, Philadelphia, Pa., Aug. 22-27, 1965. Dr. Glenn Gullickson, 30 N. Michigan, Chicago 2, Exec. Dir.

American Dermatological Association, Boca Raton, Fla., Feb. 25-March 2, 1965. Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Sec.

American Gastroenterological Association, Montreal, May 27-29, 1965. Dr. David Cayer, 2240 Cloverdale Ave., Winston-Salem, N. C.

American Gynecological Society, New York, May, 1965. Dr. C. J. Lund, 260 Crittenden Blvd., Rochester 20, N. Y., Sec.

American Hospital Association, San Francisco, Aug. 30-Sept. 2, 1965. Dr. Edwin L. Crosby, 840 North Lake Shore Dr., Chicago 11, Sec.

American Heart Association, Bal Harbour, Fla., Oct. 15-19, 1965. Mr. Rome A. Betts, 44 E. 23rd, New York 10, Exec. Dir.

American Medical Women's Association, Chicago, Nov. 10-14, 1965. Dr. Mary A. Sears, M. D. Anderson Hospital, Houston, Sec.

American Neurological Association, Atlantic City, N. J., June 14-16, 1965. Dr. M. D. Yahr, 710 W. 168th, New York 32, Sec.

American Proctologic Society, Minneapolis, Minn., June 14-17, 1965. Dr. N. D. Nigro, 7815 East Jefferson, Detroit 14, Sec.

American Public Health Association, Inc., Chicago, Oct. 18-22, 1965. Dr. B. F. Mattison, 1790 Broadway, New York 19, Exec. Sec.

American Society of Anesthesiologists, Denver, Oct. 23-27, 1965. Mr. J. W. Andes, 515 Busse Highway, Park Ridge, Ill., Exec. Sec.

American Society of Clinical Pathologists, Chicago, Oct. 15-23, 1965. Miss Eleanor F. Larson, 445 Lake Shore Drive, Chicago 11, Mgr.

American Surgical Association, Philadelphia, May 13-15, 1965. Dr. W. Altemeier, Cincinnati General Hospital, Cincinnati 29, Sec.

Association of American Physicians and Surgeons, Inc., Chicago, April 9-10, 1965. Mr. H. E. Northam, 185 N. Wabash, Chicago 1, Exec. Dir.

Gastrointestinal Research Forum. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

Gastrointestinal Research Group. Dr. George A. Hallenbeck, Mayo Clinic, Rochester, Minn., Sec.

International College of Surgeons, Las Vegas, Nev., April 25-29, 1965. Stanley E. Henwood, 1516 Lake Shore Dr., Chicago, Sec.

Radiological Society of North America, Mr. M. D. Frazer, 1744 S. 58th St., Lincoln, Neb., Sec.

South Central Association of Blood Banks, Austin, March 19-20, 1965. L. R. Guy, Ph.D., 2927 Maple, Dallas 4, Sec.

Southern Medical Association, Houston, Nov. 1-4, 1965. Mr. R. F. Butts, 2601 Highland, Birmingham 5, Exec. Sec.

Southern Psychiatric Association, Ponte Vedra, Fla., Oct. 3-5, 1965. Dr. Mark A. Griffin, Jr., Appalachian Hall, Asheville, N. C., Sec.

Southwest Allergy Forum, New Orleans, La., March 14-16, 1965. Dr. W. P. Coleman, 1828 Calhoun St., New Orleans, La., Sec.

Southwestern Medical Association. Dr. Zigmund Kosicki, 1220 North Stanton St., El Paso, Sec.

Southwest Regional Cancer Conference. Tony W. Halbert, 905 5th Ave., Fort Worth, Sec.

Southwestern Society of Nuclear Medicine, Dallas, March 26-28, 1965. Dr. J. R. Maxfield, Jr., 2711 Oak Lawn, Dallas, Sec.

Southwestern Surgical Congress, Hot Springs, Ark., May 10-13, 1965. Dr. R. B. Howard, 301 Pasteur Bldg., Oklahoma City 3, Okla.

State

South Texas District, American College of Surgeons, San Antonio, May 20, 1965. Dr. August Herff, Jr., 1526 Nix Professional Bldg., San Antonio, Sec.

Texas Academy of General Practice, Dallas, Oct. 4-6, 1965. Mr. D. C. Jackson, 1905 N. Lamar, Austin, Exec. Sec.

Texas Air-Medics Association, San Antonio, April 29-May 2, 1965. Dr. C. F. Miller, Box 1338, Waco Sec.

Texas Association of Obstetricians and Gynecologists, Fort Worth, March 6, 1965. Dr. James T. Downs, 3707 Gaston Avenue, Dallas, Sec.

Texas Association of Public Health Physicians, Fort Worth, March 1, 1965. Dr. Francine Jensen, Houston, Sec.

Texas Chapter, American Academy of Pediatrics, San Antonio, April 29-May 2, 1965. Dr. G. K. Womack, 1402 Nueces, Austin, Sec.

Texas Chapter, American College of Chest Physicians, San Antonio, April 29-May 2, 1965. Dr. Rodger G. Smyth, 331 W. Nueva St., San Antonio, Sec.

Texas Dermatological Society, San Antonio, April 29-May 2, 1965. Dr. M. A. Forbes, 607 W. 14th Street, Austin, Sec.

Texas Diabetes Association, San Antonio, April 29-May 2, 1965. Dr. Leonard R. Robbins, 3618 Yoakum, Houston, Sec.

Texas Division, American Cancer Society, Arlington, June 2, 1965. Mr. C. W. Reimann, 5014 Bull Creek Rd., Austin 3, Exec. Sec.

Texas Heart Association, Austin, Sept. 17-19, 1965. Mr. E. T. Guy, 2480 Times Blvd., Houston 5, Exec. Sec.

Texas Industrial Medical Association, San Antonio, April 28-May 2, 1965. Dr. James K. Stewart, P. O. Box 3311, Beaumont, Sec.

Texas Neuropsychiatric Association, San Antonio, April 29-May 2, 1965. Dr. Jake Peden, Box 17330, Dallas, Sec.

Texas Ophthalmological Association, San Antonio, April 29-30, 1965. Dr. J. L. Mims, Jr., 730 N. Main, San Antonio, Sec.

Texas Orthopaedic Association, San Antonio, May 1, 1965. Dr. Margaret Watkins, 3503 Fairmount, Dallas, Sec.

Texas Otolaryngological Association, San Antonio, April 29-30, 1965. Dr. Norman Wright, 1422 Tyler, Amarillo, Sec.

Texas Pediatric Society, San Antonio, Sept. 23-25, 1965. Dr. C. E. Gilmore, 811 Bonham, Paris, Sec.

Texas Physical Medicine and Rehabilitation Society, San Antonio, April 29-May 2, 1965. Dr. Charles Peterson, 1333 Moursund, Houston, Sec.

Texas Proctologic Society, San Antonio, April 29-May 2, 1965. Dr. T. Melton, 636 Hermann Professional Bldg., Houston, Sec.

Texas Public Health Association, Fort Worth, Feb. 28-March 3, 1965. J. N. Murphy, Jr., P. O. Box 5192, Austin 31, Sec.

Texas Rehabilitation Association. D. Preston, 3700 Worth, Dallas 10, Sec.

Texas Society of Anesthesiologists, San Antonio, May 1-2, 1965. Dr. Fred P. Thomas, 2342 Maroneal, Houston, Sec.

Texas Society on Aging. Mrs. Edith DeBusk, Rio Grande Bldg., Dallas, Sec.

Texas Society of Athletic Team Physicians, San Antonio, April 29-May 2, 1965. Dr. Dan Spivey, 818 Frank St., Lufkin, Sec.

Texas Society of Plastic Surgeons, San Antonio, April 29, 1965. Dr. John Gannon, 1433 Hermann Prof. Bldg., Houston, Sec.

Texas State Board of Examiners in Basic Sciences. Mrs. Betty J. Anderson, 1012 State Office Bldg., Austin, Exec. Sec.

Texas Surgical Society, Dallas, April 4-6, 1965. Dr. W. B. King, Jr., 2320 Columbus Ave., Waco, Sec.

Texas Traumatic Surgical Society, San Antonio, April 29-May 2, 1965. Dr. J. C. Long, 805 W. 8th, Plainview, Sec.

Texas Tuberculosis Association, Houston, March 18-20, 1965. Mrs. Pauline K. Matthis, 2406 Manor Road, Austin, Exec. Dir.

Texas Urological Society, Fort Worth, February, 1965. Dr. William W. Wood, Jr., Medical Arts Bldg., Fort Worth, Sec.

United States-Mexico Border Public Health Association, Los Angeles, June 7-10, 1965. Dr. Jorge Jiménez-Gandica, 501 U. S. Court House, El Paso, Sec.

District

First District Society, Pecos, Feb. 6, 1965. Dr. William R. Gaddis, 1223 Mills Bldg., El Paso, Sec.

Third District Society, Lubbock, Oct. 9, 1965. Dr. Louis Devanney, 2209 W. 7th, Amarillo, Sec.

Fifth and Sixth Districts Society, Corpus Christi, July 9-10, 1965. Dr. J. E. Albright, 2481 Medical Center, Corpus Christi, Sec.

Seventh District Society. Dr. R. Lucas, 502 W. 13th, Austin, Sec.

Eighth District Society. Dr. Weldon G. Kolb, 421 S. Oak, La Marque, Sec.

Ninth District Society, Brenham, March 18, 1965. Dr. William Sherrill, 3618 Mt. Vernon, Houston, Sec.

Tenth District Society. Dr. I. M. Richman, 3280 Fannin, Beaumont, Sec.

Eleventh District Society, Athens, March 26, 1965. Dr. F. Verheyden, 400 S. Ragsdale, Jacksonville, Sec.

Twelfth District Society. Dr. Walker Lea, Scott and White Clinic, Temple, Sec.

Thirteenth District Society, Mineral Wells, May 4, 1965. Dr. W. H. Neil, 1217 W. Cannon, Fort Worth, Sec.

Fourteenth District Society.

Fifteenth District Society.

Clinics

Blackford Memorial Cancer Lectures. Dr. A. O. Jensen, Suite N-1, Medical Center, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 21-24, 1965. Dr. J. Morris Horn, 3707 Gaston, Dallas, Dir.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1965. Dr. Mannie D. Paine, Jr., 1430 Tulane Ave., New Orleans 12, Exec. Sec.

Oklahoma City Clinical Conference. Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Exec. Sec.

Postgraduate Medical Assembly of South Texas, Houston, July 12-14, 1965. Mrs. W. H. Dahme, 209 Jesse H. Jones Library Bldg., Houston 25, Exec. Sec.

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Key to Index

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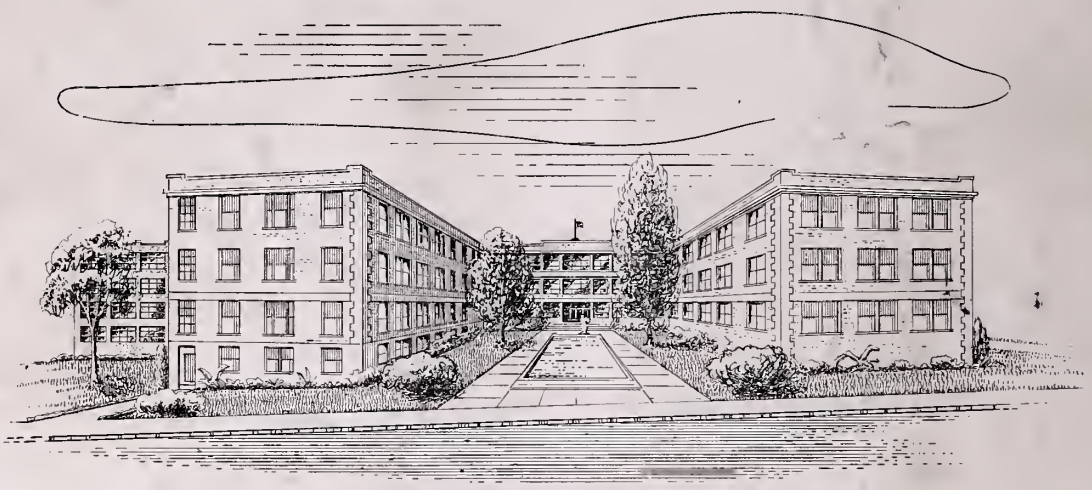

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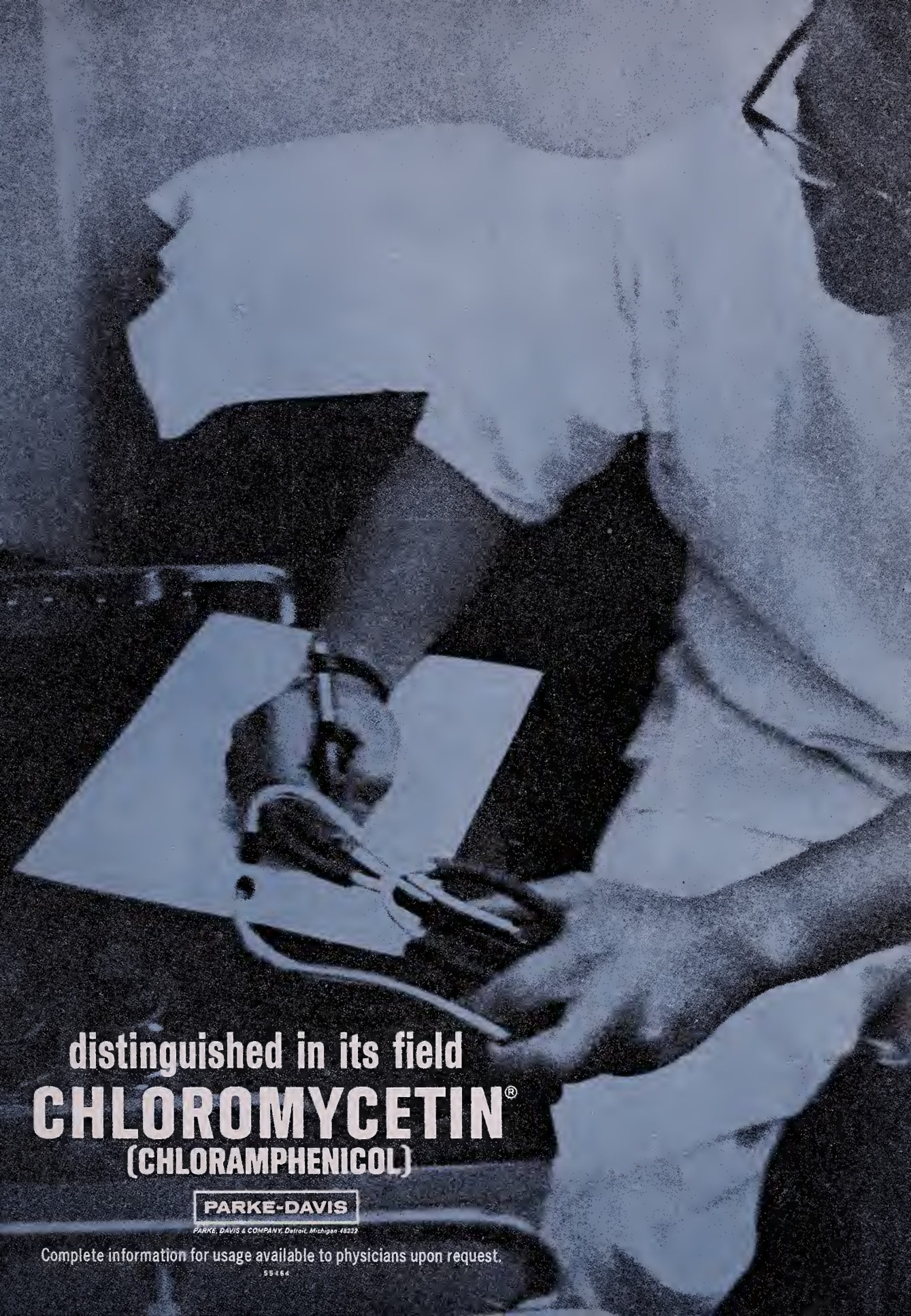
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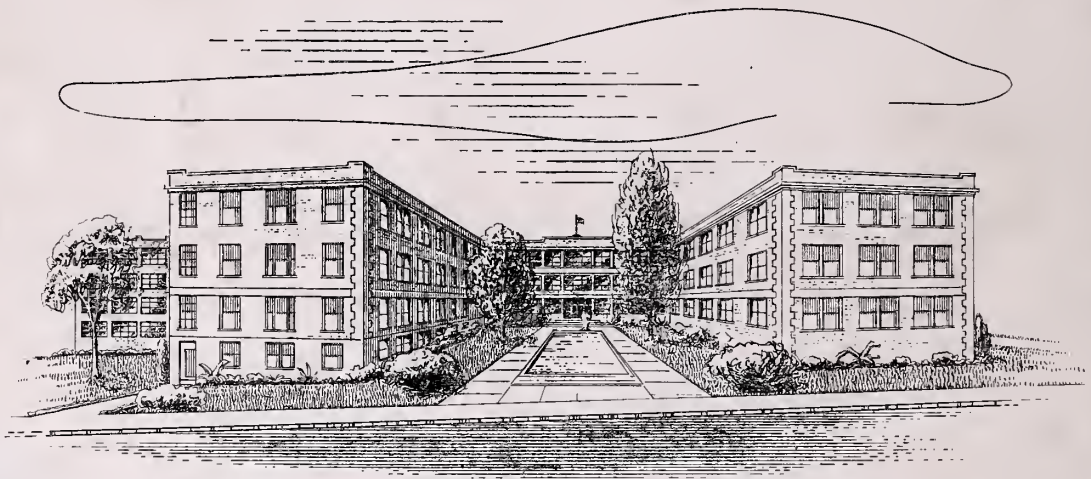

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